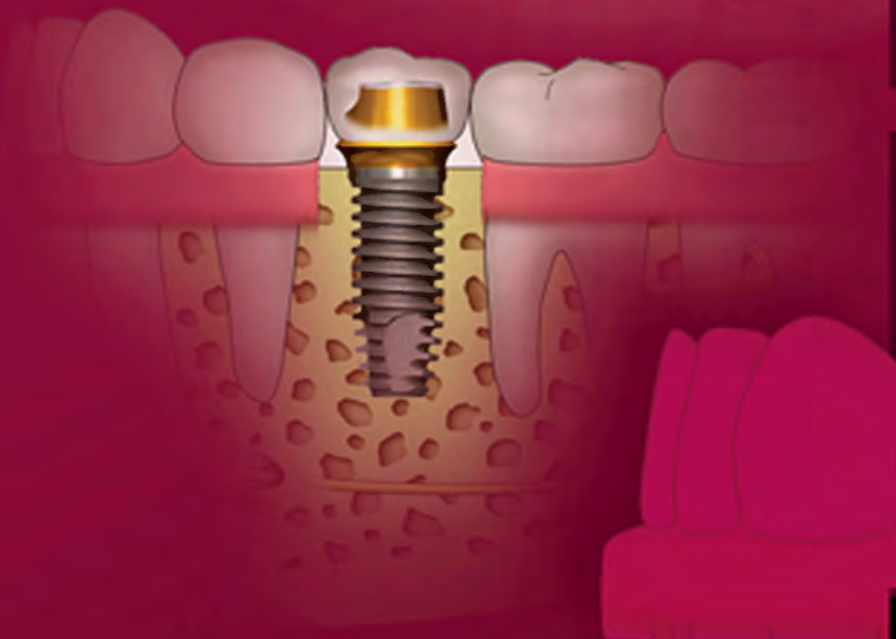
CONTEMPORARY IMPLANT DENTISTRY

This ebook is uploaded by dentalebooks.com

THIRD EDITION



CARL E. MISCH

MOSBY
ELSEVIER

Highdent laboratory

مرکز تخصصی پروتزهای دندانی

های دنت

طراحی و ساخت انواع پروتزهای دندانی بویژه ایمپلنت

برگزار کننده دوره های آموزشی تخصصی وجامع دندانسازی و...

با ما همراه باشید...

WWW.HIGHDENTlab.com

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

MOSBY
ELSEVIER

11830 Westline Industrial Drive
St. Louis, Missouri 63146

CONTEMPORARY IMPLANT DENTISTRY ISBN: 978-0-323-04373-1
Third Edition
Copyright © 2008, 1999, 1993 by Mosby, Inc., an affiliate of Elsevier Inc.

All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Permissions may be sought directly from Elsevier's Rights Department: phone: (+1) 215 239 3804 (US) or (+44) 1865 843830 (UK); fax: (+44) 1865 853333; e-mail: healthpermissions@elsevier.com. You may also complete your request online via the Elsevier website at http://www.elsevier.com/permissions.

Notice

Knowledge and best practice in this field are constantly changing. As new research and experience broaden our knowledge, changes in practice, treatment and drug therapy may become necessary or appropriate. Readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of the practitioner, relying on their own experience and knowledge of the patient, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions. To the fullest extent of the law, neither the Publisher nor the Editor assumes any liability for any injury and/or damage to persons or property arising out of or related to any use of the material contained in this book.

The Publisher

Library of Congress Control Number 2007935907

This ebook is uploaded by dentalebooks.com

Vice President and Publisher: Linda Duncan
Acquisitions Editor: John Dolan
Managing Editor: Jaime Pendill
Developmental Editors: Elizabeth Clark, Julie Nebel
Editorial Assistant: Joslyn Dumas
Publishing Services Manager: Patricia Tannian
Project Manager: Jonathan M. Taylor
Design Direction: Teresa McBryan

Working together to grow
libraries in developing countries
www.elsevier.com | www.bookaid.org | www.sabre.org
ELSEVIER BOOK AID International Sabre Foundation

Printed in Canada

Last digit is the print number: 9 8 7 6 5 4 3 2 1

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Contributors

Martha Warren Bidez, PhD
Professor
Department of Biomedical Engineering
University of Alabama at Birmingham
Birmingham, Alabama

Diana Bronstein, DDS, MS
Adjunct Assistant Clinical Professor
Department of Periodontology and Oral Implantology
Kornberg School of Dentistry
Temple University
Philadelphia, Pennsylvania

Marco Degidi, MD, DDS
Private Practice
Bologna, Italy

Giovanna Iezzi, DDS, PhD
Research Fellow, Dental School
Department of Dentistry
University of Chieti
Chieti, Italy

Louis T. Kircos, DDS, MDS, PhD
Professor
Department of Restorative Dentistry
Loma Linda University
Loma Linda, California

Attending Radiologist
North Bay Radiology
Advanced Imaging of Chicago
Chicago, Illinois

Jack E. Lemons, PhD
Professor
Department of Prosthodontics, Surgery, and Biomedical Engineering
University of Alabama at Birmingham
Birmingham, Alabama

Matthew Lyman, DO
Resident
Department of Orthopedics
Botsford Hospital
Farmington Hills, Michigan

Francine Misch-Dietsh, DDS, MDS
Associate Professor
Department of Periodontology and Oral Implantology
Kornberg School of Dentistry
Temple University
Philadelphia, Pennsylvania

Misch International Implant Institute
Beverly Hills, Michigan

Adriano Piattelli, MD, DDS
Professor of Oral Pathology and Medicine
Dean and Director of Studies and Research
Dental School
University of Chieti
Chieti, Italy

Ana Emília Farias Pontes, DDS, MSc, PhD
Associate Professor of Periodontology and Implantology
Educational Foundation of Barretos
Barretos, São Paulo
Brazil

Girish Ramaswamy, MS
Department of Biomedical Engineering
University of Alabama at Birmingham
Birmingham, Alabama

Randolph R. Resnik, DMD, MDS
Clinical Associate Professor
Department of Periodontology and Oral Implantology
Kornberg School of Dentistry
Temple University
Philadelphia, Pennsylvania

Surgical Director
Misch International Implant Institute
Beverly Hills, Michigan

W. Eugene Roberts, DDS, PhD, DHC (Med)
Jarabak Professor and Director of Graduate Orthodontics
Department of Orthodontics
School of Dentistry
Indiana University
Indianapolis, Indiana

Associate Professor
Department of Oral and Maxillofacial Implantology
School of Medicine
University of Lille II
Lille, France

This ebook is uploaded by dentalebooks.com
t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

David P. Sarment, DDS, MS
Assistant Professor
Department of Periodontics/Prevention/Geriatrics
School of Dentistry
University of Michigan
Ann Arbor, Michigan

Antonio Scarano, DDS, MD
Department of Dentistry
University of Chieti
Chieti, Italy

Gerard M. Scortecci, DDS, MDS, PhD
Chairman of the Post Graduate Oral Implant Program
Department of Oral and Maxillofacial Surgery
School of Medicine
University of Nice Sophia-Antipolis
Nice, France

Clinical Professor of Periodontology
University of Southern California
Los Angeles, California

Visiting Professor
New York University
New York, New York

Mohamed Sharawy, BDS, PhD
Professor and Director
Department of Biology/Anatomy & Oral and Maxillofacial Surgery
Medical College of Georgia
Augusta, Georgia

Miles L. Singer, DO, FACOS, FAOAO
Assistant Clinical Professor
Osteopathic Surgical Specialties
Michigan State University–College of Osteopathic Medicine
East Lansing, Michigan

Clinical Assistant Professor
Des Moines University
Des Moines, Iowa

Clinical Assistant Professor
Department of Surgery
College of Osteopathic Medicine
University of Health Science
Kansas City, Missouri

Private Practice
Orthopedic Surgery
Botsford Hospital
Farmington Hills, Michigan

J. Todd Strong, MS
COO and Executive Vice President
BioHorizons

Adjunct Professor
Department of Biomedical Engineering
University of Alabama at Birmingham
Birmingham, Alabama

Jon B. Suzuki, DDS, PhD, MBA
Professor of Microbiology and Immunology
School of Medicine
Professor of Periodontology and Oral Implantology
Associate Dean for Graduate Education, Research, and International Affairs
Kornberg School of Dentistry
Temple University
Philadelphia, Pennsylvania

Lynn D. Terracciano-Mortilla, RDH
Private Practice
Trinity, Florida

This ebook is uploaded by dentalebooks.com

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Foreword

The profession of dentistry, and in particular the field of implant dentistry, has been significantly changed and improved by Dr. Carl E. Misch. More important, however, is the dental patient who has been the major recipient of the innovations and clinical advancement of this field. From single-tooth implant placement to complex bone augmentation with multiple implant positioning, this textbook provides the science and peer-reviewed research enhancing implant success and improving clinical outcomes.

This textbook leads the clinician through a logical sequence of "growth and maturation" as an implant dentist. Basic chapters on medical history, bone, and pharmacology lead into increasingly complex surgical approaches that include virtually every clinical venue faced by the implant dentist. These well-illustrated chapters with outstanding clinical teaching cases will serve as a basic guide for the dental clinician and as a valuable resource in one's reference library.

A quarter of a century of experience at the Misch International Implant Institute is evident in this textbook. Clinical and teaching approaches that have been proved over time are directly applied in each chapter. The thousands of general dentists and specialists who have successfully completed the comprehensive series of Misch Institute three-day–weekend courses (which includes several years of Temple University and University of Pittsburgh periodontology residents) have provided significant feedback, resulting in continuous outcomes assessments of clinical procedures and research methods highlighted in this text. The reader will not only improve their clinical skills, but also their confidence with Dr. Misch. I speak from personal experience as a board-certified periodontist who completed his first surgical placement of a dental implant under the tutelage of Dr. Misch (while on a giant video monitor viewed by more than 60 dentists during one of the Misch Institute courses) over a decade ago. I continue to be mentored by Dr. Misch and feel fortunate to call him a professional colleague, research collaborator, and true friend.

Jon B. Suzuki, DDS, PhD, MBA

This ebook is uploaded by dentalebooks.com

t.me/highdent www.highdentlab.com instagram.com/high_dent

To my wife,

Francine Misch-Dietsh,

whose love and support make all things possible.

This ebook is uploaded by dentalebooks.com

About the Author

Carl E. Misch, DDS, MDS, PhD(hc)

Carl E. Misch is a Clinical Professor and Director of Implant Dentistry in the Department of Periodontology and Implant Dentistry at Temple University Kornberg School of Dentistry. He is also a Clinical Professor in the Department of Periodontics/Geriatrics at the University of Michigan School of Dentistry. Dr. Misch is currently a Clinical Professor in the Department of Restorative Dentistry at the University of Detroit Mercy School of Dentistry. He is also a Board of Trustee member-at-large for the University of Detroit Mercy School. In addition, he is an Adjunct Professor at the University of Alabama at Birmingham, School of Engineering, Department of Biomechanics. He was Co-Director or Director of the Oral Implantology Residency Program at the University of Pittsburgh School of Dental Medicine from 1986 to 1996.

Dr. Misch graduated *magna cum laude* in 1973 from the University of Detroit Dental School and received his Prosthodontic Certificate, Implantology Certificate, and Master's Degree in Dental Science from the University of Pittsburgh. He has been awarded two honoris causa PhD degrees, from the University of Yeditepe in Istanbul, Turkey, and the Carol Davila University of Medicine and Pharmacy in Bucharest, Romania. Other graduate honors include 13 fellowships in dentistry, including Fellow of the American College of Dentistry, Fellow of the International College of Dentists, Fellow of the Royal Society of Medicine, Fellow of the American Association of Hospital Dentistry, Fellow of the Academy of Dentistry International, and Fellow of the Pierre Fauchard Academy. Dr. Misch has more than 10 patents related to implant dentistry and is co-inventor of the BioHorizons Dental Implant System.

Dr. Misch is a diplomate and past president of the American Board of Oral Implantology/Implant Dentistry and served as member of the diplomate examining committee for 7 years. He is a past president of the International Congress of Oral Implantologists, the American Academy of Implant Dentistry, the Academy of Implants and Transplants, and the American College of Oral Implantologists. He is currently the Co-Chairman of the Board of Directors of the International Congress of Oral Implantologists, which represents more than 85 countries and is the world's largest implant organization.

In 1984, Dr. Misch founded the Misch International Implant Institute, which is co-sponsored with Temple University School of Dentistry. Currently, training centers for the institute are located in Michigan, Pennsylvania, Nevada, and Toronto, Canada. Over the years, the Misch International Implant Institute has had training centers in Korea, Italy, Brazil, Japan, the United Kingdom, Monaco, and Spain. In the United States and Canada, past training centers have been located in Florida, Texas, Nevada, New York, Vancouver, and Montreal. As director, Dr. Misch has trained more than 3500 doctors in a hands-on, yearly forum of education in implant dentistry. Programs are offered in both the surgical and prosthetics aspects of patient care.

Dr. Misch has now edited three editions of *Contemporary Implant Dentistry* as well as the first edition of *Dental Implant Prosthodontics*. These textbooks have been translated into Japanese, Spanish, Portuguese, Italian, and Korean, and they are used in dental schools around the world for graduate programs and postgraduate programs. Dr. Misch has published more than 250 articles related to implant dentistry. During the past 30 years, Dr. Misch has lectured more than 1000 times in all 50 states of the United States and in 47 countries throughout the world.

Dr. Misch is married to Francine Misch-Dietsh, a prosthodontist and past editor of *Contemporary Implant Dentistry*. They have six children: Paula, Carl, Lara, David, Jonathan, and Angela. Dr. Misch has maintained a private practice restricted to implant surgery (bone grafting and implant placement) and related prosthetics for more than 30 years.

t.me/highdent www.highdentlab.com instagram.com/high_dent

This page intentionally left blank

This ebook is uploaded by dentalebooks.com

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Preface

In the 1990s fixed partial dentures to replace missing teeth in a partially edentulous patient were vehemently opposed, and removable partial dentures were strongly encouraged. In 1911, Hunter blamed the "mausoleum of gold over a mass of sepsis" for complicating systemic conditions of anemia, gastritis, kidney disease, and lesions of the spinal cord.[1] Despite this popular belief, fixed partial dentures became the standard of care to replace missing teeth and are still taught in every dental school in North America. In fact, if a dental student does not perform a traditional fixed partial denture, they do not graduate and join the dental community.

In the 1970s the mere mention of dental implants was controversial. Organized dentistry feared that these devices would always fail and could lead to a brain abscess or heart failure, because there was often no barrier between the oral bacteria and the systemic pathways. A few hundred dentists around the world observed that patients readily accepted dental implants to support a mandibular complete denture or believed that a fixed implant prosthesis was more desirable than using removable restorations or preparing and joining adjacent teeth for fixed prostheses.

Today we are in the midst of a dental implant revolution. There are more scientific and clinical articles written on dental implants than any other topic in dentistry. From 1950 to 1985 there were approximately 500 refereed articles published on dental implants. Between the years 1985 and 1995 there were more than 1500 articles published on dental implants. More recently, from 1995 to 2005, there have been over 5000 articles published in refereed journals on topics related to dental implants. The dental implant is now accepted as a promising method to replace a single tooth or multiple adjacent missing teeth, or to support a removable prosthesis for a completely edentulous patient.

In the United States, the total sales of implant products to the dental profession from 1950 to 1985 was less than $1 million each year, and from 1985 to 1995 the sales increased to $100 million per year. The sale of implant-related products from 1995 to 2005 has skyrocketed to $1 billion per year. However, this dramatic increase in sales has a downside. The rapid growth of dental implants as man-made abutments to replace missing teeth has caused technology to develop quickly and often without guidelines for evaluation. The driving force behind implant treatment should not be directed by dental advertising from manufacturers. Procedures should be based on what is predictable.

An underlying theme of *Contemporary Implant Dentistry* is to base the treatment of missing teeth on the sciences related to implant dentistry. This book does not attempt to be an encyclopedia of all that is possible in implant dentistry. Instead, it is a text that relates one chapter to every other chapter and presents a common thread of science and past experience to the art of replacing teeth. Every chapter is carefully blended to be consistent in purpose: to provide a predictable outcome.

The third edition of *Contemporary Implant Dentistry* addresses the science and discipline of implant dentistry. This book has nearly doubled in size and has added new chapters in treatment planning, bone grafting, and implant surgery. In addition, more than 2000 illustrations have been used to detail related concepts.

The first part of *Contemporary Implant Dentistry* presents the rationale for the use of dental implants as man-made abutments and why biomechanics should be used as a basis of implant treatment planning as a way to reduce complications. It also presents a generic seven-step process for treatment planning. More than 50 dental criteria may influence treatment planning and prognosis. Implant dentistry does not guarantee a result, nor is it without complications. However, there is a consistent theme to reduce and eliminate many complications, and this theme starts with a biomechanically based treatment plan.

The second part of this book concentrates on specific diagnoses and treatment plans for both the partial and complete edentulous jaw. These chapters organize and present clinical considerations in a logical sequence. The chapters relate the previous general concepts to the clinical conditions most often observed in practice: the posterior missing single tooth is addressed separately from the anterior missing tooth, and the completely edentulous mandible is different from the maxilla.

The third part of *Contemporary Implant Dentistry* provides information from the related basic sciences of medical evaluation, applied anatomy, bone physiology, pharmacology, biomechanics, and biomaterials.

The fourth part organizes and presents implant surgery with generic concepts and expands the surgery by implant location and anatomic variables. These procedures blend the science of implant surgery with three decades of experience to improve clinical outcomes. This part also addresses immediate loading for a single tooth, overdentures, multiple adjacent missing teeth, and full arch fixed restorations.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

The fifth part of the book presents soft and hard tissue rehabilitation. It begins with the keys necessary for predictable results, proceeds with the traumatic extraction of a hopeless tooth, and lastly discusses barrier membrane bone grafts. This section also includes sinus grafts (complete with specific evaluation, pathology, and complications related to the maxillary sinus). Autograft block bone grafts from intraoral donor sites are also covered in detail, followed by extraoral block and particulate grafts from the ilium and tibia.

The final part of *Contemporary Implant Dentistry* presents the long-term evaluation and maintenance of dental implants. A clinical evaluation and treatment is emphasized.

Contemporary Implant Dentistry has been used over the years as a textbook for dental students, interrelated dental residents, postgraduate programs, implant residents, specialists, and generalists. Its translation into many languages and its widespread acceptance have provided a thinking process for oral implantology. This most recent edition attempts to help further elevate the science and discipline of implant dentistry and allow predictable treatment to replace missing teeth for the patients we treat and the doctors we train.

Carl E. Misch, DDS, MDS, PhD(hc)

Reference

1. Hunter W: The role of sepsis and antisepsis in medicine, *Dent Briefs* 16:852, 1911.

NOTE: Throughout the text, this symbol will appear in various illustrations. This symbol is used to denote the improper or incorrect method.

This ebook is uploaded by dentalebooks.com

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Acknowledgments

In this fourth book, I attempt to share my experience, training, and knowledge in a discipline to which I have dedicated my life. As in all of the other books, I must again acknowledge the undying efforts of my wife, Dr. Francine Misch-Dietsh. She is responsible for most of the literature review in every chapter of which I am the primary author. She directed and worked with the illustrator to ensure each drawing was accurate and clear. She read every line of every chapter repeatedly, ensuring the message was consistent and referenced. She co-coordinated my staff and the publisher in the final preparations of the book. I publicly thank her for the constant dedication to me, our family, and the profession.

There are many additional people to acknowledge and thank in the preparation and publication of *Contemporary Implant Dentistry,* third edition. Allow me to begin with all participating authors: Martha Warren Bidez, Diana Bronstein, Marco Degidi, Giovanna Iezzi, Louis T. Kircos, Jack E. Lemons, Matthew Lyman, Francine Misch-Dietsh, Adriano Piatelli, Ana Emília Farias Pontes, Girish Ramaswamy, Randolph R. Resnik, W. Eugene Roberts, David P. Sarment, Antonio Scarano, Gerard M. Scortecci, Mohammed Sharawy, Miles L. Singer, J. Todd Strong, Jon B. Suzuki, and Lynn D. Terracciano-Mortilla. Their dedication to implant dentistry, and especially their friendship and personal support to me, is greatly appreciated.

My original three mentors—Ken Judy, Hilt Tatum, and Leonard Linkow—must always be acknowledged. Implant dentistry needed early pioneers to blaze the trails for the profession. Their concepts for bone grafting, implant surgery, prosthetics, implant education, and leadership created a foundation 40 years ago that allowed the profession to build the current structure we have in implant dentistry. I especially thank each of them for providing their personal continued guidance and support to me over the last 35 years.

I would also like to acknowledge James Cole, the dental illustrator who worked overtime to meet the demands of this project and never became frustrated at the many redos to satisfy our desire for an acceptable outcome. Thank you to Jill Bertelson, Heidi Cartagena, Debi Leblanc, and Lara Vandekerckhove, our personal assistants and secretaries, for typing and coordinating each chapter (especially since each chapter was rewritten 10 to 15 times).

Each book takes a personal toll on my immediate family. During this project, not only my wife Francine, but my youngest daughter Angela bore the brunt of the time and pressures to write this book. Thank you for understanding and giving up our personal time.

I would also like to acknowledge the acquisitions editor, John Dolan of Elsevier. Thank you for your experience and guidance during this process. Without your help, this project would have not come to fruition.

The third edition of *Contemporary Implant Dentistry* is also a reflection of the thousands of doctors I have trained at the Misch International Implant Institute over the last 25 years. Those doctors have contributed by the questions asked and their desire for an organized approach to help their patients. I wish to thank each of them for their professional support.

Carl E. Misch, DDS, MDS, PhD(hc)

This ebook is uploaded by dentalebooks.com

t.me/highdent www.highdentlab.com instagram.com/high_dent

This page intentionally left blank

This ebook is uploaded by dentalebooks.com

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Chapter 1

Rationale for Dental Implants

Carl E. Misch

The goal of modern dentistry is to restore the patient to normal contour, function, comfort, esthetics, speech, and health, whether by removing caries from a tooth or replacing several teeth. What makes implant dentistry unique is the ability to achieve this goal, regardless of the atrophy, disease, or injury of the stomatognathic system.[1] However, the more teeth a patient is missing, the more challenging this task becomes. As a result of continued research, diagnostic tools, treatment planning, implant designs, materials, and techniques, predictable success is now a reality for the rehabilitation of many challenging clinical situations.

The number of dental implants used in the United States increased more than tenfold from 1983 to 2002, and another fivefold from 2000 to 2005. More than 1 million dental implants are inserted each year. This number continues to increase steadily, with almost $550 million of implant products sold to North American dentists in 2005, compared with $10 million in 1983, with an expected yearly growth sustained at 12% to 15% for the next several years.[2,3] When bone grafting materials are included in implant products, it is estimated the field of implant dentistry will sell $10 billion in services to patients by 2010.[4] More than 90% of interfacing surgical specialty dentists currently provide dental implant treatment on a routine basis in their practices, 90% of prosthodontists restore implants routinely, and more than 80% of general dentists have used implants to support fixed and removable prostheses, compared with 65% 15 years ago.[5-9]

The increased need and use of implant-related treatments result from the combined effect of several factors, including (1) aging population living longer, (2) tooth loss related to age, (3) consequences of fixed prosthesis failure, (4) anatomical consequences of edentulism, (5) poor performance of removable prostheses, (6) consequences of removable partial dentures, (7) psychological aspects of tooth loss and needs and desires of aging baby boomers, (8) predictable long-term results of implant-supported prostheses, (9) advantages of implant-supported restorations, and (10) increased public awareness.

EFFECTS OF AN AGING POPULATION

According to the literature, age is directly related to every indicator of tooth loss.[10,11] Therefore the aging population is an important factor to consider in implant dentistry. When Alexander the Great conquered the ancient world, he was only 17 years old. However, life expectancy at that time was only 22 years of age. From 1000 BC to AD 1800, life span remained less than 30 years (Figure 1-1). Since 1960, the increase in life expectancy has been more rapid than at any other time in history. In 1980, 30% of the U.S. population was older than age 45, 21% was older than 50, and 11% was older than 65. In 1995, 15 years later, all these individuals were older than age 60. The group older than age 65 is projected to increase from 12%

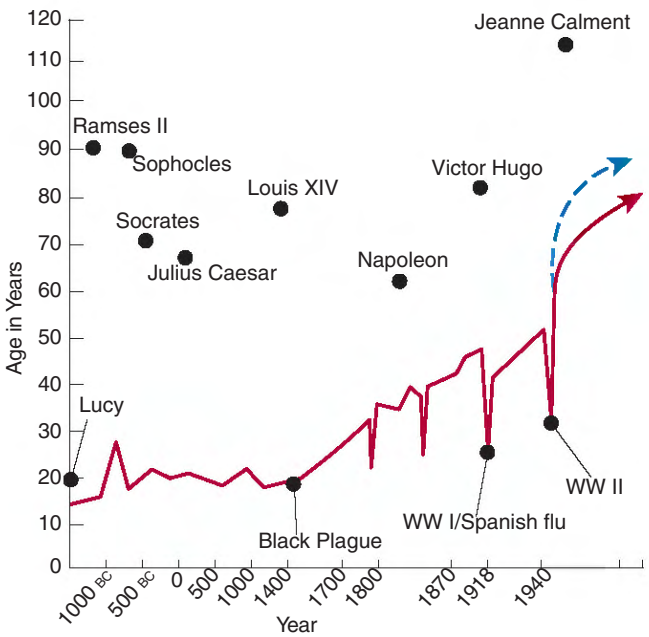


Figure 1-1 Average life expectancy remained approximately 20 to 30 years for several hundred years of human civilization. Since the late eighteenth century, there has been a gradual increase in life span. *(Redrawn from* Le Figaro Magazine, *Paris, 2004.)*

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

in 2000 to more than 20% of the population within the next 25 years[12] (Figure 1-2).

In addition, not only is the percentage of the population over 65 years increasing, but the overall population is also increasing. The population in 2000 was 282 million and is projected to increase 49% to 420 million by 2050. Considering the effect of both a population increase and a greater percentage of that population being older than age 65, a dramatic overall increase in patient numbers can be expected. In 2003, 35 million people were older than age 65. This number is expected to increase 87% by 2025, resulting in almost 70 million people being older than age 65[13] (Figure 1-3). Because older people are more likely to be missing teeth, the need for implant dentistry will dramatically increase over the next several decades.

Life expectancy has increased significantly past the age of retirement. In 1965 the average life span was 65 years; in 1990 it was 78 years. Life expectancy in 2001 was 85 years for a nonsmoking individual of normal weight.[14] A 65-year-old person can now expect to live more than 20 additional years, and an 80 year old can expect to live 9.5 more years[15-17] (Figure 1-4). Women represent two thirds of the population older than age 65.[18] It is not unusual for a 70-year-old patient to ask, "Is it worth it for me to spend $30,000 to repair my mouth at my age?" The response should be very positive, as the patient's life expectancy will extend for two more decades, and their current oral situation will normally become worse if not corrected.

Social pleasures, including dining and dating, continue throughout advanced life. In the past, geriatric dentistry meant inexpensive treatment emphasizing nonsurgical approaches. The poverty rate for the elderly, however, is less than 10%, and retiree median income has grown 8% in recent years. According to the last census, the median net worth of retirees is 15 times the net worth of those younger than age 35 and three times as high as "working families" ages 35 to 44 years.[18,19] Close to 20% of today's retirees have a net worth of more than a quarter of a million dollars. Today, the full scope of dental services for elderly patients is increasing in importance to both the public and the profession because of the increasing age of our society. Treatment alternatives that consider fixed prostheses with implant support should be presented to almost any patient. Only when all treatment options are discussed can a person's desires related to the benefit of implant dentistry be truly appreciated.

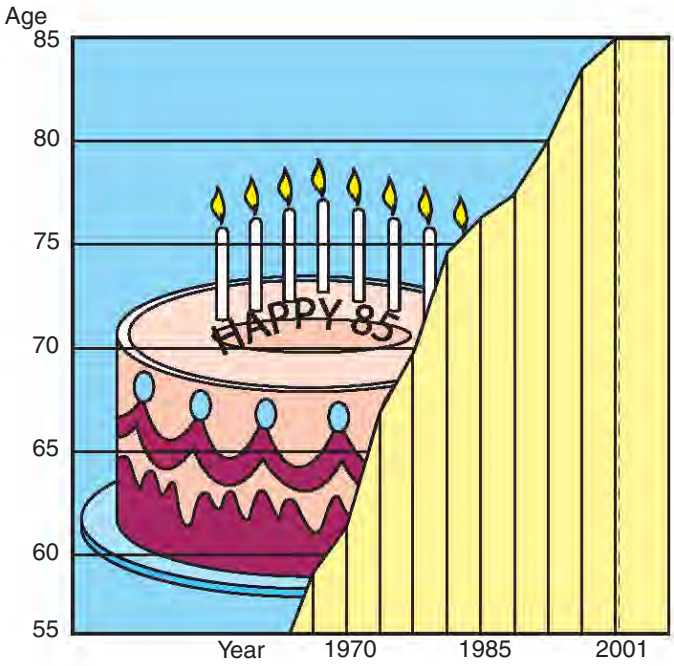


Figure 1-2 Life expectancy has increased more rapidly since 1960 than at any other time in history. Because tooth loss is directly related to age, a growing number of adults are missing teeth.

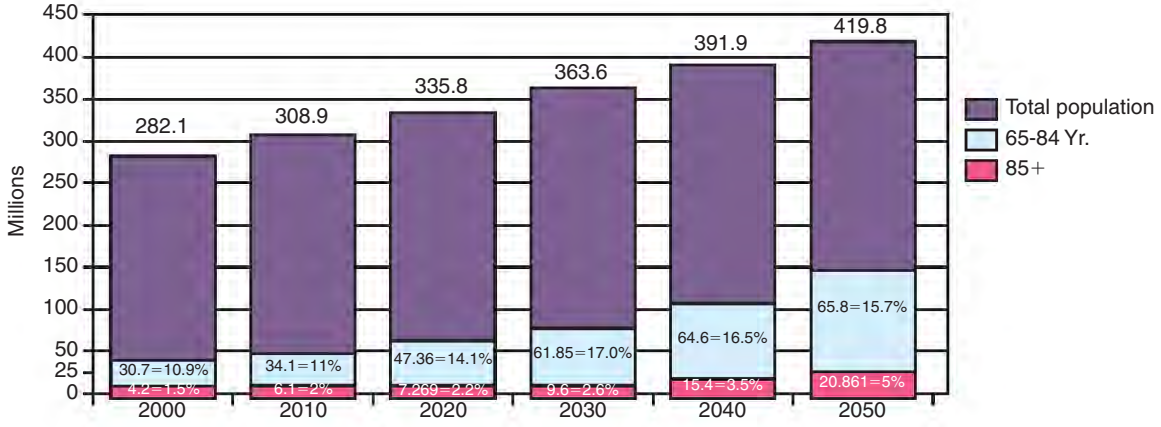


Figure 1-3 By 2050, 20.7% of the population will be older than age 65. In addition to the increasing percentage of 65 year olds, the population is also increasing. As a result, 34.9 million people were older than 65 in 2000, and 86.6 million people will reach this milestone by 2050.

This ebook is uploaded by dentalebooks.com
t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

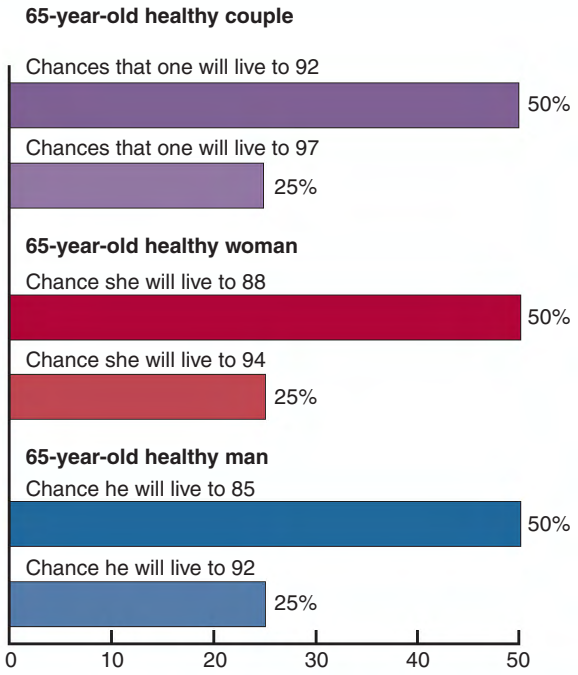


Figure 1-4 When a person reaches age 65, he or she often feels an investment in health is less appropriate. A 65-year-old healthy woman will live 23 more years 50% of the time, and 29 more years 25% of the time. Her present oral condition will become worse during this extended time frame if treatment is not rendered.

Figure 1-5 A posterior missing tooth is a frequent occurrence in a general practice. Almost 80% of the time the adjacent teeth are unrestored or have minimum restoration.

Dental services for elderly patients clearly represent a growing demand for the dental profession. In 2000, 28.8% of all income from a dentist came from patients age 60 and older—a group that represented only 12% of income in 1988. When the dentist is older than age 40, income from those older patients represents 64.3% of the dentist's income, whereas in 1988 it was 30.3%.[20] Clearly, the demographics of our population have dramatically changed the economics of dental practice.

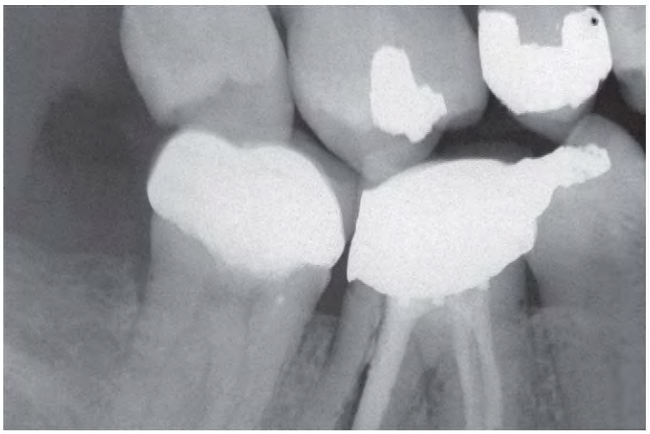


Figure 1-6 A posterior endodontically treated tooth has an increased risk of fracture or failure of the endodontic therapy.

AGE-RELATED TOOTH LOSS

Single-Tooth Edentulism

The posterior regions of the mouth often require the replacement of a single tooth[21-23] (Figure 1-5). The first molars are the first permanent tooth to erupt in the mouth and, unfortunately, are often the first teeth lost as a result of decay, failed endodontic therapy or fracture (usually after endodontics). They are important teeth for maintenance of the arch form and proper occlusal schemes. In addition, the adult patient often has one or more crowns, as a consequence of previous larger restorations required to repair the integrity of the tooth. Longevity reports of crowns have yielded very disparate results. The mean life span at failure has been reported as 10.3 years. Other reports range from a 3% failure rate at 23 years to a 20% failure rate at 3 years. The primary cause of failure of the crown is caries followed by endodontic therapy.[24-33] The tooth is at risk for extraction as a result of these complications, which are the leading causes of single posterior tooth loss in the adult (Figure 1-6). It has been estimated that a $425 crown for a 22-year-old patient will cost $12,000 during the patient's lifetime to replace and/or repair.[26]

Fixed Partial Dentures

The most common choice to replace a posterior single tooth is a three-unit fixed partial denture (FPD). This type of restoration can be fabricated within 1 to 2 weeks and satisfies the criteria of normal contour, comfort, function, esthetics, speech, and health. Because of these benefits, FPD has been the treatment of choice for the last 6 decades. Bone and soft tissue considerations in the missing tooth site are few. Every dentist is familiar with the procedure, and it is widely accepted by the profession, patients, and dental insurance companies.

In the United States, 70% of the population is missing at least one tooth. Almost 30% of the 50 to 59 year olds examined in a U.S. National Survey exhibited

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

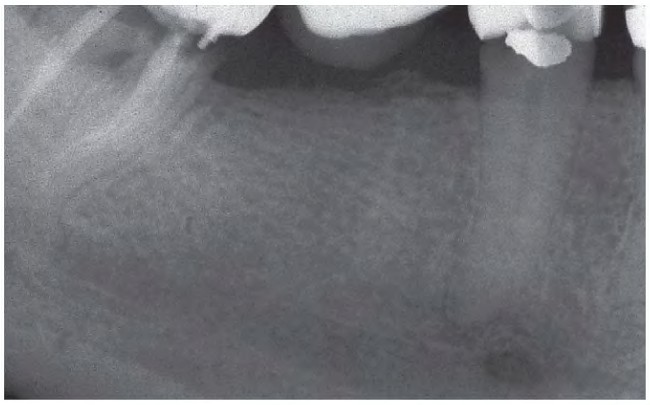


Figure 1-7 A three-unit fixed partial denture most often fails because of caries on a crown margin, next to the pontic.

either single or multiple edentulous spaces bordered by natural teeth. In 1990, more than 4 million FPDs were placed in the United States.[23-25] Treatments to replace single teeth with a fixed prosthesis represent 7% of the annual dental reimbursement from insurance companies and more than $3 billion each year. Only one third of our population in the United States has dental insurance and of those who do, only 50% of treatment costs are reimbursed. Hence, the entire three- to four-unit FPD costs in the United States may approach more than $10 billion each year.

A three-unit FPD also presents survival limitations to the restoration and, more importantly, to the abutment teeth.[30,32] In an evaluation of 42 reports since 1970, Creugers et al. calculated a 74% survival rate for FPDs for 15 years.[29] Mean life spans of 9.6 to 10.3 years have been reported by Walton et al.[31] and Schwartz et al.,[24] respectively. However, reports are very inconsistent, with as little as 3% loss over 23 years to 20% loss over 3 years.[23-32] Caries and endodontic failure of the abutment teeth are the most common causes of prostheses failure.[31,32] Up to 15% of abutment teeth for an FPD require endodontic therapy, compared with 3% of nonabutment teeth that have crown preparations. The long-term periodontal health of the abutment teeth, including bone loss, may also be at greater risk.[29]

Unfavorable outcomes of FPD failure include both the need to replace the failed prosthesis and the loss of an abutment and the need for additional pontics and abutment teeth in the replacement bridge. The abutment teeth of an FPD may be lost at rates as high as 30% within 14 years.[30] Approximately 8% to 12% of the abutment teeth holding an FPD are lost within 10 years. The most common reason for single tooth loss is endodontic failure or fracture of a tooth (usually after endodontic therapy). Because 15% of abutment teeth require endodontics, and root canal therapy may be 90% successful at the 8-year mark, abutment teeth are at increased risk of loss. In addition, abutment teeth are more prone to caries when splinted together with

Box 1-1 Single-Tooth Replacement—Fixed Partial Denture

- Estimated mean life span of FPD (50% survival) reported at 10 years
- Caries most common cause of FPD failure
- 15% of FPD abutments require endodontics
- Failure of abutment teeth of FPD 8% to 12% at 10 years and 30% at 15 years
- 80% of teeth adjacent to missing teeth have no or minimal restoration

FPD, Fixed partial denture.

Figure 1-8 A single-tooth implant in the posterior region of the mouth is most often the treatment of choice.

an intermediary pontic. Individual crowns have decay rates below 2%; however, the risk of caries in abutment teeth is approximately 20%, mainly because the pontic region acts as a plaque reservoir. The carious lesion at the crown margin may cause structural failure, even if endodontic treatment is possible (Figure 1-7).

Almost 80% of abutments prepared for a three-unit FPD have no existing or only minimal restorations.[34] Rather than removing sound tooth structure and crowning two or more teeth—thus increasing the risk of decay and endodontic therapy (and splinting teeth together with pontics, which have the potential to cause additional tooth loss)—a dental implant may replace the single tooth (Box 1-1).

Single-Tooth Implants

A treatment option to replace a posterior single missing tooth is a single-tooth implant (Figure 1-8). For years, patients were advised to put their desires aside and accept the limitations of an FPD. However, many feel the most natural method to replace a tooth is to use an implant, rather than preparing adjacent teeth and joining them together with a prosthesis. The primary reasons for suggesting the FPD were its clinical ease and reduced treatment time. However, if this concept were expanded, extractions would replace endodontics and dentures could even replace orthodontics. The primary

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

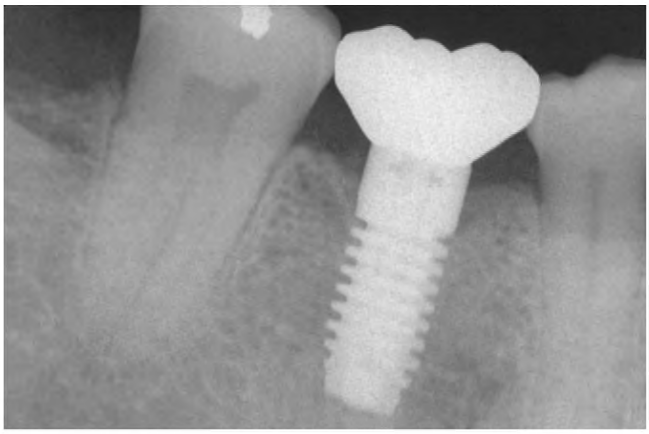


Figure 1-9 A single-tooth implant to replace a missing tooth has the highest success rate, and the adjacent teeth are less likely to decay, require endodontics, or result in additional tooth loss.

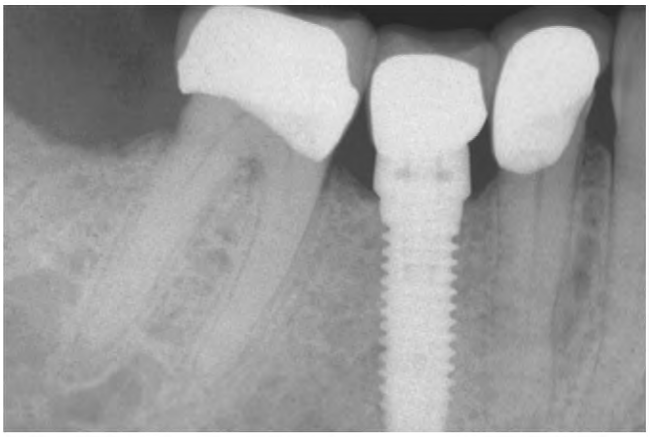


Figure 1-10 Even when teeth adjacent to the missing tooth require crowns, an implant is the treatment of choice because single crowns on teeth adjacent to implants have fewer complications and increased longevity compared with abutments for a three-unit fixed partial denture.

Box 1-2 Single-Tooth Implants—Advantages

- High success rates (above 97% for 10 years)
- Decreased risk of caries of adjacent teeth
- Decreased risk of endodontic problems on adjacent teeth
- Improved ability to clean the proximal surfaces of the adjacent teeth
- Improved esthetics of adjacent teeth
- Improved maintenance of bone in the edentulous site
- Decreased cold or contact sensitivity of adjacent teeth
- Psychological advantage
- Decreased abutment tooth loss

reason to suggest or perform a treatment should not be related to treatment time or difficulty of the procedure, but instead should consider the best possible long-term solution for each individual.

From 1993 to the present, single-tooth implant survival reports have validated this procedure as the most predictable method of tooth replacement. There are more refereed reports in the literature for single-tooth implant replacement than for any other method of tooth replacement,[35-47] and all reports demonstrate a higher survival rate for single-tooth implants. Goodacre et al. performed a Medline literature review from 1980 to 2001 and found the single-tooth implant success rate to be in the range of 97%—higher than any other implant restoration.[35] In 1995, Haas et al. reported on 76 single-tooth implants over a 6-year period and found a 97% survival rate and a 2.6% implant loss.[38] Fugazzotto evaluated 1472 implants over a 13-year period and found a 97% survival rate during that period.[39]

Although posterior single-tooth replacement is a relatively new treatment alternative, many studies have been published since 1990, and survival rates reported range from a low of 94.6% to a high of 100% for 1 to 15 years.[35-45] The median of these reports is a 2.8% implant loss with a mode of 5 years. In comparison, FPD failure rates may be as high as 20% within 3 years, and 50% rates at 10 years are expected. As a result, the single-tooth implant exhibits the highest survival rates presented for single-tooth replacement. As important, no reports indicate a loss of an adjacent tooth, which is a considerable advantage. On the other hand, the longevity of the implant crown has not been adequately determined, as these reports do not extend as long as those of other treatment options.

Despite some limitations and obvious clinical challenges, the single-tooth implant represents the treatment of choice from both a health and value standpoint. When adjacent teeth are healthy, or when the patient refuses their preparation for the fabrication of a traditional three-unit fixed partial restoration, a posterior single-tooth implant is an excellent solution (Figure 1-9). Health-related advantages of this modality over a fixed partial restoration are listed in Box 1-2 and include a decreased risk of decay and periodontal disease, decreased risk of abutment tooth loss from endodontic failure or caries, and improved esthetics (Figure 1-10). Psychological advantages, especially with congenitally missing teeth or the loss of a tooth after a crown restoration, are significant as well. These advantages are so significant to the health and periodontal condition of the adjacent teeth and maintenance of the arch form that the single-tooth implant has become the treatment of choice in most situations. Economical considerations may play in disfavor of the implant restoration only during the first 7 years. The single-tooth implant becomes more advantageous economically, not only for health considerations, after the break-even point of 7 years, at which time the patient will not need a replacement prosthesis. The savings will offset the initial higher cost.[47]

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Partial Edentulism

The prevalence of partial edentulism is also of interest because a growing number of implants are used in these patients. A 1988 to 1991 survey in the United States found that only 30% of these patients had all 28 teeth. Partially dentate patients had an average of 23.5 teeth.[10,23,48] In the 1999 to 2002 follow-up survey, the average number of missing teeth was fewer than two of 28 teeth for the 20- to 39-year-old group. However, this number rapidly increased to an average of nine teeth missing in adults older than age 60. Partially edentulous seniors older than age 60 age have lost an average of 10 teeth, with older seniors having lost three more teeth than the younger seniors. Statistics for partial edentulism are similar for both men and women. The greatest transition from an intact dental arch to a partially edentulous condition in the 1987 study occurred in the 35- to 54-year-old group.[20,23] The growth rate of this baby boomer portion of the population was approximately 30% in 1982 and is continuing to increase, more than any other age group. For example, in 1982 the baby boomer age group (born from 1946 to 1964) increased from 39 million Americans to 79 million in 2005. Although the number of teeth missing per patient may seem to decrease, the overall number of missing teeth will continue to increase. Therefore the need for implant services in partially edentulous patients will dramatically increase during the next several decades.[15]

The most common missing teeth are molars.[22] Partial free-end edentulism is of particular concern because in these patients, teeth are often replaced with removable partial prostheses. This condition is rarely found in persons younger than age 25. Mandibular free-end edentulism is greater than its maxillary counterpart in all age groups. Unilateral free-end edentulism is more common than bilateral edentulism in both maxillary and mandibular arches in the younger age groups (ages 25 to 44). About 13.5 million persons in these younger age groups have free-end edentulism in either arch.

In 45- to 54-year-old patients, 31.3% have mandibular free-end edentulism and 13.6% have free-end edentulism in the maxillary arch. Approximately 9.9 million persons in the 45- to 54-year-old group have at least one free-end edentulous quadrant, and almost half of these have bilateral partial edentulism.[11] The pattern of posterior edentulism evolves in the 55- to 64-year-old group, in which 35% of mandibular arches show free-end edentulism, compared with 18% of maxillary arches. As a result, approximately 11 million individuals in this age group are potential candidates for implants. An additional 10 million show partial free-end edentulism at age 65 or older. Additional studies have documented that in the population of noninstitutionalized U.S. civilians, 1 of 5 had a removable prosthesis of some type.[49-51] The total number of potential patients in the U.S. survey with at least one quadrant of posterior missing teeth is more than 44 million people.[11] If each of these arches requires three implants to support a fixed prosthesis, 132 million implants added to the 192 million for edentulous patients would be required.

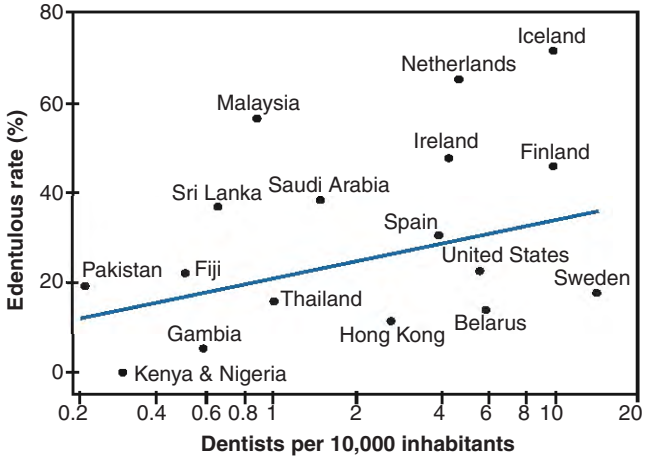


Figure 1-11 The incidence of complete edentulism averages 20% of the adult population around the world. It is interesting to note that often the greater the number of dentists per population, the greater the rate of edentulism. *(Adapted from Mojon P: The world without teeth: demographic trends. In Feine JS, Carlsson GE, editors:* Implant overdentures: the standard of care for edentulous patients, *Carol Stream, Ill, 2003, Quintessence.)*

Total Edentulism

Edentulism is not an eventual, healthy occurrence in an adult population. Rather it is most often the result of repeated tooth extractions from the combined pathologic processes of dental caries, periodontal disease, or a method to reduce the costs associated with dental treatment.[52-54] Similar to other pathologic outcomes of disease, the occurrence of total loss of teeth is directly related to the age of the patient. The rate of edentulism increases at 4% per 10 years in early adult years and increases to more than 10% per decade after age 70.[54]

The average total edentulous rate around the world is 20% at age 60, although there is wide disparity from the countries with the highest and lowest rates[54] (Figure 1-11). For example, from the 65- to 74-year age group, the total edentulous rate in Kenya and Nigeria was 0%, whereas the Netherlands and Iceland have a 65.4% and 71.5% rate, respectively. The edentulous Canadian rate was 47% at 65 to age 69 and 58% from ages 70 to 98 (with Quebec at 67% for those older than age 65, compared with Ontario with a 41% rate). One of the main factors influencing total edentulism was the level of education. In data from the Canadian Health Promotion Survey from 1990, the least educated had an edentulous rate of 50%, whereas those with a college education had a low 4% rate.[55] The United States showed a similar pattern in the period 1988 to 1994, with an edentulous rate of 22% for those with less than 8 years of education, 12% for those with 9 to

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

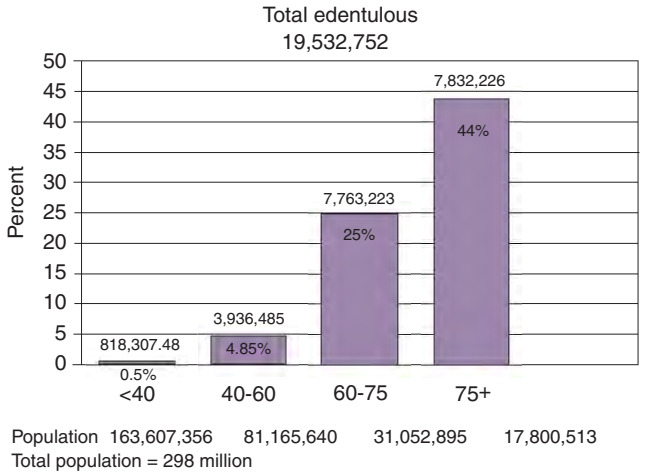


Figure 1-12 The U.S. population completely edentulous rate ranges from 5% for 40 year olds to 44% for those older than age 75. As a result, 20 million people (10.5% of the population) in the United States have no teeth. An additional 12 million people (7% of the adult population) have no maxillary teeth opposing at least some mandibular teeth.

11 years of school, 8% for those with 12 years of school, and 5% for individuals with more than 12 years of education.[48] Income is often related to education and may also play a role in the rate of edentulism.

In the National Institute of Dental Research national surveys, the occurrence of total edentulism (absence of teeth) of a single arch (35 times more frequent in the maxilla) was slight in the 30- to 34-year-old age group, but it increased at around age 45 to 11% and then remained constant after 55 years at approximately 15% of the adult population. A total of approximately 12 million individuals in the United States have edentulism in one arch, representing 7% of the adult population overall.

The 1999 to 2002 survey found that total edentulism of both arches occurred in 7.7% of the adult population in the United States, or almost 20 million people.[48] The present younger population is benefiting from today's advanced knowledge and restorative techniques. Edentulism has been noted in 5% of employed adults ages 40 to 44, gradually increasing to 26% at age 65, and almost 44% in seniors older than age 75[10] (Figure 1-12). As expected, older persons are more likely to be missing all their teeth. Gender was not found to be associated with tooth retention or tooth loss once adjustments were made for age.

The percentages of one- or two-arch edentulism translate into more than 30 million people or about 17% of the entire U.S. adult population.[17] To put these numbers in perspective, 30 million people represent approximately the entire U.S. African American population, or the whole population of Canada. Although the edentulism rate is decreasing every decade, the elderly population is rising so rapidly that the adult population in need of one or two complete dentures will actually *increase* from 33.6 million adults in 1991 to 37.9 million adults in 2020. The total number of edentulous arches is estimated at 56.5 million in 2000, 59.3 million in 2010, and 61 million in 2020.[56] Complete edentulism, therefore, remains a significant concern, and affected patients often require implant dentistry to solve several related problems. If four implants were used to help support each complete edentulous arch in 2000, a total of 226 million implants would be required. Yet only 1 million implants were inserted for all patient treatment (partially or completely edentulous) each year. Almost 70% of dentists spend less than 1% to 5% of their treatment time on edentulous patients, leaving a great unfulfilled need for implant dentistry.

When the partially edentulous figures are added to the complete edentulous percentages, almost 30% of the adult U.S. population are candidates for a complete or partial removable prosthesis. The need for additional retention, support, and stability and the desire to eliminate a removable prosthesis are common indications for dental implants. As a result, 74 million adults (90 million arches) are potential candidates for dental implants. Because a minimum of five appointments is required to implant and restore a patient, every U.S. dentist would need approximately 20 appointments every month for 20 years to treat the present posterior partial and complete edentulous population with implant-supported prostheses. The population's evolution to an increased average age, combined with the existing population of partially and completely edentulous patients, guarantee implant dentistry's future for several generations of dentists.

ANATOMICAL CONSEQUENCES OF EDENTULISM

Consequences on the Bony Structures

Basal bone forms the dental skeletal structure, contains most of the muscle attachments, and begins to form in the fetus before teeth develop. Alveolar bone first appears when Hertwig's root sheath of the tooth bud evolves[57] (Figure 1-13). The alveolar bone does not form in the absence of primary or secondary tooth development. The close relationship between the tooth and the alveolar process continues throughout life. Wolff's law (1892) states that bone remodels in relationship to the forces applied.[58] Every time the function of bone is modified, a definite change occurs in the internal architecture and external configuration.[59] In dentistry, the consequences of complete edentulous and remaining bone volume was noted by Misch in 1922, where he described the skeletal structure of a 90-year-old woman without teeth for several decades.[60]

Bone needs stimulation to maintain its form and density. Roberts et al.[61] report that a 4% strain to the skeletal system maintains bone and helps balance the resorption and formation phenomena. Teeth transmit

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Figure 1-13 The alveolar bone forms as a result of the tooth root formation. When no tooth root is present, the alveolar process does not form (i.e., ectodermal dysplasia when partial or complete anodontia of both primary and secondary teeth occurs).

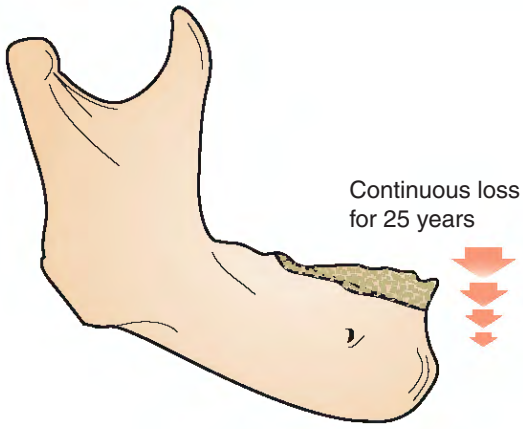


Figure 1-14 After the initial extraction of teeth, the average first-year bone loss is more than 4 mm in height and 30% in crestal bone width. Although the rate of bone loss is slower after the first year, the bone loss is continuous throughout life.

compressive and tensile forces to the surrounding bone. These forces have been measured as a piezoelectric effect in the imperfect crystals of durapatite that compose the inorganic portion of bone.[62] When a tooth is lost, the lack of stimulation to the residual bone causes a decrease in trabeculae and bone density in the area, with loss in external width, then height, of the bone volume.[63] There is a 25% decrease in width of bone during the first year after tooth loss and an overall 4-mm decrease in height during the first year after extractions for an immediate denture.[64] In a longitudinal 25-year study of edentulous patients, lateral cephalograms demonstrated continued bone loss during this time span; a fourfold greater loss was observed in the mandible[65] (Figure 1-14). However, because initially the mandible height is twice that of the maxilla, maxillary bone loss is also significant in the long-term edentulous patient.

A tooth is necessary to the development of alveolar bone, and stimulation of this bone is required to maintain its density and volume. A removable denture (complete or partial) does not stimulate and maintain bone; rather, it accelerates bone loss. The load from mastication is transferred to the bone surface only, not the whole bone. As a result, blood supply is reduced and total bone volume loss occurs.[66] This issue, which is of utmost importance, has been observed but not addressed in the past by traditional dentistry. Doctors most often overlook the insidious bone loss that will occur after tooth extraction. The patient is often not educated about the anatomical changes and the potential consequences of continued bone loss. The bone loss accelerates when the patient wears a poorly fitting soft tissue–borne prosthesis. Patients do not understand that bone is being lost over time and at a greater rate beneath poorly fitting dentures (Figure 1-15). Patients do not return for regular visits for evaluation of their condition; instead, they return after several years when denture teeth are worn down or can no longer be tolerated. Hence the traditional method of tooth replacement often affects

Figure 1-15 Atwood described six different stages of resorption in the anterior mandible. Stage 1 represents the tooth and surrounding alveolar process and basal bone. Stages II and III illustrate the initial residual ridge after tooth loss. Stages IV to VI primarily describe a continuous loss in length of anterior residual bone.

bone loss in a manner not sufficiently considered by the doctor and the patient. The doctor should inform the patient that a denture replaces more bone and soft tissue than teeth, and every 5 years a reline or new denture is suggested to replace the additional bone loss by atrophy (Figure 1-16).

Preventive dentistry has traditionally emphasized methods to decrease tooth loss. No predictable therapy had been accepted by the profession to avoid the bone changes resulting from tooth loss. Today the profession must consider the loss of both teeth and bone. The loss of teeth causes remodeling and resorption of the surrounding alveolar bone and eventually leads to atrophic edentulous ridges. Although the patient often is not aware or informed of the potential consequences, over time consequences will occur. The rate and amount of bone loss may be influenced by such things as gender, hormones, metabolism, parafunction, and ill-fitting dentures. Yet almost 40% of denture wearers have been wearing an ill-fitting prosthesis for more

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

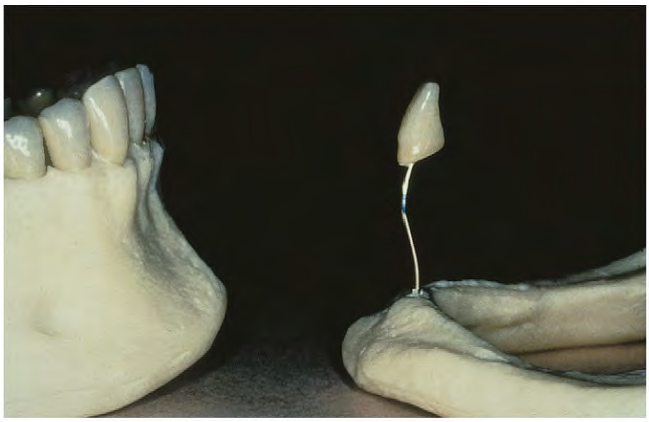


Figure 1-16 Loss of bone height in the mandible may be measured in inches and often is ignored. Such bone loss is often more significant than the bone loss (in millimeters) from periodontal disease. The patient should understand that a denture often replaces more bone than teeth to restore the proper dimensions of the face.

Figure 1-17 A patient with moderate to severe atrophy usually has the intraoral muscles above the residual ridge, including the floor of the mouth and the mentalis and buccinator muscles.

Box 1-3 Consequences of Bone Loss in Fully Edentulous Patients

- Decreased width of supporting bone
- Decreased height of supporting bone
- Prominent mylohyoid and internal oblique ridges with increased sore spots
- Progressive decrease in keratinized mucosa surface
- Prominent superior genial tubercles with sore spots and increased denture movement
- Muscle attachment near crest of ridge
- Elevation of prosthesis with contraction of mylohyoid and buccinator muscles serving as posterior support
- Forward movement of prosthesis from anatomical inclination (angulation of mandible with moderate to advanced bone loss)
- Thinning of mucosa, with sensitivity to abrasion
- Loss of basal bone
- Paresthesia from dehiscent mandibular neurovascular canal
- More active role of tongue in mastication
- Effect of bone loss on esthetic appearance of lower third of face
- Increased risk of mandibular body fracture from advanced bone loss
- Loss of anterior ridge and nasal spine, causing increased denture movement and sore spots during function

than 10 years.[67] Patients wearing dentures day and night place greater forces on the hard and soft tissues, which accelerates bone loss. Nonetheless, 80% of dentures are worn both day and night.[68]

Atrophic edentulous ridges are associated with anatomical problems that often impair the predictable results of traditional dental therapy (Figure 1-17). Several of these anatomical problems are listed in Box 1-3. The loss of bone first causes decreased bone width. The remaining narrow residual ridge often causes discomfort when the thin overlying tissues are loaded under a soft tissue–borne removable prosthesis. The continued atrophy of the posterior mandible eventually causes prominent mylohyoid and internal oblique ridges covered by thin, movable, unattached mucosa. The anterior residual alveolar process also continues to resorb, and the superior genial tubercles (which are 20 mm below the crest of bone when teeth are present) eventually become the most superior aspect of the anterior mandibular ridge. There is little to prevent the prosthesis from moving forward against the lower lip during function or speech. This condition is further compromised by the vertical movement of the distal aspect of the prosthesis during contraction of the mylohyoid and buccinator muscles and the anterior incline of the atrophic mandible compared with that of the maxilla.[69]

Loss of bone in the maxilla or mandible is not limited to alveolar bone; portions of the basal bone may be resorbed also (Figure 1-18), especially in the posterior aspect of the mandible where severe resorption may result in more than 80% bone loss.[66] The contents of the mandibular canal or mental foramen eventually become dehiscent and serve as part of the support area of the prosthesis.[70] As a result, acute pain and transient to permanent paresthesia of the areas supplied by the mandibular nerve are possible. The body of the mandible also is at increased risk of fracture, even under very-low-impact forces (Figure 1-19). The mandibular fracture causes the jaw to shift to one side and makes stabilization and an esthetic result most difficult to obtain during treatment of the fracture.

The complete anterior ridge and even the nasal spine may be resorbed in the maxilla, causing pain and an increase in maxillary denture movement during function.[67] Masticatory forces generated by short facial types (brachiocephalics) can be three to four times that of long facial types (dolichocephalics). Short facial–

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

A B

Figure 1-18 **A,** Lateral cephalogram of a patient demonstrates the restored vertical dimension of occlusion with a denture. However, because of the advanced basal bone loss in the mandible, the superior genial tubercles are positioned above the residual anterior ridge. The body of the mandible is only a few millimeters thick, and the mandibular canal is completely dehiscent (one body is superimposed on top of the other in this view). In the maxillary anterior ridge, only the nasal spine remains (not the original alveolar ridge), and the posterior maxillary bone is paper thin because of basal bone loss at the crest and the pneumatization of the maxillary sinus. **B,** A denture may restore the vertical dimension of the face, but the bone loss of the jaws can continue until the basal bone is paper thin in the maxilla and the mandible becomes the size of a toothpick (this is a different patient from the one in **A**).

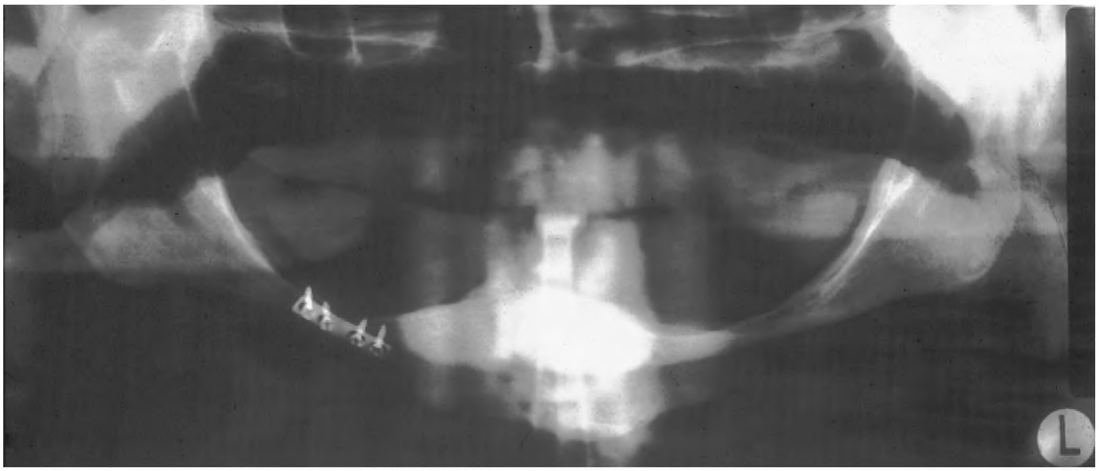


Figure 1-19 Resorption of an edentulous mandible may result in dehiscence of the mandibular canal and associated paresthesia. The patient may fear that a tumor is growing against the nerves. The body of the mandible may continue to resorb until minor trauma causes fracture (e.g., during mastication, the bump of a baby's head held closely to the face, or an accidental bump from the elbow).

type patients are at increased risk for developing severe atrophy[71,72] (see Box 1-3).

Many of these similar conditions exist in the partially edentulous patient wearing a removable soft tissue–borne prosthesis (Figure 1-20). In addition, the natural abutment teeth, on which direct and indirect retainers are designed, must submit to additional lateral forces. Because these teeth are often compromised by deficient periodontal support, many partial dentures are designed to minimize the forces applied to them. The result is an increase in mobility of the removable prosthesis and greater soft tissue support. These conditions protect the remaining teeth but accelerate the bone loss in the edentulous regions.[73]

Soft Tissue Consequences

As bone loses width, then height, then width and height again, the attached gingiva gradually decreases. A very thin attached tissue usually lies over the advanced atrophic mandible or is absent entirely. The increasing zones of unkeratinized gingiva are prone to abrasions caused by the overlaying prosthesis. In addition, unfavorable high muscle attachments and

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Figure 1-20 Panoramic radiograph demonstrates that more bone is maintained below the anterior periodontal diseased teeth compared with the basal bone loss and severe atrophy of the posterior edentulous segment. Wearing of a mandibular Class I removable partial denture has escalated the posterior bone loss.

Box 1-4 Soft Tissue Consequences of Edentulism

- Attached, keratinized gingiva is lost as bone is lost
- Unattached mucosa for denture support causes increased soft spots
- Thickness of tissue decreases with age, and systemic disease causes more sore spots for dentures
- Tongue increases in size, which decreases denture stability
- Tongue has more active role in mastication, which decreases denture stability
- Decreased neuromuscular control of jaw in the elderly

hypermobile tissue often complicate the situation. The thickness of the mucosa on the atrophic ridge is also related to the presence of systemic disease and the physiologic changes that accompany aging. Conditions such as hypertension, diabetes, anemia, and nutritional disorders have a deleterious effect on the vascular supply and soft tissue quality under removable prostheses. These disorders result in a decreased oxygen tension to the basal cells of the epithelium. Surface cell loss occurs at the same rate, but the cell formation at the basal layer is slowed. As a result, thickness of the surface tissues gradually decreases. Therefore, sore spots and uncomfortable removable prostheses result.

The tongue of the patient with edentulous ridges often enlarges to accommodate the increase in space formerly occupied by teeth. At the same time, it is used to limit the movements of the removable prostheses, and takes a more active role in the mastication process. As a result, the removable prosthesis decreases in stability. The decrease in neuromuscular control, often associated with aging, further compounds the problems of traditional removable prosthodontics. The ability to wear a denture successfully may be largely a learned, skilled performance. The aged patient who recently became edentulous may lack the motor skills needed to adjust to the new conditions (Box 1-4).

Esthetic Consequences

The facial changes that naturally occur in relation to the aging process can be accelerated and potentiated by the loss of teeth. Several esthetic consequences result from the loss of alveolar bone (Figures 1-21, 1-22). A decrease in facial height from a collapsed vertical dimension causes several facial changes. The loss of labiomental angle and deepening of vertical lines in the area create a harsh appearance. As the vertical dimension progressively decreases, the occlusion evolves toward a pseudo Class III malocclusion. As a result, the chin rotates forward and creates a prognathic facial appearance (Figure 1-23). These conditions result in a decrease in the horizontal labial angle at the corner of the lips; the patient appears unhappy when the mouth is at rest. Short facial types suffer higher bite forces, greater bone loss, and more dramatic facial changes with edentulism compared with others.

A thinning of the vermillion border of the lips results from the poor lip support provided by the prosthesis and the loss of muscle tone; its retruded position is related to the loss of premaxilla ridge and the loss of tonicity of the muscles involved in facial

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

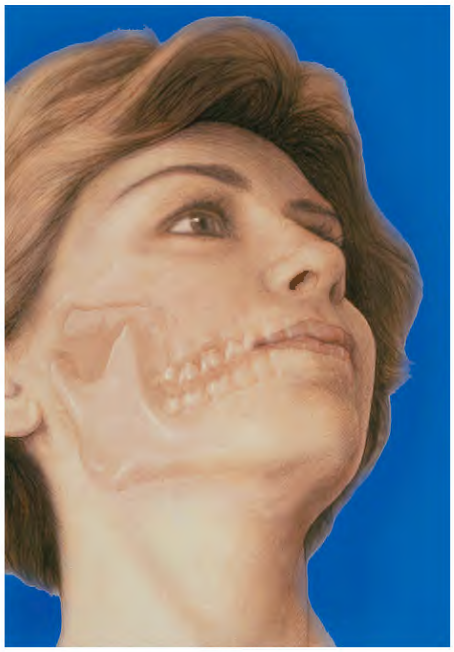


Figure 1-21 Esthetics of the inferior third of the face are related to the position of the teeth and include the muscles that attach to the bone.

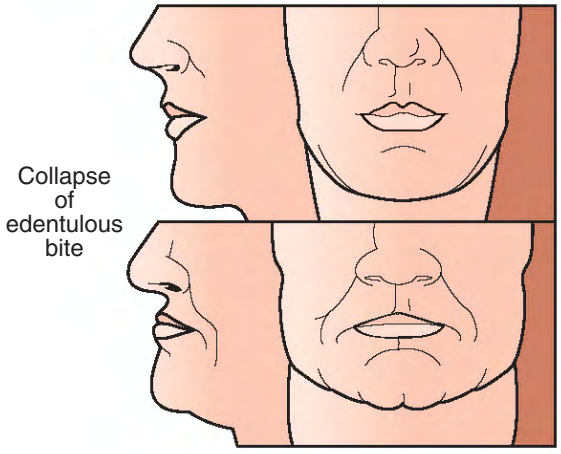


Figure 1-22 A patient often wears a denture for more than 15 years. The loss of bone height during this time is associated with many extraoral facial changes. The loss of vertical dimension results in many changes, including closed bite, a mandible that rotates forward, a receding maxilla, reverse smile line, increased number and depth of lines in the face, more acute angle between the nose and the face, loss of vermillion border in the lips, jowls, and witch's chin from loss of muscle attachment.

expression. In a study of 179 white patients at different stages of jaw atrophy, the collapse of the lips and circumoral musculature were evaluated by Sutton et al. The contraction of the orbicularis oris and buccinator muscles in the patient with moderate to advanced bone atrophy displaces the modiolus and muscles of facial expression medially and posteriorly. As a result, a narrowing of the commissure, inversion of the lips, and hollowing of the cheeks were very characteristic findings[74] (Figure 1-24). Women often use one of two

Figure 1-23 Loss of bone height can lead to a closed bite with rotation of the chin anterior to the tip of the nose.

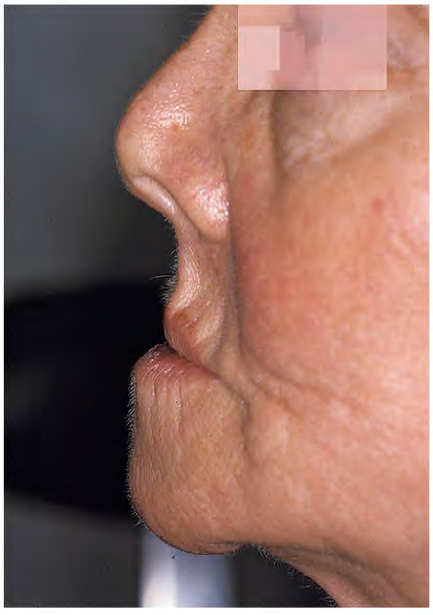
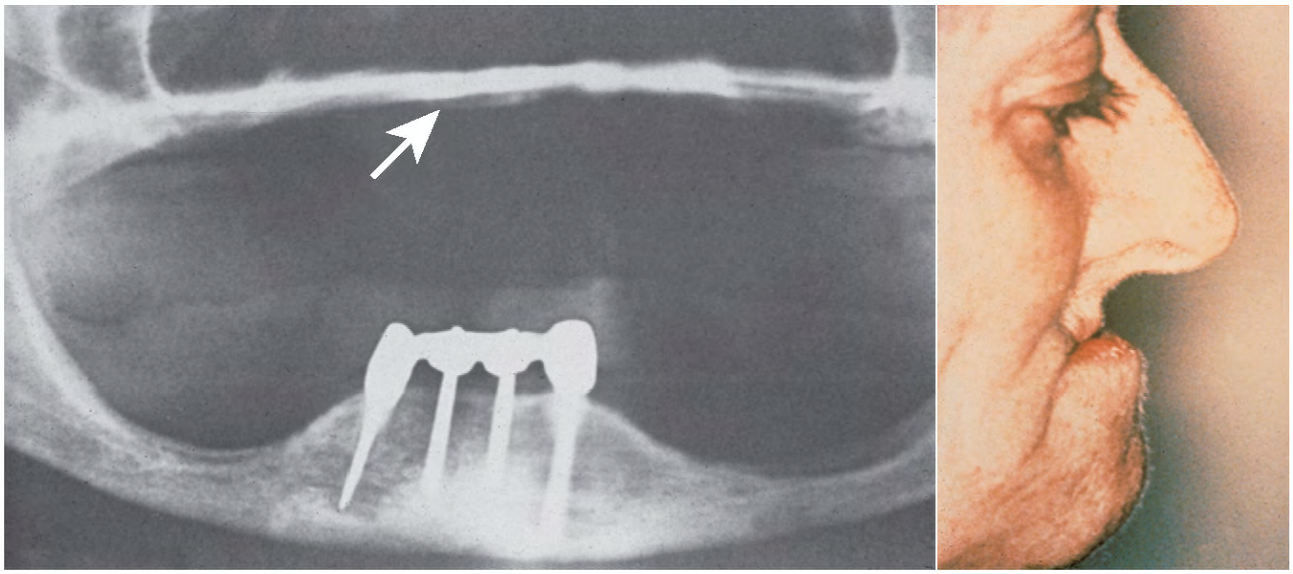


Figure 1-24 This patient has severe bone loss in the maxilla and mandible. Although she is wearing her 15-year-old dentures, the facial changes are significant. The loss of muscle attachments lead to ptosis of the chin (witch's chin), loss of vermillion border (lipstick is applied to the skin), reverse lip line (decrease in horizontal angles), increased vertical lines in the face and lips, increased lip angle under the nose, and a lack of body in the masseter and buccinator muscles.

techniques to hide this cosmetically undesirable appearance: either no lipstick and minimal makeup, so that little attention is brought to this area of the face; or lipstick drawn on the skin over the vermilion border to give the appearance of fuller lips. A deepening of the nasolabial groove and an increase in the depth of other vertical lines in the upper lip are related to normal

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت



A B

Figure 1-25 **A,** Panoramic radiograph of a 68-year-old female. The maxillary arch has severe atrophy and almost complete basal bone loss, including most of the nasal spine. Implants were inserted in the anterior mandible 15 years before this film. The anterior bone has been maintained. The posterior mandible has continued to resorb, and the mandibular canal is dehiscent on one side. **B,** Profile view. Note the maxillary bone loss effect: the lack of vermillion border of the lip, deep labial folds, and the columella-philtrum angle. Yet the lower lip has a normal vermillion border and the muscles to the lower jaw are still attached, providing a normal contour.

Box 1-5 Esthetic Consequences of Bone Loss

- Decreased facial height
- Loss of labiomental angle
- Deepening of vertical lines in lip and face
- Chin rotates forward—gives a prognathic appearance
- Decreased horizontal labial angle of lip—makes patient look unhappy
- Loss of tone in muscles of facial expression
- Thinning of vermillion border of the lips from loss of muscle tone
- Deepening of nasolabial groove
- Increase in columella-philtrum angle
- Increased length of maxillary lip, so less teeth show at rest and smiling—ages the smile
- Ptosis of buccinator muscle attachment—leads to jowls at side of face
- Ptosis of mentalis muscle attachment—leads to "witch's chin"

aging but are accelerated with bone loss. This usually is accompanied by an increase in the columella-philtrum angle. This can make the nose appear larger than if the lip had more support (Figure 1-25). Men often grow a moustache to minimize this effect. The maxillary lip naturally becomes longer with age as a result of gravity and loss of muscle tone, resulting in less of the anterior teeth shown when the lip is at rest. This has a tendency to "age" the smile, because the younger the patient, the more the teeth show in relation to the upper lip at rest or when smiling. Loss of muscle tone is accelerated in the edentulous patient, and the lengthening of the lip occurs at a younger age.

The attachments of the mentalis and buccinator muscles to the body and symphysis of the mandible also are affected by bone atrophy. The tissue sags, producing "jowls" or a "witch's chin." This effect is cumulative because of the loss in muscle tone with the loss of teeth, the associated decrease in bite force, and the loss of bone in the regions where the muscles used to attach.

Patients are unaware that these hard and soft tissue changes are from the loss of teeth. Among denture wearers, 39% have been wearing the same prosthesis for more than 10 years.[66] The profession is unable to evaluate patients unless they return yearly. Therefore the consequences of tooth loss must be explained to the partially or completely edentulous patient during the early phases of treatment (Box 1-5).

DECREASED PERFORMANCE OF COMPLETE DENTURES

The difference in maximum occlusal forces recorded in a person with natural teeth and one who is completely edentulous is dramatic. In the first molar region of a dentate person, the average force has been measured at 150 to 250 psi.[75] A patient who grinds or clenches the teeth may exert a force that approaches 1000 psi. The maximum occlusal force in the edentulous patient is reduced to less than 50 psi. The longer patients are

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Box 1-6 Negative Effects of Removable Prostheses

- Bite force is decreased from 200 psi to 50 psi
- 15-year denture wearers have reduced bite force to 6 psi
- Masticatory efficiency is decreased
- More drugs are necessary to treat gastrointestinal disorders
- The life span may be decreased
- Food selection is limited
- Healthy food intake is decreased

Box 1-7 Problems with Removable Partial Dentures

- Low survival rate—60% at 4 years
- 35% survival rate at 10 years
- Repair of abutment teeth rate—60% at 5 years and 80% at 10 years
- Increased mobility, plaque, bleeding upon probing, and caries of abutment teeth
- 44% abutment tooth loss within 10 years
- Accelerated bone loss in edentulous region if wearing removable partial denture

edentulous, the less force they are able to generate. Patients wearing complete dentures for more than 15 years may have a maximum occlusal force of 5.6 psi.[76]

As a result of decreased occlusal force and the instability of the denture, masticatory efficiency also decreases with tooth loss. Within the same 15-year time frame, 90% of the food chewed with natural teeth fits through a No. 12 sieve; this is reduced to 58% in the patient wearing complete dentures.[77] The tenfold decrease in force and the 40% decrease in efficiency affects the patient's ability to chew. In persons with dentures, 29% are able to eat only soft or mashed foods[78]; 50% avoid many foods; and 17% claim they eat more efficiently without the prosthesis.[79] A study of 367 denture wearers (158 men and 209 women) found that 47% exhibited a low masticatory performance.[79] Lower intakes of fruits, vegetables, and vitamin A by women were noted in this group. These patients took significantly more drugs (37%), compared with those with superior masticatory ability (20%); 28% were taking medications for gastrointestinal disorders. The reduced consumption of high-fiber foods could induce gastrointestinal problems in edentulous patients with deficient masticatory performance. In addition, the coarser bolus may impair proper digestive and nutrient extraction functions.[80]

The literature includes several reports suggesting that a compromised dental function causes poor swallowing and masticatory performance, which in turn may influence systemic changes favoring illness, debilitation, and shortened life expectancy[81-86] (Box 1-6).

In a study evaluating the ability to eat fruit, vegetables, and other dietary fiber in edentulous subjects, 10% claimed difficulty, and blood tests demonstrated reduced levels of plasma ascorbate and plasma retinol compared with dentate subjects. These two blood tests are correlated to an increased risk of dermatologic and visual problems in aging adults.[87]

In a study, the masticatory performance and efficiency in denture wearers were compared with dentate individuals.[88] This report noted that when appropriate connections were made for different performance norms and levels, the chewing efficiency of a denture wearer was less than one sixth of a person with teeth.

Several reports in the literature correlate a patient's health and life span to dental health.[89-93] Poor chewing ability may be a cause of involuntary weight loss in old age, with an increase in mortality.[94] In contrast, persons with a substantial number of missing teeth were more likely to be obese.[89]After conventional risk factors for strokes and heart attacks were accounted for, there was a significant relationship between dental disease and cardiovascular disease, the latter still remaining as the major cause of death.[92-96] It is logical to assume that restoring the stomatognathic system of these patients to a more normal function may indeed enhance the quality and length of their lives.[89,96-98]

NEGATIVE CONSEQUENCES OF REMOVABLE PARTIAL DENTURES

Removable soft tissue–borne partial dentures have one of the lowest patient acceptance rates in dentistry. Half the patients with a removable partial denture chew better without the device. A 44-year Scandinavian study revealed that only 80% of patients were wearing such prostheses after 1 year. The number further decreased to only 60% of the free-end partial dentures worn by the patients after 4 years.[99,100]

A 5-year survival rate of partial dentures based on tolerance and use of the prosthesis was approximately 60% for distal extension prostheses. This was reduced to 35% at 10 years.[101-103] In another study, few partial dentures survived more than 6 years.[104] Although one of five U.S. adults has had a removable prosthesis of some type, 60% reported at least one problem with it (Box 1-7).[50]

Reports of removable partial dentures indicate the health of the remaining dentition and surrounding oral tissues often deteriorates.[90,99,105] In a study that evaluated the need for repair of an abutment tooth as the indicator of failure, the survival rate of conventional removable partial dentures was 40% at 5 years and 20% at 10 years.[101] Those patients wearing the partial dentures often exhibit greater mobility of the abutment teeth, greater plaque retention, increased bleeding upon

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Box 1-8 Psychological Effects of Tooth Loss

- Range from minimal to neuroticism
- Romantic situations affected (especially in new relationship)
- "Oral invalids" unable to wear dentures
- 88% claim some difficulty with speech, and 25% claim significant problems
- More than $200 million each year spent on denture adhesive to decrease embarrassment
- Dissatisfaction with appearance, low self-esteem
- Avoidance of social contact

probing, higher incidence of caries, speech inhibition, taste inhibition, and noncompliance of use.[102-105] A report by Shugars et al. found abutment tooth loss for a removable partial denture may be as high as 23% within 5 years and 38% within 8 years.[30] Aquilino et al. reported a 44% abutment tooth loss within 10 years for a removable partial denture (see Box 1-7).[106] In addition, it should be noted those patients wearing the removable device accelerate bone loss in the soft tissue support regions. Therefore, alternative therapies that improve oral conditions and maintain bone are often warranted.

PSYCHOLOGICAL ASPECTS OF TOOTH LOSS

The psychological effects of total edentulism are complex and varied, and range from very minimal to a state of neuroticism (Box 1-8). Although complete dentures are able to satisfy the esthetic needs of many patients, there are those who feel their social life is significantly affected.[107,108] They are concerned with kissing and romantic situations, especially if a new partner in a relationship is unaware of their oral handicap. Fiske et al., in a study of interviews with edentulous subjects, found tooth loss was comparable to the death of a friend or loss of other important parts of a body in causing a reduction of self-confidence ending in a feeling of shame or bereavement.[107]

One dental survey of edentulous patients found 66% were dissatisfied with their mandibular complete dentures. Primary reasons were discomfort and lack of retention causing pain and discomfort.[109] Past dental health surveys indicate that only 80% of the edentulous population are able to wear both removable prostheses all the time.[90,110] Some patients wear only one prosthesis, usually the maxillary; others are able to wear their dentures for short periods only. In addition, approximately 7% of patients are not able to wear their dentures at all and become dental cripples or "oral invalids." They rarely leave their home environment and when they feel forced to venture out, the thought of meeting and talking to people when not wearing their teeth is unsettling.

A report of 104 completely edentulous patients seeking treatment was performed by Misch.[79] Of the patients studied, 88% claimed difficulty with speech, with one fourth having great difficulty. As a consequence, it is easy to correlate the reported increase with concern relative to social activities. Awareness of movement of the mandibular denture was cited by 62.5% of these patients, although the maxillary prosthesis stayed in place most of the time at almost the same percentage. Mandibular discomfort was listed with equal frequency as movement (63.5%), and surprisingly, 16.5% of the patients stated they never wear the mandibular denture. In comparison, the maxillary denture was uncomfortable half as often (32.6%), and only 0.9% were seldom able to wear the prosthesis. Function was the fourth most common problem reported by these 104 denture wearers. Half the patients avoided many foods, and 17% claimed they were able to masticate more effectively without the prostheses. The psychological effects of the inability to eat in public can be correlated with these findings. Other reports agree that the major motivating factors for patients to undergo treatment were related to the difficulties with eating, denture fit, and discomfort.

The psychological need of the edentulous patient is expressed in many forms. For example, in 1970, Britons used approximately 88 tons of denture adhesive.[111] In 1982, more than 5 million Americans used denture adhesives (Ruskin Denture Research Associates: AIM study, unpublished data, 1982), and a report shows that in the United States, more than $200 million is spent each year on denture adhesives, representing 55 million units sold.[112] The patient is willing to accept the unpleasant taste, need for recurring application, inconsistent denture fit, embarrassing circumstances, and continued expense for the sole benefit of increased retention of the prosthesis. Clearly the lack of retention and psychological risk of embarrassment in the denture wearer with removable prostheses is a concern the dental profession must address.

ADVANTAGES OF IMPLANT-SUPPORTED PROSTHESES

The use of dental implants to provide support for prostheses offers many advantages, compared with the use of removable soft tissue–borne restorations (Box 1-9). A primary reason to consider dental implants to replace missing teeth is the maintenance of alveolar bone (Figure 1-26). The dental implant placed into the bone serves both as an anchor for the prosthetic device and as one of the better preventive maintenance procedures in dentistry. Stress and strain may be applied to the bone surrounding the implant. As a result, the decrease in trabeculation of bone that occurs after

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Box 1-9 Advantages of Implant-Supported Prostheses

- Maintain bone
- Restore and maintain occlusal vertical dimension
- Maintain facial esthetics (muscle tone)
- Improve esthetics (teeth positioned for appearance versus decreasing denture movement)
- Improve phonetics
- Improve occlusion
- Improve/regain oral proprioception (occlusal awareness)
- Increase prosthesis success
- Improve masticatory performance/maintain muscles of mastication and facial expression
- Reduce size of prosthesis (eliminate palate, flanges)
- Provide fixed versus removable prostheses
- Improve stability and retention of removable prostheses
- Increase survival times of prostheses
- No need to alter adjacent teeth
- More permanent replacement
- Improve psychological health

tooth extraction is reversed. There is an increase in bone trabeculae and density when the dental implant is inserted and functioning. The overall volume of bone is also maintained with a dental implant. Even grafts of iliac bone to the jaws, which usually resorb without dental implant insertion within 5 years, are instead stimulated and maintain overall bone volume and implant integration. An endosteal implant can maintain bone width and height as long as the implant remains healthy.[113] As with a tooth, periimplant bone loss may be measured in tenths of a millimeter and may represent a more than twentyfold decrease in lost structure, compared with the resorption that occurs with removable prostheses.

Figure 1-26 Bone loss in the edentulous patient may be dramatically stopped by using implants to support the prosthesis.

The benefit of bone maintenance is especially noteworthy in the maxillary edentulous arch. Rather than using implants only in the edentulous mandibular arch, because the main mechanical denture problems are in this arch, the maxillary arch should also be addressed. Once implant prostheses are placed to support and retain the mandibular restoration, the bone in the maxillary region continues to be lost and eventually the patient may complain of loss of retention and inability of the maxillary denture to function.[114] The loss of facial esthetics is most often first noted in the maxillary arch, with the loss of vermillion border of the lip, increased length of the maxilla lip, and lack of facial bone support. Implants should be used to treat the continued bone loss and prevent the later complications found in the maxillary arch (Figures 1-27 to 1-31).

A mandibular denture often moves when the mylohyoid and buccinator muscles contract during speech or

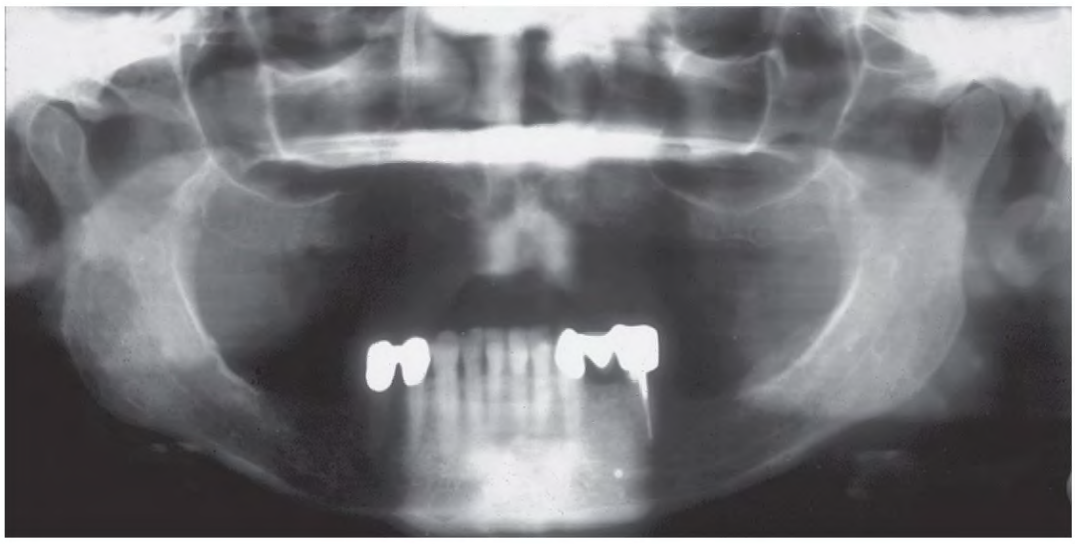


Figure 1-27 A panoramic radiograph of an edentulous maxillary arch with moderate sinuses and a resorbed anterior maxilla.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

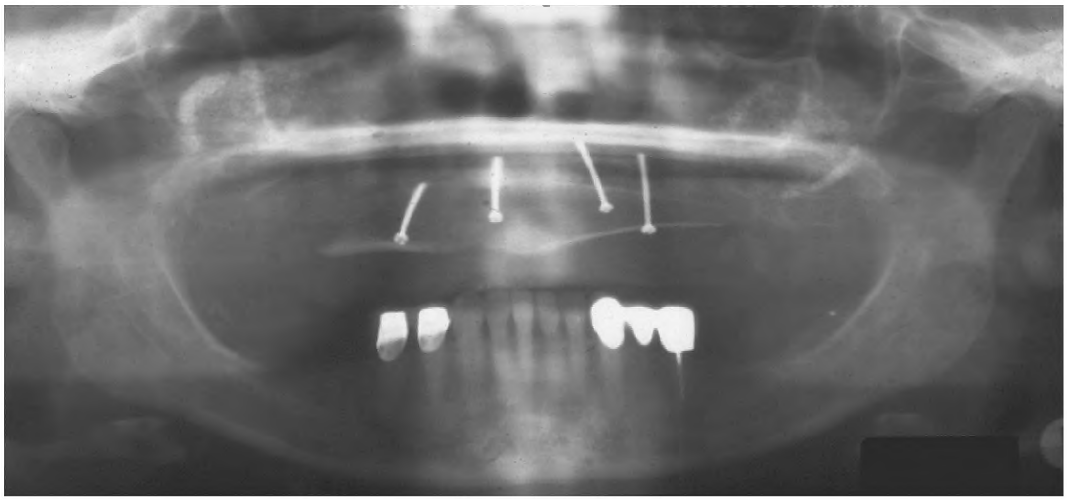


Figure 1-28 An iliac crest bone graft was inserted into the maxilla, and bilateral sinus grafts were also made to the posterior regions.

Figure 1-29 Maxillary implants were inserted after the graft in Figure 1-28 matured, and a maxillary fixed prosthesis was fabricated.

mastication. The teeth are often positioned for denture stability rather than where natural teeth usually reside. With implants, the teeth may be positioned to enhance esthetics and phonetics rather than in the neutral zones dictated by traditional denture techniques to improve the stability of a prosthesis.

The features of the inferior third of the face are closely related to the supporting skeleton. When vertical bone is lost, the dentures only act as "oral wigs" to improve the contours of the face. The dentures become bulkier as the bone resorbs, making it more difficult to control function, stability, and retention. With implant-supported prostheses, the vertical dimension may be restored, similar to natural teeth. In addition, the implant-supported prosthesis allows a cantilever of anterior teeth for ideal soft tissue and lip contour and improved appearance in all facial planes. This happens without the instability that usually occurs when an anterior cantilever is incorporated in a traditional denture. The facial profile may be enhanced for the long term with implants, rather than deteriorating over the years, as can occur with traditional dentures.

Occlusion is difficult to establish and stabilize with a completely soft-tissue–supported prosthesis. Because the mandibular prosthesis may move as much as 10 mm or more during function,[115,116] proper occlusal contacts occur by chance, not by design. But an implant-supported restoration is stable. The patient can more consistently return to centric-relation occlusion rather than adopt variable positions dictated by the prosthesis' instability. Proprioception is awareness of a structure in time and place. The receptors in the periodontal membrane of the natural tooth help determine its occlusal position. Although endosteal implants do not have a periodontal membrane, they provide greater occlusal awareness than complete dentures. Patients

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

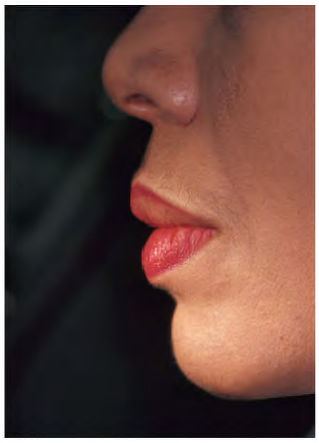


Figure 1-30 The lateral profile of the patient shown in Figures 1-27 to 1-29. Note the support of the maxillary lip.

Figure 1-31 The high smile line of the patient in Figures 1-25 to 1-30 with the final restoration in place.

with natural teeth can perceive a difference of 20 µm between the teeth, whereas implant patients can determine a 50-µm differences with rigid implant bridges, compared with 100 microns in those with complete dentures (either one or two).[117]

As a result of improved occlusal awareness, the patient functions in a more consistent range of occlusion. With an implant-supported prosthesis, the direction of the occlusal loads is controlled by the restoring dentist. Horizontal forces on removable prostheses accelerate bone loss, decrease prosthesis stability, and increase soft tissue abrasions. Therefore the decrease in horizontal forces that are applied to implant restorations improves the local parameters and helps preserve the underlying soft and hard tissues.

In a randomized clinical trial by Kapur et al., the implant group of patients demonstrated a higher level of eating enjoyment and improvement of speech, chewing ability, comfort, denture security, and overall satisfaction.[118] The ability to eat several different foods among complete denture versus mandibular overdenture patients was evaluated by Awad and Feine. The implant overdenture was superior for eating not only harder foods, such as carrots and apples, but also softer foods, such as bread and cheese.[119] Geertman et al. evaluated complete denture wearers with severely resorbed mandibles before and after mandibular implant overdentures. The ability to eat hard or tough foods significantly improved.[120,121]

Researchers at McGill University evaluated blood levels of patients who had complete dentures and 30 maxillary dentures and mandibular implant prostheses 6 months after treatment. Within this rather short period, implant patients had higher B_{12} hemoglobin (related to iron increase) and albumin levels (related to nutrition). These patients also had greater body fat in their shoulders and arms, with decreased body fat in their waists.[122]

The success rate of implant prostheses varies, depending on a host of factors that change for each patient. However, compared with traditional methods of tooth replacement, the implant prosthesis offers increased longevity, improved function, bone preservation, and better psychological results. According to 10-year survival surveys of fixed prostheses on natural teeth, decay is indicated as the most frequent reason for replacement; survival rates are approximately 75%.[31] In the partially edentulous patient, independent tooth replacement with implants may preserve intact adjacent natural teeth as abutments, further limiting complications such as decay or endodontic therapy, which are the most common causes of fixed prosthesis failure. A major advantage of the implant-supported prosthesis is that the abutments cannot decay and never require endodontics. The implant and related prosthesis can attain a 10-year survival of more than 90%.

The maximum occlusal force of a traditional denture wearer ranges from 5 to 50 lb. Patients with an implant-supported fixed prosthesis may increase their maximum bite force by 85% within 2 months after the completion of treatment. After 3 years, the mean force may reach more than 300%, compared with pretreatment values. As a result, an implant prosthesis wearer may demonstrate a force similar to that of a patient with a fixed restoration supported by natural teeth. Chewing efficiency with an implant prosthesis is greatly improved, compared with that of a soft tissue–borne restoration. The masticatory performance of dentures, overdentures, and natural dentition was evaluated by Rissin et al.[77] The traditional denture showed a 30% decrease in chewing efficiency; other reports indicate a denture wearer has less than 60% of the function of people with natural teeth. The supported overdenture loses only 10% of chewing efficiency compared with natural teeth. These findings are similar with implant-supported overdentures. In addition, rigid, implant-supported fixed bridges may function the same as natural teeth. Beneficial effects such as a decrease in fat, cholesterol, and the carbohydrate food groups have been reported, as well as significant improvement in eating enjoyment and social life.[123-131]

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

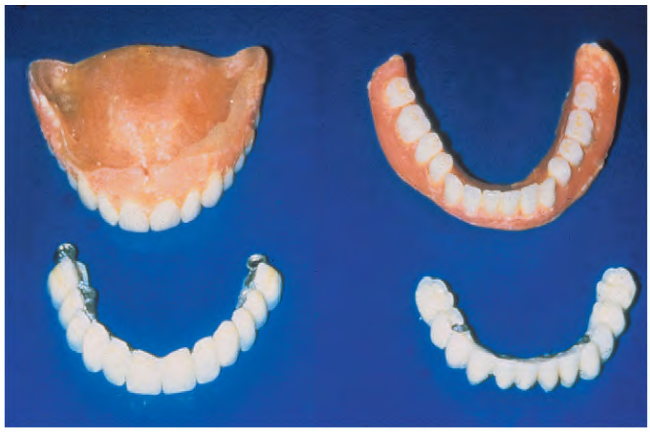


Figure 1-32 Implant prostheses can maintain bone and reduce the bulk of the soft tissue–borne prostheses.

Stability and retention of an implant-supported prosthesis are great improvements over soft tissue–borne dentures (Figure 1-32). Mechanical means of implant retention are far superior to the soft tissue retention provided by dentures or adhesives and cause fewer associated problems. The implant support of the final prosthesis is variable, depending on the number and position of implants; yet all treatment options demonstrate significant improvement.

Phonetics may be impaired by the instability of a conventional denture. The buccinator and mylohyoid muscles may flex and propel the posterior portion of the denture upward, causing clicking, regardless of the vertical dimension.[116] As a result, a patient in whom the vertical dimension already has collapsed 10 to 20 mm may still produce clicking sounds during speech. Often the tongue of the denture wearer is flattened in the posterior areas to hold the denture in position. The anterior mandibular muscles of facial expression may be tightened to prevent the mandibular prosthesis from sliding forward. The implant prosthesis is stable and retentive and does not require these oral manipulations. The implant restoration allows reduced flanges or palates of the prostheses. This is of special benefit to the new denture wearer, who often reports discomfort with the bulk of the restoration. The extended soft tissue coverage also affects the taste of food, and the soft tissue may be tender in the extended regions. The palate of a maxillary prosthesis may cause gagging in some patients, which can be eliminated in an implant-supported overdenture.

Patients treated with implant-supported prostheses judge their overall psychological health as improved by 80% compared with their previous state while wearing traditional, removable prosthodontic devices. They perceived the implant-supported prosthesis as an integral part of their body.[120,132-136] For example, Raghoebar et al. evaluated 90 edentulous patients in a randomized multicenter study. Five years after treatment, a validated questionnaire targeted patient esthetic satisfaction, retention, comfort, and the ability to speak and eat with either a complete mandibular denture, complete mandibular denture with vestibuloplasty, or mandibular two-implant overdenture. Implant overdentures had significantly higher ratings, whereas no significant difference was found between the two complete-denture groups.[135] Geertman et al. reported similar results comparing chewing ability of conventional complete dentures with mandibular implant overdentures.[120,121]

SUMMARY

The goal of modern dentistry is to return patients to oral health in a predictable fashion. The partial and complete edentulous patient may be unable to recover normal function, esthetics, comfort, or speech with a traditional removable prosthesis. The patient's function when wearing a denture may be reduced to one sixth of that level formerly experienced with natural dentition; however, an implant prosthesis may return the function to near-normal limits. The esthetics of the edentulous patient are affected as a result of muscle and bone atrophy. Continued bone resorption leads to irreversible facial changes. An implant prosthesis allows normal muscle function, and the implant stimulates the bone and maintains its dimension in a manner similar to healthy natural teeth. As a result, the facial features are not compromised by lack of support as often required for removable prostheses. In addition, implant-supported restorations are positioned in relation to esthetics, function, and speech, not in neutral zones of soft tissue support. The soft tissues of the edentulous patients are tender from the effects of thinning mucosa, decreased salivary flow, and unstable or unretentive prostheses. The implant-retained restoration does not require soft tissue support and improves oral comfort. Speech is often compromised with soft tissue–borne prostheses because the tongue and perioral musculature may be compromised to limit the movement of the mandibular prosthesis. The implant prosthesis is stable and retentive without the efforts of the musculature.

Implant prostheses often offer a more predictable treatment course than traditional restorations. Thus the profession and the public are becoming increasingly aware of this dental discipline. Manufacturers' sales have increased from a few million dollars to more than several hundred million dollars. Almost every professional journal now publishes refereed reports on dental implants. All U.S. dental schools now teach implant dentistry to all interfacing specialties. Implant dentistry has finally been accepted by organized dentistry. The current trend to expand the use of implant dentistry will continue until every restorative practice uses this modality for abutment support of both fixed and removable prostheses on a regular basis as the primary option for all tooth replacement.[137]

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

References

1. Tatum OH: *The Omni implant system*, Birmingham, Ala, 1988, Alabama Implant Congress.
2. National Institutes of Health consensus development conference statement on dental implants, *J Dent Educ* 52:686-691, 1988.
3. *Millenium Research Group report: US Markets for Dental Implants 2006*, USDI 06, June, 2006.
4. *Implant based dental reconstruction. The worldwide implant and bone graft market,* September 2005. Available at http://www.kaloramainformation.com. Accessed July 14, 2007.
5. Stillman N, Douglass CW: Developing market for dental implants, *J Am Dent Assoc* 124:51-56, 1993.
6. Watson MT: Implant dentistry: a 10-year retrospective report, *Dental Products Report* 30:26-32, 1996.
7. Watson MT: Specialist's role in implant dentistry rooted in history: a survey of periodontists and maxillofacial surgeons, *Dental Products Report* 31:14-18, 1997.
8. Reis-Schmidt T: Surgically placing implants—a survey of oral maxillofacial surgeons and periodontists, *Dental Products Report* 32:26-30, 1998.
9. Goff S: Trends in dentistry, *Dental Products Report* 36: 16-24, 2002.
10. Marcus SE, Drury JF, Brown LS et al: Tooth retention and tooth loss in the permanent dentition of adults: United States, 1988-1991, *J Dent Res* 75(spec issue):684-695, 1996.
11. Meskin LH, Brown LJ: Prevalence and patterns of tooth loss in U.S. employed adult and senior populations, 1985-86, *J Dent Educ* 52:686-691, 1988.
12. Murdock SH, Hogue MN: Current patterns and future trends in the population of the United States: implications for dentists and the dental profession in the 21st century, *J Am Coll Dent* 65:29-38, 1998.
13. Census 2000 Data on Aging. Available at http://www.aoa.gov/prof/statistics/census2000/census2000.asp. Accessed July 14, 2007.
14. Hellmich N: Extra weight shaves years off lives. *USA Today* A1, January 7, 2003.
15. Dychtwald K: *Age wave: the challenges and opportunities of an aging America,* New York, 1988, St. Martin's Press.
16. *A profile of older americans,* Washington, DC, 1993, American Association of Retired Persons.
17. *Health, United States, 2004, Life expectancy at 65 and 75 years.* Available at http://www.cdc.gov/nchshus.htm. Accessed July 14, 2007.
18. *U.S. Census Bureau 2000: Summary file 1, matrices P13 and PCT12*. Available at http://www.factfinder.census.gov. Accessed July 14, 2007.
19. Aschenbrener CA: The future is in the present: the implant of generations, *J Am Coll Dent* 65:23-28, 1998.
20. Meskin LH, Berg R: Impact of older adults on private dental practices, 1988-1998, *J Am Dent Assoc* 131:1188-1195, 2000.
21. Palmqvist S, Swartz B: Artificial crowns and fixed partial dentures 18 to 23 years after placement, *Int J Prosthodont* 6:279-285, 1993.
22. Hirschfeld L, Wasserman B: A long term survey of tooth loss in 600 treated periodontal patients, *J Periodontol* 49:225-237, 1978.
23. Bloom B, Gaft HC, Jack SS: *National Center for Health Statistics. Dental Services and Oral Health. United States, 1989 Vital Health Stat 10(183)*. DHHS Pat No (PAS) 93-1511. Washington, DC, 1992, U.S. Government Printing Office.
24. Schwartz NL, Whitsett LD, Berry TG: Unserviceable crowns and fixed partial dentures: life-span and causes for loss of serviceability, *J Am Dent Assoc* 81:1395-1401, 1970.
25. Americal Dental Association Survey Center: Changes in dental services rendered 1959-1990. In *1990 Survey of Dental Services Rendered*. Chicago, 1994, American Dental Association, pp 24-38.
26. Cohen BD, Milobsky SA: Monetary damages in dental-injury cases. *Trial Lawyers Quarterly* 29:80-81, 1989.
27. Cheung GSP, Dimmer A, Mellor R et al: A clinical evaluation of conventional bridgework, *J Oral Rehabil* 17:131-136, 1990.
28. Priest GF: Failure rates of restorations for single tooth replacements, *Int J Prosthodont* 9:38-45, 1996.
29. Creugers NH, Kayser HF, Van't Hof MA: A meta analysis of durability data on conventional fixed bridges, *Community Dent Oral Epidemiol* 22:448-452, 1994.
30. Shugars DA, Bader JD, White BA et al: Survival rates of teeth adjacent to treated and untreated posterior bounded edentulous spaces, *J Am Dent Assoc* 129:1085-1095, 1998.
31. Walton JN, Gardner FM, Agar JR: A survey of crown and fixed partial denture failures, length of service and reasons for replacement, *J Prosthet Dent* 56:416-421, 1986.
32. Goodacre CJ, Bernal G, Rungcharassaeng K: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
33. Schillinburg HT, Hobo S, Whitsett LD et al: *Fundamentals of fixed prosthodontics,* ed 3, Chicago, 1997, Quintessence.
34. Priest GG: Single tooth implants and their role in preserving remaining teeth: a 10 year survival study, *Int J Oral Maxillofac Implants* 14:181-188, 1999.
35. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants in implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
36. Schmitt A, Zarb GA: The longitudinal clinical effectiveness of osseointegrated dental implants for single tooth replacement, *Int J Prosthodont* 6:187-202, 1993.
37. Ekfeldt A, Carlsson, Borgeson G: Clinical evaluation of single tooth restorations supported by osseointegrated implants: a retrospective study, *Int J Oral Maxillofac Implants* 9:179-183, 1994.
38. Haas R, Mensdorff-Pouilly N, Mailath G et al: Brånemark single tooth implants: a preliminary report of 76 implants, *J Prosthet Dent* 73:274-279, 1995.
39. Fugazzotto PA: Success and failure rates of osseointegrated implants in function in regenerated bone for 72 to 133 months, *Int J Oral Maxillofac Imp* 20:77-83, 2005.
40. Laney WR, Jemt T, Harris D et al: Osseointegrated implants for single tooth replacement: a progress report from a multicenter prospective study after 3 years, *Int J Oral Maxillofac Implants* 9:49-54, 1994.
41. Henry PH, Laney WR, Jemt T et al: Osseointegrated implants for single tooth replacement: a prospective 5 year multicenter study, *Int J Oral Maxillofac Implants* 11:450-455, 1996.
42. Malevez C, Hermans M, Daelemans P: Marginal bone levels at Brånemark system implants used for single tooth restoration: the influence of implant design and anatomical region, *Clin Oral Implants Res* 7:162-169, 1996.
43. Gomez-Roman G, Schulte W, d'Hoedt B et al: The frialit-2 implant systems: 5 year clinical experience in single

t.me/highdent www.highdentlab.com instagram.com/high_dent

tooth and immediately post extraction applications, *Int J Oral Maxillofac Implants* 12:209-309, 1997.
44. Kline R, Hoar JE, Beck GH et al: A prospective multicenter clinical investigation of a bone quality based dental system, *Implant Dent* 11:224-234, 2002.
45. Misch CE, Poitras Y, Dietsh-Misch F et al: Endosteal implants in the edentulous posterior maxilla—rationale clinical report, *Oral Health* Aug:7-16, 2000.
46. Misch CE, d'Alessio R, Misch-Dietsh F: Maxillary partial anodontia and implant dentistry—maxillary anterior partial anodontia in 255 adolescent patients: a 15-year retrospective study of 276 implant site replacements, *Oral Health* Aug:45-57, 2005.
47. Priest G, Priest J: The economics of implants for single missing teeth, *Dent Econ* May:130-138, 2004.
48. Centers for Disease Control and Prevention: Surveillance for dental caries, dental sealants, tooth retention, edentulism and enamel fluorosis—United States, 1988-1994 and 1999-2002. In Beltram-Aguilar ED, Bazker LK, Canto MT: *Surveillance summaries,* August 26, 2005, MMWR 2015-2054 (No. SS3).
49. Weintraub JA, Bret BA: Oral health status in the United States: tooth loss and edentulism, *J Dent Ed* 49:368-378, 1988.
50. Meskin LH, Brown LJ, Brunelle JA: Patterns of tooth loss and accuracy of prosthodontic treatment potential in U.S. employed adults and seniors, *Gerodontics* 4:126-135, 1988.
51. Redford M, Drury TF, Kingman A et al: Denture use and the technical quality of dental prostheses among persons 18-74 years old in the United States between 1988 and 1991, *J Dent Res* 75(spec issue):714-725, 1996.
52. Takala L, Utriainen P, Alanen P: Incidence of edentulousness, reasons for full clearance, and health status of teeth before extractions in rural Finland, *Community Dent Oral Epidemiol* 22:254-257, 1994.
53. Steele JG, Treasure E, Pritts NB et al: Total tooth loss in the United Kingdom in 1998 and implications for the future, *Br Dent J* 189:598-603, 2000.
54. Mojon P: The world without teeth: demographic trends. In Feine JS, Carlsson GE, editors: *Implant overdentures: the standard of care for edentulous patients,* Carol Stream, Ill, 2003, Quintessence.
55. Health Promotion Survey Canada: Statistics Canada, 1990, record number 3828. Available at http://www.statcan. Accessed July 14, 2007.
56. Doug CW, Shih A, Ostry L: Will there be a need for complete dentures in the United States in 2020? *J Prosthet Dent* 87:5-8, 2002.
57. Freeman E, Ten Cate AR: Development of the periodontium: an electron microscopic study, *J Periodontol* 42:387-395, 1971.
58. Wolff J: *The laws of bone remodeling,* Berlin, 1986, Springer (Translated by Maquet P, Furlong R; originally published in 1892).
59. Murray PDF: *Bones: a study of the development and structure of the vertebrae skeleton,* Cambridge, 1936, Cambridge University Press.
60. Misch J: *Lehrbuch der Grenzgebiete der Medizin und Zahnheilkunde,* Leipzig, Germany, 1922, FC Vogel.
61. Roberts WE, Turley PK, Brezniak N et al: Implants: bone physiology and metabolism, *Cal Dent Assoc J* 15:54-61, 1987.
62. Bassett CA: Biologic significance of piezoelectricity, *Calcif Tissue Res* 1:252-272, 1968.
63. Pietrokovski J: The bony residual ridge in man, *J Prosthet Dent* 34:456-462, 1975.
64. Carlsson G, Persson G: Morphologic changes of the mandible after extraction and wearing of dentures: a longitudinal clinical and x-ray cephalometric study covering 5 years, *Odont Revy* 18:27-54, 1967.
65. Tallgren A: The reduction in face height of edentulous and partially edentulous subjects during long-term denture wear: a longitudinal roentgenographic cephalometric study, *Acta Odontol Scand* 24:195-239, 1966.
66. Gruber H, Solar P, Ulm C: Maxillomandibular anatomy and patterns of resorption during atrophy. In Watzek G, editor: *Endosseous implants: scientific and clinical aspects,* Chicago, 1996, Quintessence.
67. Brodeur JM, Laurin P, Vallee R et al: Nutrient intake and gastrointestinal disorders related to masticatory performance in the edentulous elderly, *J Prosthet Dent* 70:468-473, 1993.
68. Marcus P, Joshi A, Jones J et al: Complete edentulism and denture use for elders in New England, *J Prosthet Dent* 76:260-265, 1996.
69. Hickey JC, Zarb GA, Bolender CL editors: *Boucher's prosthodontic treatment for edentulous patients*, ed 10, St Louis, 1990, Mosby, pp 3-27.
70. Gabriel AC: Some anatomical features of the mandible, *J Anat* 92:580-589, 1958.
71. Sassouni V: A classification of skeletal facial types, *Am J Orthod* 55:109-123, 1969.
72. Mercier P, Lafontant R: Influence of facial morphology classes on residual alveolar ridge atrophy, *J Prosthet Dent* 41:90-100, 1979.
73. Rissin L, House JE, Conway C et al: Effect of age and removable partial dentures on gingivitis and periodontal disease, *J Prosthet Dent* 42:217-223, 1979.
74. Sutton DM, Lewis BRK, Patel M et al: Changes in facial form relative to progressive atrophy of the edentulous jaw, *Int J Oral Maxillofac Surg* 33:676-682, 2004.
75. Howell AW, Manley RS: An electronic strain gauge for measuring oral forces, *J Dent Res* 27:705, 1948.
76. Carr A, Laney WR: Maximum occlusal force levels in patients with osseointegrated oral implant prostheses and patients with complete dentures, *Int J Oral Maxillofac Implants* 2:101-110, 1987.
77. Rissin L, House JE, Manly RS et al: Clinical comparison of masticatory performance and electromyographic activity of patients with complete dentures, overdentures and natural teeth, *J Prosthet Dent* 39:508-511, 1978.
78. Carlsson GE, Haraldson T: Functional response. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue integrated prostheses: osseointegration in clinical dentistry,* Chicago, 1985, Quintessence.
79. Misch LS, Misch CE: Denture satisfaction: a patient's perspective, *Int J Oral Implant* 7:43-48, 1991.
80. Feldman RS, Kapur KK, Alman JE et al: Aging and mastication changes in performance and in the swallowing threshold with natural dentition, *J Am Geriatr Soc* 28:97-103, 1980.
81. Chen MK, Lowenstein F: Masticatory handicap, socioeconomic status and chronic conditions among adults, *J Am Dent Assoc* 109:916-918, 1984.
82. Hildebrandt GH, Dominguez BL, Schock MA et al: Functional units, chewing, swallowing and food avoidance among the elderly, *Prosthet Dent* 77:588-595, 1997.
83. Joshipura KJ, Wilkett WC, Douglass CW: The impact of edentulousness on food and nutrient intake, *J Am Dent Assoc* 127:459-467, 1996.

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

84. Sheiham A, Steele JC, Marcenes W et al: The impact of oral health on stated ability to eat certain food; findings from the National Diet and Nutrition Survey of Older People in Great Britain, *Gerontology* 16:11-20, 1999.
85. Krall E, Hayes C, Garcia R: How dentition status and masticatory function affect nutrient intake, *J Am Dent Assoc* 129:20-23, 1998.
86. Sheiham A, Steele JG, Marcenes W et al: The relationship among dental status, nutrient intake, and nutritional status in older people, *J Dent Res* 80:408-413, 2001.
87. Sheiham A, Steele J: Does the condition of the mouth and teeth affect the ability to eat certain foods, nutrient and dietary intake and nutritional status amongst older people? *Public Health Nutr* 4:797-803, 2001.
88. Kapur KK, Soman SD: Masticatory performance and efficiency in denture wearers, J *Prosthet Dent* 14:687-694, 1964.
89. Sheiham A, Steele JG, Marcenes W et al: The relationship between oral health status and body mass index among older people: a national survey of older people in Great Britain, *Br Dent J* 192:703-706, 2002.
90. Agerberg G, Carlsson GE: Chewing ability in relation to dental and general health, *Acta Odontol Scand* 39:147-153, 1981.
91. Hildebrandt GH, Loesche WJ, Lin CF et al: Comparison of the number and type of dental functional units in geriatric populations with diverse medical backgrounds, *J Prosthet Dent* 73:253-261, 1995.
92. DeStefano F, Anda RF, Kahn HS et al: Dental disease and risk of coronary heart disease and mortality, *Br Med J* 306:688-691, 1993.
93. Syrjonen J, Peltola J, Valtonen V et al: Dental infections in association with cerebral infarction in young and middle-aged men, *Intern Med* 225:179-184, 1989.
94. Sullivan D, Walls R, Lipschitz D: Protein-energy undernutrition and risk of mortality within 1 year of hospital discharge in a select population of geriatric rehabilitation patients, *Am J Clin Nutr* 43:559-605, 1991.
95. Mattila KJ, Nieminen MS, Valtonen V et al: Association between dental health and acute myocardial infarction, *Br Med J* 298:779-782, 1989.
96. Loesche WJ: Periodontal disease as a risk factor for heart disease, *Compend Contin Educ Dent* 15:976-992, 1994.
97. Carlsson GE: Masticatory efficiency: the effect of age, the loss of teeth, and prosthetic rehabilitation, *Int Dent J* 34:93-97, 1984.
98. Gunne HS, Wall AK: The effect of new complete dentures on mastication and dietary intake, *Acta Odontol Scand* 43:257-268, 1985.
99. Koivumaa KK, Hedegard B, Carlsson GE: Studies in partial denture prostheses: I. An investigation of dentogingivally-supported partial dentures, *Suom Hammaslaak Toim* 56:248-306, 1960.
100. Carlsson GE, Hedegard B, Koivumaa KK: Studies in partial denture prosthesis: IV. A 4-year longitudinal investigation of dentogingivally-supported partial dentures, *Acta Odontol Scand* 23:443-472, 1965.
101. Wetherell J, Smales R: Partial dentures failure: a long-term clinical survey, *J Dent* 8:333-340, 1980.
102. Wilding R, Reddy J: Periodontal disease in partial denture wearers—a biologic index, *J Oral Rehab* 14:111-124, 1987.
103. Vermeulen A, Keltjens A, Vant'hof M et al: Ten-year evaluation of removable partial dentures: survival rates based on retreatment, not wearing and replacement, *J Prosthet Dent* 76:267-272, 1996.
104. Roberts BA: Survey of chrome cobalt partial dentures, *N Z Dent J* 74:203-209, 1978.
105. Waerhaug J: Periodontology and partial prosthesis, *Int Dent J* 18:101-107, 1968.
106. Aquilino SA, Shugars DA, Bader JD et al: Ten-year survival rates of teeth adjacent to treated and untreated posterior bounded edentulous spaces, *J Prosthet Dent* 85:455-60, 2001.
107. Fiske J, Davis DM, Frances C et al: The emotional effects of tooth loss in edentulous people, *Br Dent J* 184:90-93, 1998.
108. Slade GD: *Measuring oral health and quality of life,* Chapel Hill, NC, 1997, University of North Carolina Department of Dental Ecology.
109. Berg E: The influence of some anamnestic demographic and clinical variables on patient acceptance of new complete dentures, *Acta Odontol Scand* 42:119-127, 1984.
110. Bergman B, Carlsson GE: Clinical long term studies of complete denture wearers, *J Prosthet Dent* 53:56-61, 1985.
111. Stafford GD: Denture adhesives: a review of their use and composition, *Dent Pract* 21:17-19, 1970.
112. Pinto D, editor: *Chain Drug Review* 20:46, 1998.
113. Zarb G, Schmitt A: Edentulous predicament. I. A prospective study of the effectiveness of implant supported fixed prostheses, *J Am Dent Assoc* 127:59-72, 1996.
114. Narhi TO, Geertman ME, Hevinga M et al: Changes in the edentulous maxilla in persons wearing implant-retained mandibular overdentures, *J Prosthet Dent* 84: 43-49, 2000.
115. Sheppard IM: Denture base dislodgement during mastication, *J Prosthet Dent* 13:462-468, 1963.
116. Smith D: The mobility of artificial dentures during comminution, *J Prosthet Dent* 13:834-856, 1963.
117. Lundqvist S, Haraldson T: Occlusal perception of thickness in patients with bridges on osteointegrated oral implants, *Scand J Dent Res* 92:88, 1984.
118. Kapur KK, Garrett NR, Hamada MO et al: Randomized clinical trial comparing the efficacy of mandibular implant supported overdentures and conventional dentures in diabetic patients. Part III: comparisons of patient satisfaction, *J Prosthet Dent* 82:416-427, 1999.
119. Awad MA, Feine JJ: Measuring patient satisfaction with mandibular prostheses, *Community Dent Oral Epidemiol* 26:400-405, 1998.
120. Geertman ME, Boerrigter EM, van't Hof MA et al: Two-center clinical trial of implant-retained mandibular overdentures versus complete dentures—chewing ability, *Community Dent Oral Epidemiol* 24:79-84. 1996.
121. Geertman ME, Van Waas MA, van't Hof MA et al: Denture satisfaction in a comparative study of implant-retained mandibular overdenture: a randomized clinical trial, *Int J Oral Maxillofac Implants* 11:194-2000, 1996.
122. McGill University: *Health and Nutrition Letter* (2)21, April 2003.
123. Humphries GM, Healey T, Howell RA et al: The psychological impact of implant-retained mandibular prostheses: a cross-sectional study, *Int J Oral Maxillofac Implants* 10:437-44, 1995.
124. Meijer HJ, Raghoebar GM, van't Hof MA et al: Implant-retained mandibular overdentures compared with complete dentures; a 5 years' follow up study of clinical aspects and patient satisfaction, *Clin Oral Implants Res* 10:238-244, 1999.
125. Harle TH, Anderson JD: Patient satisfaction with implant supported prostheses, *Int J Prosthodont* 6:153-162, 1993.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

126. Wismeijer D, van Waas MA, Vermeeren JI et al: Patient satisfaction with implant-supported mandibular overdentures: a comparison of three treatment strategies with ITI-dental implants, *Int J Oral Maxillofac Surg* 26:263-267, 1997.
127. Leung AC, Cheung LK: Dental implants in reconstructed jaws: patients' evaluation of functional and quality of life outcomes, *Int J Oral Maxillofac Implants* 18:127-134, 2003.
128. Grogono AL, Lancaster DM, Finger IM: Dental implants: a survey of patients' attitudes, *J Prosthet Dent* 62:573-576, 1989.
129. Kapur KK: Veterans Administration cooperative dental implant study: comparisons between fixed partial dentures supported by blade-vent implants and removable partial dentures. Part II. Comparisons of mastication performances between two treatment modalities, *J Prosthet Dent* 65:272-283, 1991.
130. Kapur KK: Veterans Administration cooperative dental implant study. Part IV. Comparisons of patient satisfaction between two treatment modalities, *J Prosthet Dent* 66:517-530, 1991.
131. Garrett NR, Kapur KK, Hasse AL: Veterans Administration cooperative dental implant study. Part V: comparisons of pretreatment and posttreatment dietary intakes, *J Prosthet Dent* 77:153-161, 1997.
132. Blomberg S: Psychological response. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue integrated prostheses: osseointegration in clinical dentistry,* Chicago, 1985 Quintessence.
133. Albrektsson T, Blomberg S, Brånemark A et al: Edentulousness: an oral handicap. Patient reactions to treatment with jawbone-anchored prosthesis, *J Oral Rehab* 14:503-511, 1987.
134. Blomberg S, Lundquist S: Psychological reactions to edentulousness and treatment with jawbone-anchored bridges, *J Prosthet Dent* 50:262-270, 1983.
135. Raghoebar GM Meijer HJ, Steigenga B et al: Effectiveness of three treatment modalities for the edentulous mandible: a five year randomized clinical trial, *Clin Oral Implants Res* 11:195-201, 2000.
136. Al-Omiri M, Hantash RA, Al-Wahadni A: Satisfaction with dental implants: a literature review, *Implant Dent* 14:399-406, 2005.
137. Misch CE: Dental education: meeting the demands of implant dentistry, *J Am Dent Assoc* 121:334-338, 1990.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Chapter 2

Generic Root Form Component Terminology

Carl E. Misch

An endosteal implant is an alloplastic material surgically inserted into a residual bony ridge, primarily as a prosthodontic foundation.[1] The prefix *endo* means "within," and *osteal* means "bone."[2] The major subcategory of endosteal implants covered in this text are root form implants. The term *endosseous* is also used in the literature, and because the term *osseous* also indicates bone, either term is acceptable. However, *endosteal*, *periosteal*, and *transosteal* are preferred. Many endosteal implant designs have been used in the past, including tapered pegs, pin shapes, and plate forms.[3,4] Today, an endosteal implant in the shape of a tooth root is the design most often used in the restoration of partial or complete edentulous patients.

The desire has always been to replace missing teeth with something similar to the root of a tooth. Implant dentistry is the second oldest discipline in dentistry (oral surgery [exodontia] is the oldest). Root form implant history dates back thousands of years and includes civilizations such as the ancient Chinese who, 4000 years ago, carved bamboo sticks in the shape of pegs and drove them into the bone for fixed tooth replacement. The Egyptians, 2000 years ago, used precious metals with a similar peg design. A skull was found in Europe with a ferrous metal tooth inserted into a skull with a tooth peg design that dated back to the time of Christ. Incas from Central America took pieces of sea shells and, similar to the ancient Chinese, tapped them into the bone to replace missing teeth[5] (Figure 2-1). In other words, history shows that it has always made sense to replace a tooth with an implant in the approximate shape of a tooth. In reality, if the lay public were given a choice to replace a missing tooth with an implant or to grind down several adjacent teeth and connect them with a prosthesis to replace a missing tooth and then attempt to make the adjacent teeth look similar to the condition prior to their preparation, the implant would be the obvious choice.

Maggiolo introduced the more recent history of implant dentistry in 1809 with use of gold in the shape of a tooth root.[6] In 1887, Harris reported the use of teeth made of porcelain into which lead-coated platinum posts were fitted.[7] Many materials were tested, and in the early 1900s, Lambotte fabricated implants of aluminum, silver, brass, red copper, magnesium, gold, and soft steel plated with gold and nickel.[8] He identified the corrosion of several of these metals in body tissues related to electrolytic action. The first root form design that differed significantly from the shape of a tooth root was the Greenfield latticed-cage design in 1909, made of iridoplatinum.[9] This was also the first two-piece implant, which separated the abutment from the endosteal implant body at the initial placement. The surgery was designed to use a calibrated trephine bur to maintain an inner core of bone within the implant body. The implant crown was connected to the implant body with an antirotational internal attachment after several weeks. Reports indicate this implant had a modicum of success. Seventy-five years later, this implant design was reintroduced by ITI in Europe and later by Core-Vent in the United States.[10,11]

Surgical cobalt chromium molybdenum alloy was introduced to oral implantology in 1938 by Strock (Boston, Mass.) when he replaced a single maxillary left incisor tooth[12] with a root form, one-piece implant

Figure 2-1 This mandible, dated AD 600, was found in Honduras. This jaw shows three carved, implanted incisors made from carved sea shells. Calculus formation on these three implants indicate this was not a burial ceremony, but a fixed, functional, and esthetic tooth replacement.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

that lasted more than 15 years. A direct bone-implant interface to titanium was initially called *bone fusing* and was first reported in 1940 by Bothe and coworkers.[13] In 1946, Strock designed the first titanium, two-piece screw implant that was initially inserted without the permucosal post. The abutment post and individual crown were added after complete healing.[14] The desired implant interface described by Strock was a direct bone-implant connection, which was called *ankylosis.* The first submerged implant placed by Strock was still functioning 40 years later[15] (Figure 2-2).

Brånemark began extensive experimental studies in 1952 on the microscopic circulation of bone marrow healing. These studies led to a dental implant application in the early 1960s in which a 10-year implant integration was established in dogs without significant adverse reactions to hard or soft tissues. Implant clinical studies in humans with the Brånemark philosophy began in 1965, were followed for 10 years, and were reported in 1977.[16] The term *osseointegration* (rather than *bone fusing* or *ankylosis*) was defined by Brånemark as a direct contact of living bone with the surface of an implant at the light microscopic level of magnification.[17] The terms *bone fusing, ankylosis,* and *osseointegration* may be interchangeable and may address the microscopic bone-implant interface. The percentage of direct bone-implant contact was not initially addressed and has been found to be highly variable. Today, the term *osseointegration* has become common in the implant discipline and describes not only a microscopic condition, but also the clinical condition of rigid fixation. *Rigid fixation* is a clinical term that implies no observable movement of the implant when a force of 1 to 500 g is applied. The prefix *osteo* (e.g., *osteo*blast, *osteo*tomy) also is widely used by the profession. Rigid fixation is the clinical result of a direct bone interface but has also been reported with a fibrous tissue interface.[18]

The osseointegration concepts of Brånemark have been promoted more than those of any other person in recent history. The documentation of past clinical case studies, research of surgery and bone physiology, healing of soft and hard tissues, and restorative applications from Brånemark's laboratory were unprecedented. Adell et al. published the Brånemark 15-year clinical case series report in 1981 on the use of implants in completely edentulous jaws.[19] Approximately 90% of the reported anterior mandibular implants that were in the mouths of patients after the first year of loading were still in function 5 to 12 years later. Lower survival rates were observed in the anterior maxilla. In the initial Brånemark clinical reports, no implants were inserted into the posterior regions of the mouth, and all reported prostheses were fixed restorations.

The use of dental implants in the treatment of complete and partial edentulism has become an integral treatment modality in restorative dentistry.[20-22] The 1988 National Institutes of Health consensus panel on

Figure 2-2 The patient shown here received a two-stage endosteal implant (invented by Strock in 1948) to replace a maxillary lateral incisor. The patient presented in 1986 with the implant still in function 38 years later.

dental implants recognized that restorative procedures using implants differ from those of traditional dentistry and stressed the necessity for advanced education.[22] During the past 15 years, implant dentistry has become a routine method to replace teeth in a restorative practice.

Many practitioners are taught the use of a specific manufacturer's implant system, rather than the theory and comprehensive practice of implant dentistry. The increasing number of manufacturers entering the field use trade names for their implant components (often unique to a particular system), and such names have proliferated to the point of creating confusion. Several different terms or abbreviations now exist that describe similar basic components.[23-28]

To make conditions worse, in the team approach to implant treatment, the widening referral base often requires that the restoring practitioner be knowledgeable regarding many implant systems. With the required knowledge of multiple systems and the lack of uniformity in component names, communication is hampered among manufacturers, dentists, staff, laboratory technicians, students, and researchers. In addition, the incorporation of implant dentistry into the curriculum of most predoctoral and postdoctoral programs further emphasizes the need for standardization of terms and components in implant dentistry.[29]

GENERIC PROSTHETIC COMPONENT TERMINOLOGY

A generic language for endosteal implants was developed by Misch and Misch in 1992.[30] The order in which it is presented follows the chronology of insertion to restoration. In formulating the terminology, five commonly used implant systems in the United States were referenced. Fifteen years later, the dramatic evolution of the U.S. implant market has resulted in changes in nearly

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Figure 2-3 Implant body designs generally relate to three different categories: cylinder implants *(top row)*, screw design implants *(middle row)*, or a combination *(bottom row)*, which usually are pressed into position and have a macro body design similar to a thread form. *(Courtesy Charles English.)*

all the implant lines and component designs. In 2000 the U.S. market alone had to choose from more than 1300 different implant designs and 1500 abutments in various materials, shapes, sizes, diameters, lengths, surfaces, and connections.[31] More than ever, a common language is needed. In pharmacology the variety of pharmaceutical components makes it impossible to list them all by proprietary names, but a list by category of drugs is useful. Likewise, implant components still can be classified into broad application categories, and the practitioner should be able to recognize a certain component category and know its indications and limitations.

This book incorporates a generic terminology, first introduced by Misch and Misch for endosteal implants, that blends a continuity and familiarity of many implant systems with established definitions from the terms of the *Illustrated Dictionary of Dentistry* and the glossaries from *Terms of The Academy of Prosthodontics, American Academy of Implant Dentistry,* and *International Congress of Oral Implantologists.*[1,2,32,33]

GENERIC IMPLANT BODY TERMINOLOGY

Root form implants are a category of endosteal implants designed to use a vertical column of bone, similar to the root of a natural tooth. Although many names have been applied, the 1988 National Institutes of Health consensus statement on dental implants and the American Academy of Implant Dentistry recognized the term *root form.*[1,22,33] The exponential growth of implant use over the last 20 years has been paralleled by an explosion of the implant manufacturing field.[34] There are currently more than 90 implant body designs available, offering countless combinations of design features: screws, baskets, plateaus, balls, cylinders, diameters, lengths, prosthetic connections, and surface conditions[35-54] (Figure 2-3).

The most common root form design combines a separate implant body and prosthodontic abutment to permit only the implant body placement during bone healing. A second procedure is required to attach the implant abutment. The design and surgical philosophy is to achieve clinical rigid fixation that corresponds to a microscopic direct bone-to-implant interface without intervening fibrous tissue occurring over any significant portion of the implant body before the prosthetic phase of the procedure. Over the years, three different surgical approaches have been used for the two-piece implant systems: one stage, two stage, and immediate restoration (loading) (Figure 2-4). The two-stage surgical process places the implant body below the soft tissue, until the initial bone healing has occurred. During a

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

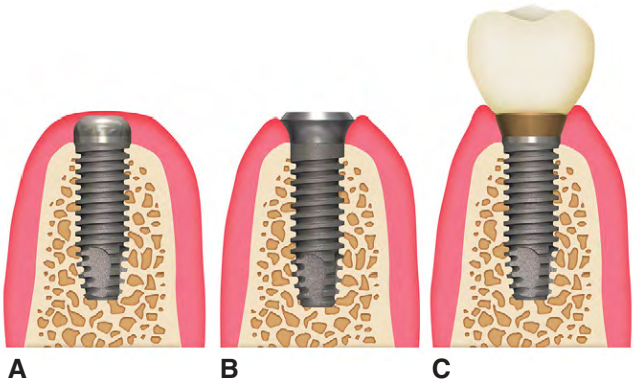


Figure 2-4 There are three different surgical approaches for two-piece implant systems: **(A)** two stage (healing submerged, then uncovery surgery), **(B)** one stage (implant with permucosal healing, no uncovery surgery), and **(C)** immediate restoration (restoration placed at the time of the surgical placement).

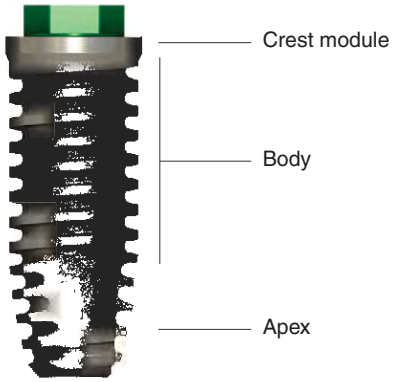


Figure 2-5 An implant body is the portion of the dental implant that is designed to be placed into the bone to anchor prosthetic components. The implant body has a crest module, a body, and an apex.

second-stage surgery, the soft tissues are reflected to attach a permucosal element or abutment. During a one-stage surgical approach, the implant body and the permucosal abutment above the soft tissue are both placed until initial bone maturation has occurred. The abutment of the implant then replaces the permucosal element without the need for a secondary soft tissue surgery.

The immediate restoration approach places the implant body and the prosthetic abutment at the initial surgery. A restoration is then attached to the abutment (out of occlusal contacts in partially edentulous patients) at the appointment.

An implant body especially designed for one surgical method may also be selected. For example, a permucosal element may already be attached to the implant body by the manufacturer to facilitate a one-stage surgical approach. An implant body also may have a prosthetic abutment, which may be part of the implant body, for the one-piece implant to be inserted and restored at the initial surgery. This was the original concept first introduced by Strock in the 1930s.[12]

There are three primary types of root form body endosteal implants based on design; cylinder, screw, or combination.[30]

Cylinder (press-fit) root form implants depend on a coating or surface condition to provide microscopic retention to the bone. Most often the surface is either coated with a rough material (e.g., hydroxyapatite, titanium plasma spray) or a macro retentive design (e.g., sintered balls). Cylinder implants are usually pushed or tapped into a prepared bone site. They can be a paralleled wall cylinder or a tapered implant design. Screw root forms are threaded into a slightly smaller prepared bone site and have the macroscopic retentive elements of a thread for initial bone fixation. They may be machined, textured, or coated. There are three basic screw-thread geometries: V-thread, buttress (or reverse buttress) thread, and power (square) thread designs. Threaded implants are primarily available in a parallel cylinder or tapered cylinder design. Micro or macro thread features, variable thread pitch, depth, and angle, as well as self-tapping features, can be combined to create a myriad of implant designs. Combination root forms have macroscopic features from both the cylinder and screw root forms. The combination root form designs also may benefit from microscopic retention to bone through varied surface treatments (machined, textured, and the addition of coatings).[55-68] Root forms also have been described by their means of insertion, healing, surgical requirements, surface characteristics, and interface.[28,69,70]

IMPLANT BODY REGIONS

The implant body may be divided into a crest module (cervical geometry), a body, and an apex (Figure 2-5). Each section of an implant has features that are of benefit in the surgical or prosthetic application of the implant.

Implant Body

An implant body is primarily designed for either surgical ease or prosthetic loading to the implant bone interface. Years ago, the implant body was the primary design feature. A round implant permits round surgical drills to prepare the bone. A smooth-walled cylinder implant allows the implant to be pressed or tapped into position, similar to a nail into a piece of wood. A tapered cylinder fits into the top of the osteotomy for further ease of placement.

A cylinder implant design system offers the advantage of ease of placement, even in difficult access locations. The cover screw of the implant also may be attached to the implant before implant placement. For example, in the very soft D4 bone of the posterior regions of the

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

maxilla, the surgeon must rotate a threaded implant design into place. Very soft bone may strip during a threaded implant insertion. This may result in lack of initial fixation, and the implant will not be rigid. A tapered cylinder implant may be pressed by hand into soft bone and can be initially fixated more easily. The speed of implant rotation during insertion and the amount of apical force in implant insertion in soft bone are less relevant for a press-fit cylinder. The cylinder system also presents some benefits for the single-tooth implant application, especially if adjacent to teeth with tall clinical crowns. Thread extenders are needed for the screw implant placement in these situations, as well as additional tools to insert the cover screw of the implant. In dense bone, cylinder systems also are easier and faster to place because bone tapping is not required.

Most cylinder implants are essentially smooth-sided and bullet-shaped implants that require a bioactive or increased surface area coating for retention in the bone. When these materials are placed on an implant, the surface area of bone contact increases more than 30%. The greater the functional surface area of the bone implant contact, the better the support system for the prosthesis.

A solid screw implant body design is the most commonly reported in the literature. A solid screw body is defined as an implant of a circular cross section without penetrating any vents or holes. A number of manufacturers provide this design (e.g., Nobel Biocare, Biomet, Zimmer, ITI, BioHorizons, LifeCore, Bio-Lok). The thread may be V-shaped, buttress, reverse buttress, or square (power thread) in design. The V-shaped threaded screw has the longest history of clinical use.[16,17] The most common outer thread diameter is 3.75 mm, with a 0.38-mm thread depth, and a 0.6-mm thread pitch (distance). The various body lengths usually range from 7 to 16 mm, although lengths from 5 mm to 56 mm are available. Similar body designs are offered in a variety of diameters (narrow, standard, wide) to respond to the mechanical, esthetic, and anatomical requirements in different areas of the mouth.[31,71]

A solid screw implant body permits the osteotomy and placement of the implant in dense cortical bone as well as in fine trabecular bone. The surgery may be easily modified to accommodate both extremes in bone density. The solid screw permits the implant removal at the time of surgery if placement is not ideal. It also permits implant removal at the Stage II surgery if angulation or crestal bony contours are not deemed adequate for long-term prosthesis success. The solid screw implant body may be machined or roughened to increase marginally the functional surface area or to take advantage of biochemical properties related to the surface coating (e.g., bone bonding or bone growth factors).

A threaded implant body is primarily designed to increase the bone-implant surface area and to decrease the stresses at the interface during occlusal loading. The functional surface area of a threaded implant is greater than a cylinder implant by a minimum of 30% and may exceed 500%, depending on the thread geometry. This increase in functional implant surface area decreases the stress imposed on the implant-bone interface and is directly related to the thread geometry.

Crest Module

The crest module of an implant body is that portion designed to retain the prosthetic component in a one-piece or two-piece implant system. It also represents the transition zone from the implant body design to the transosteal region of the implant at the crest of the ridge. The abutment connection area usually has a platform on which the abutment is seated; the platform offers physical resistance to axial occlusal loads. An antirotation feature also is included on the platform (external hex) or extends within the implant body (internal hex, octagon, Morse taper or cone screw, internal grooves or cam tube, and pin slots). The implant body has a design to transfer stress/strain to the bone during occlusal loads (e.g., threads or large spheres), whereas the crest module often is designed to reduce bacterial invasion. (e.g., smoother to impair plaque retention if crestal bone loss occurs). Its smoother dimension varies greatly from one system to another (0.5 to 5 mm). When the crest module is smooth, polished metal, it is often called a *cervical collar*.

A high-precision fit of the external or internal anti-rotational component (flat to flat dimension) is paramount to the stability of the implant body/abutment connection.[31,71-74] The prosthetic connection to the crest module is received by slip-fit or friction-fit with a butt or bevel joint. All prosthetic connections aim at providing a precise mating of the two components with minimal tolerance.

Another antirotational feature of an implant body may be flat sides or grooves along the body or apical region of the implant body. When bone grows against the flat or groove regions, the bone is placed in compression with rotational loads. The apical end of each implant should be flat rather than pointed. This allows for the entire length of the implant to incorporate design features that maximize desired strain profiles. Additionally, if an opposing cortical plate is perforated, a sharp, V-shaped apex may irritate or inflame the soft tissues if any movement occurs (e.g., the inferior border of the mandible).

IMPLANT SURGERY

At the time of insertion of a two-stage implant body (stage I surgery), a first-stage cover screw is placed into the top of the implant to prevent bone, soft tissue, or

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

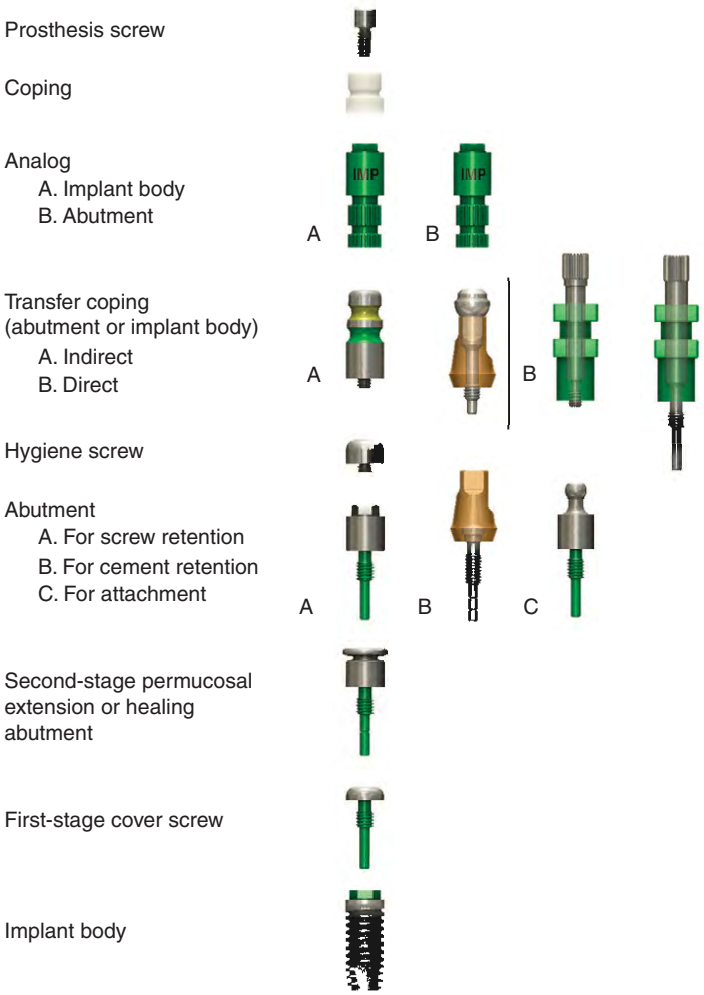


Figure 2-6 Implant components frequently have terms that are different for each company, but a generic language exists that applies to any product. This language permits improved communication between referring doctors and laboratories, which often must be familiar with several different systems.

debris from invading the abutment connection area during healing (Figure 2-6).

After a prescribed healing period sufficient to allow a supporting bone interface to develop, a second-stage procedure may be performed to expose the two-stage implant or to attach a transepithelial portion.[32] This transepithelial portion is termed a *permucosal extension* because it extends the implant above the soft tissue and results in the development of a permucosal seal around the implant. This implant component has also been called a *healing abutment* because stage II uncovery surgery often uses this device for initial soft tissue healing (Figure 2-7).

In the case of a one-stage procedure, the surgeon may have placed the permucosal extension at the time of implant insertion or may have selected an implant body design with a cervical collar of sufficient height to be supragingival. In the case of immediate load, the permucosal healing abutment may not be used at all if a temporary prosthesis is delivered on the day of surgery or may be used until the suture removal appointment and the temporary teeth delivery. The permucosal extension is available in multiple heights to accommodate soft tissue variations. It also can be straight, flared, or anatomical to assist in the initial contour of the soft tissue healing.

Prosthetic Attachments

The abutment is the portion of the implant that supports or retains a prosthesis or implant superstructure.[32] A *superstructure* is defined as a metal framework that attaches to the implant abutment(s) and provides either retention for a removable prosthesis[1] (e.g., a cast bar retaining an overdenture with attachments) or the framework for a fixed prosthesis. Three main categories of implant abutments are described, according to the method by which the prosthesis or superstructure is retained to the abutment: (1) an abutment for screw retention uses a screw to retain the prosthesis or superstructure (Figure 2-8, *A*), (2) an abutment for cement retention uses dental cement to retain the prosthesis or superstructure (Figure 2-8, *B*), and (3) an abutment for attachment uses an attachment device to retain a removable prosthesis (such as an O-ring attachment) (Figure 2-8, *C*). The abutment for cement/

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

A

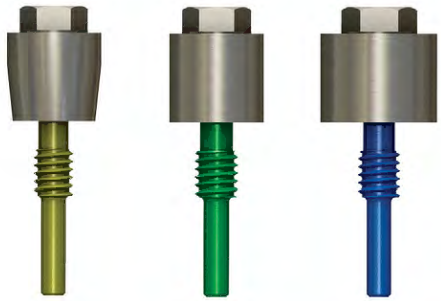
B

Figure 2-7 **A,** The permucosal extension (PME) attaches to the implant body and allows the soft tissue to heal and mature around the future implant abutment. The PME may be the same size as the crest module of the implant body *(left)* or slightly larger *(right)* and helps develop the emergence contour of the implant crown. **B,** An intraoral view of eight second-stage permucosal extensions that were inserted into the implant bodies.

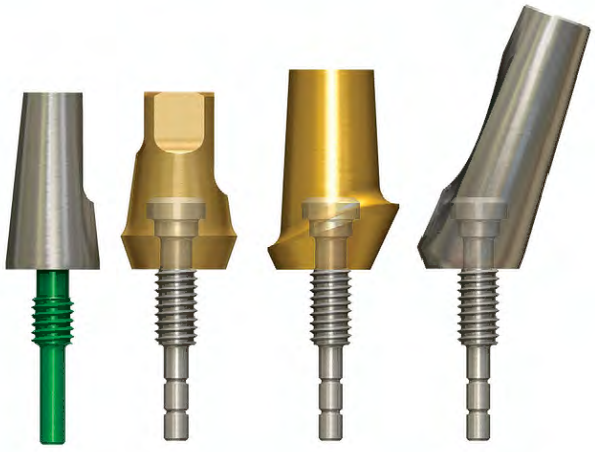
screw/attachment may be screwed or cemented into the implant body, but this aspect is not delineated within the generic terminology.

Each of the three abutment types may be further classified as straight or angled abutments, describing the axial relationship between the implant body and the abutment. An abutment for screw retention uses a hygiene cover screw placed over the abutment to prevent debris and calculus from invading the internally threaded portion of the abutment retention during prosthesis fabrication between prosthetic appointments.

The lack of abutment design of a decade ago has been replaced by a variety of options. The expansion of implant dentistry, its applications for esthetic dentistry, and the creativity of manufacturers in this very competitive market is responsible for the explosion of implant abutment styles available today (Figure 2-9). In the abutment for cement category, the doctor may choose from one- and two-piece abutments; UCLA type (plastic castable, machined/plastic castable, gold sleeve castable); two-piece esthetic; two-piece anatomical; two-piece shoulder; preangled (several angulations); or



A



B

C

Figure 2-8 **A,** An abutment for screw retention is used for a screw-retained bar or fixed prosthesis. **B,** Abutments for cement retention may be one piece *(far left)* or two pieces, which are retained by a separate abutment screw. **C,** Abutments for attachment are used for removable prostheses that are implant retained. These may be used for complete dentures and partial dentures. *(**A,** Courtesy BioHorizons, Birmingham, Ala.)*

ceramic, Zirconia, or computer-assisted custom designs. The abutment for screw category also has been enlarged with one- and two-piece overdenture abutments of different contours and heights.

Many manufacturers classify the prosthesis as *fixed* whenever cement retains the prostheses, *fixed/removable* when screws retain a fixed prosthesis, and *removable* when the restoration is removed by the patient. This description implies that only screw-retained prostheses may be removed. This is not an accurate description, because a fixed, cemented prosthesis also may be

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

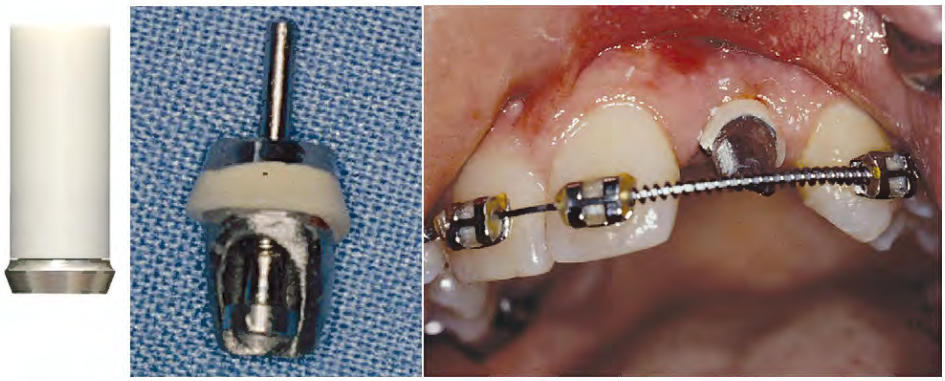


Figure 2-9 **A,** A custom abutment can be fabricated to address specific angulation or esthetic requirements. **B,** On a custom abutment, porcelain may be built between the crown margin and the abutment-to-implant position. This buildup allows the crown margin to be above the bone, yet the subgingival region has pink or tooth-colored porcelain and a customized shape to improve esthetics. **C,** A custom abutment with tooth-colored ceramic and ideal emergence profile from the implant body. If the tissue shrinks in the future, the crown will appear longer, but the titanium color of the abutment will not be visible.

removed by the dentist (especially when a temporary cement is used). The generic language in this chapter separates prostheses into either fixed or removable in a method similar to traditional prosthetics.

Prosthesis Fabrication

An impression is necessary to transfer the position and design of the implant or abutment to a master cast for prosthesis fabrication. A transfer coping is used in traditional prosthetics to position a die in an impression[32] (Figure 2-10). Most implant manufacturers use the terms *transfer* and *coping* to describe the component used for the final impression. Therefore a transfer coping is used to position an analog in an impression and is defined by the portion of the implant it transfers to the master cast, either the implant body transfer coping or the abutment transfer coping.

Two basic implant restorative techniques are used to make a master impression, and each uses a different design transfer coping, based on the transfer technique performed. An indirect transfer coping uses an impression material requiring elastic properties.[32] The indirect transfer coping is screwed into the abutment or implant body and remains in place when a traditional "closed tray" impression is set and removed from the mouth. The indirect transfer coping is usually slightly tapered to allow ease in removal of the impression and often has flat sides or smooth undercuts to facilitate reorientation in the impression after it is removed.

A direct transfer coping usually consists of a hollow transfer component, often square, and a long central screw to secure it to the abutment or implant body and may be used as a pick-up impression coping. An "open tray" impression tray is used to permit direct access to the long central screw securing the indirect transfer coping. After the impression material is set, the direct transfer coping screw is unthreaded to allow removal of the impression from the mouth. Direct transfer copings take advantage of impression materials having rigid properties and eliminate the error of permanent deformation because they remain within the impression until the master model is poured and separated (Figure 2-11).

Laboratory Fabrication

An *analog* is defined as something that is analogous or similar to something else.[32] An implant analog is used in the fabrication of the master cast to replicate the retentive portion of the implant body or abutment (implant body analog, implant abutment analog). After the master impression is obtained, the corresponding analog (e.g., implant body, abutment for screw) is attached to the transfer coping and the assembly is poured in stone to fabricate the master cast (Figure 2-12).

A prosthetic coping is a thin covering,[32] usually designed to fit the implant abutment for screw retention. It serves as the connection between the abutment and the prosthesis or superstructure. A prefabricated coping usually is a metal component machined precisely to fit the abutment. A castable coping usually is a plastic pattern cast in the same metal as the superstructure or prosthesis. A screw-retained prosthesis or superstructure is secured to the implant body or abutment with a prosthetic screw.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

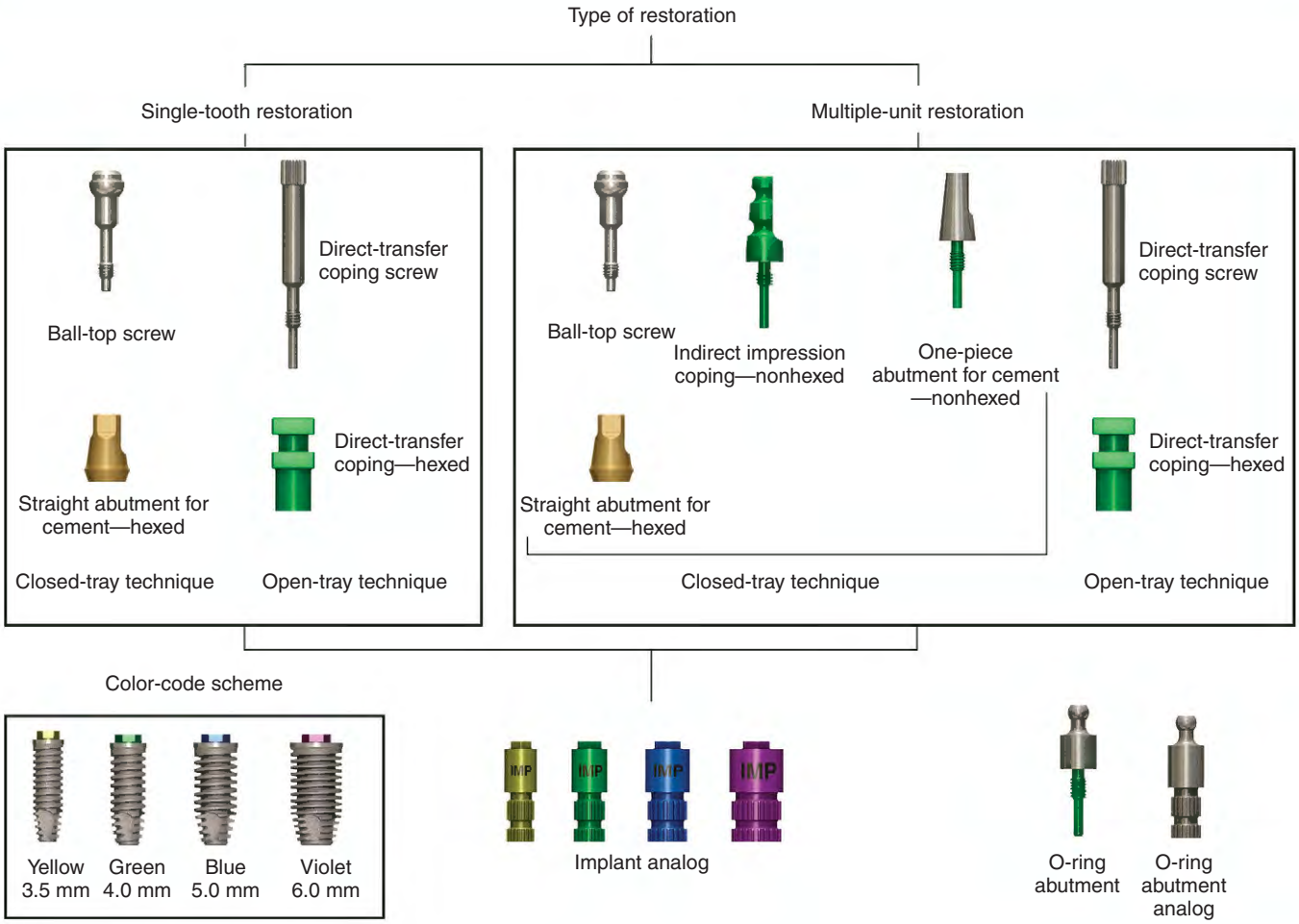


Figure 2-10 An indirect transfer *(far left and center)* is inserted into an implant body or abutment for screw retention, and a closed tray impression is made. The impression is removed, and the transfers are connected to an analog and reinserted into the impression. A direct impression transfer *(far right)* uses an open tray to make the impression. The direct transfer coping screw must be unthreaded before the impression is removed from the mouth. *(Courtesy BioHorizons, Birmingham, Ala.)*

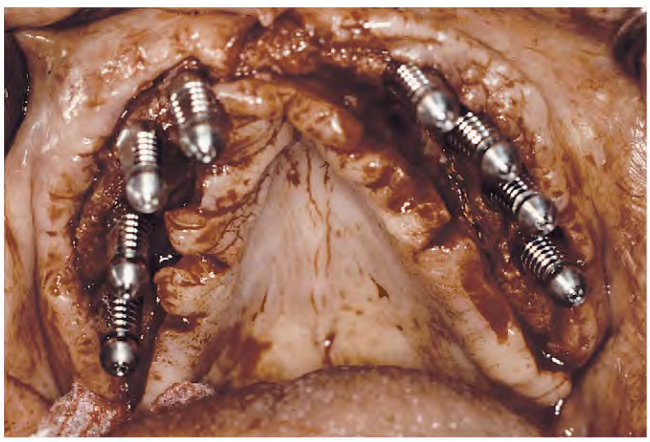


Figure 2-11 These eight maxillary implants are connected to two-piece indirect impression transfers, which engage the hexagon of the implant platform. A closed-tray impression is made, and the indirect transfer copings are unthreaded from the implant bodies, connected to implant body analogs, and reinserted into the impression before pouring of the cast.

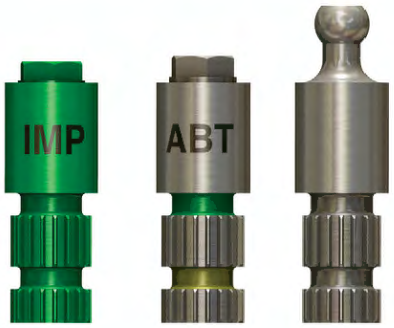
SUMMARY

A generic terminology has been developed to facilitate communication among implant team members. Regardless of the implant system used, the generic term is descriptive of the function of the component, rather than its proprietary name. The surgery phase of treatment most often uses an implant body with a first-stage cover screw. The second-stage surgery removes the first-stage cover screw and inserts a permucosal extension. The restoring dentist removes the permucosal extension and places the abutment or makes an implant body impression.

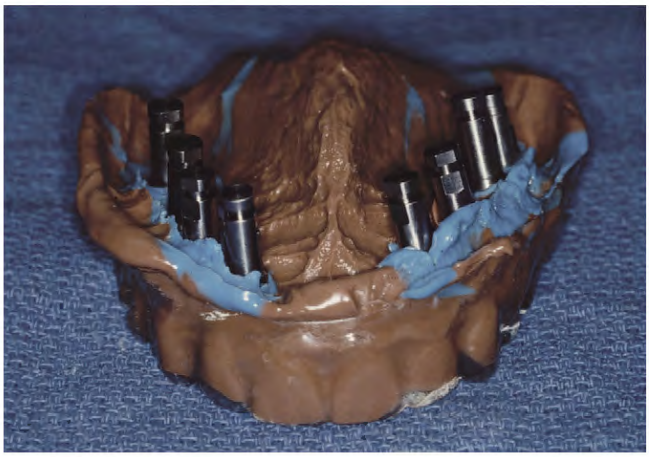
The implant body impression may use a direct or indirect technique. The laboratory uses analogs to fabricate the prosthesis. The prosthesis is most often cement retained in partially edentulous patients and uses an abutment for cement retention. Regardless of the implant system selected, the staff and laboratory should be familiar with generic, descriptive terminology.

t.me/highdent www.highdentlab.com instagram.com/high_dent

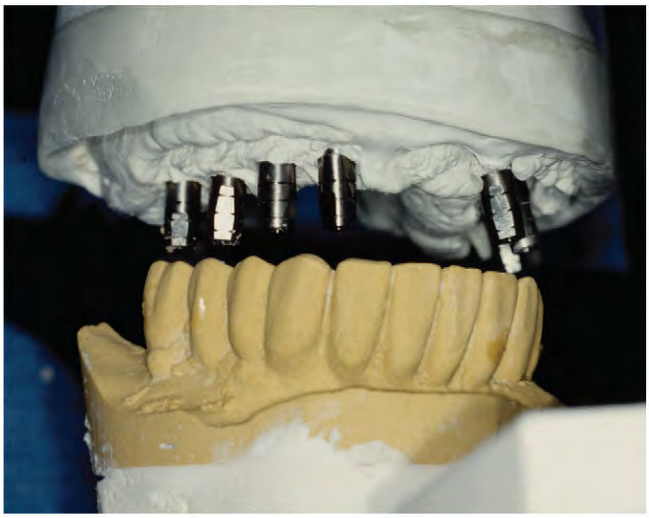
همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت



A



B



C

Figure 2-12 **A,** Analogs may represent an abutment for screw retention, an implant body *(left)*, or an abutment for attachment *(right)*. **B,** An indirect closed-tray impression in a "customized" impression tray, which also relates the incisal edge of the patient to the laboratory technician. The indirect impression transfers have been attached to implant body analogs and reinserted into the closed-tray impression. **C,** The impression in **B** is mounted against the opposing arch (with a bite registration not pictured). In this particular system the indirect transfer coping may be a one- or two-piece component. A one-piece component does not engage the hexagon and does not transfer the position of the antirotational hexagon from the mouth to the model. The two-piece indirect transfer does allow the transfer. The laboratory can remove the indirect transfer copings, insert the abutments for cement retention, and prepare them before making a transition prosthesis. Some dentists also will make a metal casting for the final restoration on this model.

References

1. Cranin AN: Glossary of implant terms, *J Oral Implant* 16:57-63, 1990.
2. Soblonsky S, editor: *Illustrated dictionary of dentistry,* Philadelphia, 1982, WB Saunders.
3. Chercheve R: Endosseous implantology. In Cranin AN, editor: *Oral implantology.* Springfield, Ill, 1970, Thomas.
4. Linkow LI: Endosseous oral implantology: a 7-year progress report, *Dent Clin North Am* 14:185-199, 1970.
5. Anjard R: Mayan dental wonders, *Oral Implant* 9:423, 1981.
6. Maggiolo: *Manuel de l'art dentaire* [Manual of dental art], Nancy, France, 1809, C Le Seure.
7. Harris LM: An artificial crown on a leaden root, *Dent Cosmos* 55:433, 1887.
8. Lambotte A: New instruction for the banding of bone: "banding with a screw," *J C Ann Soc Belge Chir* 9:113, 1909.
9. Greenfield EJ: Implantation of artificial crowns and bridge abutments, *Dent Cosmos* 55:364-430, 1913.
10. Schroeder A, Sutter F, Krekeler G, editors: *Orale Implantologie. Allegemeine Grundlagen und ITI Hohlzlindersystem,* Stuttgart, 1988, Thieme.
11. Niznick GA: The Core-Vent implant system, *Oral Health* 73:13-17, 1983.
12. Strock AE: Experimental work on dental implantation in the alveolus, *Am J Orthod Oral Surg* 25:5, 1939.
13. Bothe RT, Beaton LE, Davenport HA: Reaction of bone to multiple metallic implants, *Surg Gynecol Obstet* 71: 598-602, 1940.
14. Strock AE, Strock MS: Further studies on inert metal implantation for replacement, *Alpha Omega,* Sept 1949.
15. Shulman L: Personal communication, 1990.
16. Brånemark PI, Hansson BO, Adell R et al: Osseointegrated implants in the treatment of the edentulous jaw: experience from a 10-year period, *Scand J Plast Reconstr Surg Suppl* 16:1-132, 1977.
17. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 6:387, 1981.
18. Koth DL, McKinney RV. The single crystal sapphire endosteal dental implant. In Hardin JF: *Clark's clinical dentistry,* Philadelphia, 1981, JB Lippincott.
19. Adell R, Ericsson B, Lekholm U et al: A long-term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws, *Int J Oral Maxillofac Impl* 5:347-359, 1990.
20. van Steenberghe D, Lekholm U, Bolender C et al: The applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study on 558 fixtures, *Int J Oral Maxillofac Impl* 3:272-281, 1990.
21. Kline R, Hoar JE, Beck GH et al: A prospective multicenter clinical investigation of bone quality-based dental implant system, *Implant Dent* 11:224-234, 2002.
22. National Institutes of Health consensus development conference statement on dental implants, *J Dent Educ* 52:824-827, 1988.
23. 3i, Osseotite, Certain and external hex restorative and surgical manuals, Palm Beach Garden, Fla, 2005.
24. Straumann Dental Implants Product Catalog, Andover, Mass, 2005.
25. Maestro and Prodigy surgical and prosthetic manuals, Birmingham, Ala, 2005, BioHorizons Implant Systems.
26. *Nobel Perfect Implant Placement/Restorative Manual,* Goteburg, Sweden, 2005, Nobelbiocare AB.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

27. Screw-Vent and Advent surgical and prosthetic manuals, Carlsbad, Calif, 2005, Zimmer Dental.
28. English CE: Implants—part three: an overview, *CDA J* 16:34-38, 1988.
29. Misch CE: Dental education: meeting the demands of implant dentistry, *J Am Dent Assoc* 121:334-338, 1990.
30. Misch CE, Misch CM: Generic terminology for endosseous implant prosthodontics, *J Prosthet Dent* 68:809-812, 1992.
31. Binon PP: Implants and components: entering the new millennium, *Int J Oral Maxillofac Surg* 15:76-94, 2000.
32. The glossary of prosthodontic terms, *J Prosthet Dent* 94: 10-92, 2005.
33. Jalbout Z, Tabourina G, *International Congress of Oral Implantologyists*: *glossary of implant terms,* Upper Montclair, NJ, 2003, ICOI/NYU.
34. Millenium Research Group report: *U.S. markets for dental implants 2003*, USDI 03, Toronto, January 2003.
35. English CE: Externally hexed implants, abutments, and transfer devises: a comprehensive overview, *Implant Dent* 1:273-282, 1992.
36. Jemt T: Fixed implant supported prostheses in the edentulous maxilla: a five-year follow-up report, *Clin Oral Implants Res* 5:142-147, 1994.
37. Lekholm U, van Steenberghe D, Herrmann I et al: Osseointegrated implants in the treatment of partially edentulous jaws: a prospective 5-year multicenter study, *Int J Oral Maxillofac Implants* 9:627-635, 1994.
38. Wie H: Registration of localization, occlusion and occluding materials for failing screw-joints in the Brånemark implant system, *Clin Oral Implants Res* 6:47-53, 1995.
39. Becker W, Becker BE: Replacement of maxillary and mandibular molars with single endosseous implant restorations: a retrospective study, *J Prosthet Dent* 74: 51-55, 1995.
40. Balshi TJ: First molar replacement with osseointegrated implants, *Quintessence Int* 21:61-65, 1990.
41. Balshi TJ, Hernandez RE, Pryszlak MC: A comparative study of one implant versus two replacing a single molar, *Int J Oral Maxillofac Implants* 11:372-378, 1996.
42. Eckert SE, Wollan PC: Retrospective review of 1170 endosseous implants placed in partially edentulous jaws, *J Prosthet Dent* 79:415-421, 1998.
43. Niznick GA: The implant abutment connection: the key to prosthetic success, *Compend Contin Educ Dent* 12:932-937, 1991.
44. Schulte W, d'Hoedt B, Axmann D et al: The first 15 years of the Tuebinger implant and its further development to the Frialit-2 system, *Z Zahnarztl Implantol* 8:3-22, 1992.
45. Arvidson K, Bystedt I, Ericsson I: Histometric and ultrastructural studies of tissues surrounding Astra dental implants in dogs, *Int J Oral Maxillofac Implants* 5:127-134, 1990.
46. Binon PP: The Spline implant: design, engineering, and evaluation, *Int J Prosthodont* 9:419-433, 1996.
47. Patrick D, Zosky J, Lubar R et al: The longitudinal clinical efficacy of Core-Vent dental implants: a five year study, *J Oral Implantol* 15:95-103, 1989.
48. Langer B, Langer L, Herrmann I et al: The wide fixture: a solution for special bone situations and a rescue for the compromised implant—part 1, *Int J Oral Maxillofac Implants* 8:400-408, 1993.
49. Graves SL, Jansen CE, Siddiqui M et al: Wide diameter implants: indications, considerations and preliminary results over a two year period, *Aust Prosthodont J* 8:31-37, 1994.
50. d'Hoedt B: *Dentale Implantate aus polykristaliner aluminiumoxydkeramik Einheilung und Langzeitergebrusse* [thesis; in German]. Tübingen, Germany, 1991, University of Tübingen.
51. Gomez-Roman G, Schulte W, d'Hoedt B et al: The Frialit-2 implant system: five year clinical experience in single tooth and immediately post extraction applications, *Int J Oral Maxillofac Implants* 12:299-309, 1997.
52. Sutter F, Weber IIP, Sorensen J et al: The new restorative concept of the ITI dental implant system: design and engineering, *Int J Periodontics Restorative Dent* 13:409-431, 1993.
53. Deporter DA, Watson PA, Pilliar RM et al: A histological comparison in the dog of porous coated vs. threaded dental implant, *J Dent Res* 69:1138-1143, 1990.
54. Strong TJ, Misch CE, Bidez NW et al: Functional surface area: thread-form parameter optimization for implant body design, *Compend Contin Educ Dent* 19:4-9, 1998.
55. Brunette DM: The effects of implant surface topography on the behavior of cell, *Int J Oral Maxillofac Implants* 3:231-246, 1988.
56. Carlsson L, Rostlund T, Albrektsson B et al: Removal torques for polished and rough titanium implants, *Int J Oral Maxillofac Implant* 3:21-24, 1988.
57. Kieswetter K, Schwartz Z, Dean DD et al: The role of implant surface characteristics in the healing of bone, *Crit Rev Oral Biol Med* 7:329-345, 1996.
58. Buser D, Schenk RK, Steinemann S et al: Influence of surface characteristics on bone integration of titanium implants: a histomorphometric study in miniature pigs, *J Biomet Mater Res* 25:889-902, 1991.
59. Cochran DL, Schenk RK, Lussi A et al: Bone response to unloaded and loaded titanium implants with sand-blasted and acid etched surfaces: a histometric study in the canine mandible, *J Biomed Mater Res* 40:1-11, 1998.
60. Wennerberg A, Albrektsson T, Andersson B: Bone tissue response to commercially pure titanium implant blasted with fine and coarse particles of aluminum oxide, *Int J Oral Maxillofac Implants* 11:38-45, 1996.
61. Klokkevold PR, Nishimura RD, Adachi M et al: Osseointegration enhanced by chemical etching of titanium surface: a torque removal study on the rabbit, *Clin Oral Implants Res* 8:442-447, 1997.
62. Lazzara RJ, Testori T, Trisi P et al: A human histologic analysis of osseotite and machined surfaces using implants with 2 opposing surfaces, *Int J Periodontics Restorative Dent* 19:117-129, 1999.
63. Deporter DA, Watson PA, Pilliar RM et al: A clinical trial of a partially porous coated endosseous dental implant in humans: protocol and 6 month results. In Laney WR, Tolman DE, editors: *Tissue integration in oral orthopedic and maxillofacial reconstruction*, Chicago, 1992, Quintessence.
64. de Groot K, Geesink R, Klein CP et al: Plasma sprayed coatings of hydroxylapatite, *J Biomed Mater Res* 21:1375-1381, 1987.
65. Block MS, Kent JN, Kay JF: Evaluation of hydroxylapatite coated titanium implants in dogs, *J Oral Maxillofac Surg* 45:601-607, 1987.
66. Cook SD, Kay JF, Thomas KA et al: Interface mechanics and histology of titanium and hydroxylapatite coated titanium for dental implant applications, *Int J Oral Maxillofac Implants* 2:15-22, 1987.
67. Burgess AV, Story BJ, La D et al: Highly crystalline MP-I hydroxylapatite coating. Part I: in vitro characterization and comparison to other plasma sprayed hydroxylapatite coatings, *Clin Oral Implants Res* 10:245-256, 1999.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

68. Hoar JE, Beck GH, Crawford EA et al: Prospective evaluation of crestal bone remodeling of a bone density-based dental implant system, *Compendium* 19:17-24, 1998.
69. English CE: Implants—part I: cylindrical implants, *CDA J* 16:17-26, 1988.
70. English CE: Implants—part II: questions need answering, *CDA J* 16:26-34, 1988.
71. Binon PP: Evaluation of three slip fit hexagonal implant, *Implant Dent* 5:235-248, 1996.
72. Sutter F, Schroeder A, Buser DA: The new concept of ITI hollow-cylinder and hollow-screw implants—part 1: engineering and design, *Int J Oral Maxillofac Implants* 3:161-172, 1988.
73. Binon PP: The evolution and evaluation of two interference-fit implant interfaces, *Postgraduate Dent* 2: 1-15, 1996.
74. Boggan RS, Strong JT, Misch CE et al: Influence of hex geometry and prosthetic table width on static and fatigue strength of dental implants, *J Prosthet Dent* 82:436-440, 1999.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Chapter 3

Diagnostic Imaging and Techniques

Randolph R. Resnik, Louis T. Kircos, Carl E. Misch

Diagnostic imaging and techniques help develop and implement a cohesive and comprehensive treatment plan for the implant team and the patient. The implant team requires the services or functions of a number of professionals and may include the referring dentist, laboratory technician, prosthodontist, periodontist, oral surgeon, implantologist, anesthesiologist, radiologist, hygienist, and staff. Information acquired from the patient's medical and dental history, including clinical examination, laboratory tests, diagnostic casts, diagnostic wax-up, and diagnostic imaging, plays a role in developing the patient's treatment plan and objectives.

IMAGING OBJECTIVES

The objectives of diagnostic imaging depend on a number of factors, including the amount and type of information required and the period of the treatment rendered. The decision of when to image along with which imaging modality to use depends on the integration of these factors and can be organized into three phases.

Phase 1

Phase 1 is termed *presurgical implant imaging* and involves all past radiologic examinations and new radiologic examinations chosen to assist the implant team in determining the patient's final and comprehensive treatment plan. The objectives of this phase of imaging include all necessary surgical and prosthetic information to determine the quantity, quality, and angulations of bone; the relationship of critical structures to the prospective implant sites; and the presence or absence of disease at the proposed surgery sites.

Phase 2

Phase 2 is termed *surgical and intraoperative implant imaging* and is focused on assisting in the surgical and prosthetic intervention of the patient. The objectives of this phase of imaging are to evaluate the surgery sites during and immediately after surgery, assist in the optimal position and orientation of dental implants, evaluate the healing and integration phase of implant surgery, and ensure that abutment position and prosthesis fabrication are correct.

Phase 3

Phase 3 is termed *postprosthetic implant imaging*. This phase commences just after the prosthesis placement and continues as long as the implants remain in the jaws. The objectives of this phase of imaging are to evaluate the long-term maintenance of implant rigid fixation and function, including the crestal bone levels around each implant, and to evaluate the implant complex.

IMAGING MODALITIES

The decision to image the patient is based on the patient's clinical needs. After a decision has been made to obtain images, the imaging modality is used that yields the necessary diagnostic information related to the patient's clinical needs and results in the least radiologic risk. The opinion of a radiologist may be required for more complex modalities or for situations in which the attending dentist is less experienced.

Maximizing the ratio of benefit to risk for imaging examinations is a fundamental tenet of radiology. Examinations known to produce this result are not necessarily the examinations that cost the least, are in proximity to the dentist, or produce the lowest radiation exposure.[1-3] However, they enable the dentist to provide the proper care or treatment for the patient.

Many imaging modalities have been reported as useful for dental implant imaging, including devices recently developed specifically for dental implant imaging (Box 3-1).[4,5]

These imaging modalities can be described as analog or digital and two- or three-dimensional. Most dentists are more familiar with analog two-dimensional imaging (see Box 3-1). Analog imaging modalities are two-

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Box 3-1 Types of Imaging Modalities

- Periapical radiography (analog)
- Panoramic radiography (analog)
- Occlusal radiography (analog)
- Cephalometric radiography (analog)
- Tomographic radiography
- Computed tomography (three-dimensional)
- Magnetic resonance imaging (three-dimensional)
- Interactive computed tomography (three-dimensional)

dimensional systems that use radiograph film or intensifying screens as the image receptors. The image quality of these systems is characterized by resolution/modulation transfer function, contrast/H and D curve, noise/Weiner spectrum, and sensitivity.[6] The clinical performance of these imaging systems is gauged by receiver operator characteristics.[7,8]

Digital images also can be produced with each imaging modality (see Box 3-1). A digital two-dimensional image is described by an image matrix that has individual picture elements called *pixels*. A digital image is described by its width and height and pixels (i.e., 512×512). For larger digital images (i.e., $1.2\ M \times 1.2\ M$, where *M* is megapixels), the image is alternatively described as a 1.5-M image. Each picture element, or pixel, has a discrete digital value that describes the image intensity at that particular point. The value of a pixel element is described by a scale, which may be as low as 8 bits (256 values) or as high as 12 bits (4096 values) for black-and-white imaging systems, or 36 bits (65 billion values) for color imaging systems.[9-11] Black-and-white digital images are displayed optimally on a dedicated black-and-white monitor. Generally, 8 bits or 256 levels can be displayed effectively on a monitor.

A digital three-dimensional image is described by an image matrix that has individual image/picture elements called *voxels*. A digital three-dimensional image is described not only by its width and height and pixels (i.e., 512×512) but also by its depth/thickness. An imaging volume or three-dimensional characterization of the patient is produced by contiguous images, which produce a three-dimensional structure of volume elements (i.e., computed tomography [CT], magnetic resonance imaging [MRI], and interactive computed tomography [ICT]). Each volume element has a value that describes its intensity level. Typically, three-dimensional modalities have an intensity scale of 12 bits or 4096 values. Box 3-1 identifies the three-dimensional imaging modalities.

PRESURGICAL IMAGING (PHASE 1)

In the field of oral implantology, there exist numerous radiographic imaging modalities available for the presurgical assessment of the dental implant patients. In the past, intraoral radiographs along with panoramic images were used as the sole determinants of implant diagnosis and treatment planning. With the advancement of radiographic technology, various three-dimensional imaging systems are now available to the dental profession allowing the implant team an infinite amount of diagnostic information.

The goal of presurgical radiographic evaluation is to assess the available bone quality and quantity, angulation of bone, selection of potential implant sites, and to verify absence of pathology. However, there exists no ideal radiographic imaging technique in the field of oral implantology that would be acceptable for all patients. All imaging techniques in the field of dentistry have inherent advantages and disadvantages and have been shown to exhibit false-negative and false-positive images.[12-14]

When selecting a radiographic modality for preoperative assessment, a careful examination of the available imaging options should be evaluated for selection as per the patient's needs. In dental and medical radiology, a recommended principle when selecting the appropriate radiographic modality is based on radiation dosage. The "as low as reasonably achievable" (ALARA) principle should always be adhered to that states that the diagnostic imaging technique selected should include the lowest possible radiation dose to the patient. However, patient care and treatment planning should not be jeopardized in response to radiation dose. Studies have shown that an overwhelming number (90%) of dentists prescribe panoramic images as the sole determinant for implant treatment planning.[15,16] In comparison, less than 10% prescribe conventional or CT. On the other hand, the American Academy of Oral and Maxillofacial Radiology guidelines state that all implant site evaluations should be evaluated with a three-dimensional imaging technique such as conventional or computerized tomography.[17]

This phase of implant imaging is intended to evaluate the current status of the patient's teeth and jaws and to develop and refine the patient's treatment plan. Evaluation of the patient by members of the dental implant team is accomplished with a review of the patient's history, a thorough clinical examination, and a review of the patient's radiologic examinations. At this point, the dentist should be able to rule out dental or bone disease and establish a tentative clinical objective that meets the patient's functional and esthetic needs. If the dentist cannot rule out dental or bone disease, further clinical or radiologic examination is necessary.

The global objective of this phase of treatment is to develop and implement a treatment plan for the patient that enables restoration of the patient's function and esthetics by the accurate and strategic placement of dental implants. The patient's functional and esthetic needs can be transformed physically into a three-dimensional diagnostic template, which enables the implant team to identify the specific sites of prospective implant surgery

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Box 3-2 Objectives of Preprosthetic Imaging

- Identify disease
- Determine bone quality
- Determine bone quantity
- Determine implant position
- Determine implant orientation

Box 3-3 Periapical Radiographic Images

Advantages

- Low radiation dose
- Minimal magnification with proper alignment and positioning
- High resolution
- Inexpensive

Limitations

- Distortion and magnification
- Minimal site evaluation
- Difficulty in film placement
- Technique sensitive
- Lack of cross-sectional imaging

Indications

- Evaluation of small edentulous spaces
- Alignment and orientation during surgery
- Recall/maintenance evaluation

in the imaging examinations. The specific objectives of preprosthetic imaging listed in Box 3-2.

All the modalities identified in Box 3-1 have been used in the first diagnostic phase of treatment.[4,5] However, dental implant cases are inherently three-dimensional problems relating to the final prosthetics, occlusion, and function of the patient's three-dimensional anatomy. A three-dimensional treatment plan ideally identifies at each prospective implant site the amount of bone width, the ideal position and orientation of each implant, its optimal length and diameter, the presence and amount of cortical bone on the crest, the degree of mineralization of trabecular bone, and the position or relationship of critical structures to the proposed implant sites. Thus the modalities of choice for presurgical implant treatment planning provide high-resolution and dimensionally accurate three-dimensional information about the patient at the proposed implant sites.

The imaging modalities listed in Box 3-1 can be subdivided into planar two-dimensional, quasi–three-dimensional, and three-dimensional imaging modalities. Planar imaging modalities include periapical, bite-wing, occlusal, and cephalometric imaging and are simply two-dimensional projections of the patient's anatomy. Thus the dentist cannot possibly develop a three-dimensional perspective of the patient's anatomy with a single image. However, with a number of cleverly oriented projections, development of some useful three-dimensional information is possible.

Quasi–three-dimensional imaging modalities include x-ray tomography and some cross-sectional panoramic imaging techniques. These techniques produce a number of closely spaced tomographic images, and the three-dimensional perspective of the patient's anatomy is developed by viewing each image and mentally filling in the gaps. Three-dimensional imaging techniques include CT and MRI and enable the dentist to view a volume of the patient's anatomy. These techniques are quantitatively accurate, and three-dimensional models of the patient's anatomy can be derived from the image data and used to produce stereotactic surgical guides and prosthetic frameworks.

PERIAPICAL RADIOGRAPHY

Periapical radiographs are images of a limited region of the mandibular or maxillary alveolus. Periapical radiographs are produced by placing the film intraorally parallel to the body of the alveolus with the central ray of the x-ray device perpendicular to the alveolus at the region of interest, producing a lateral view of the alveolus.[18] Periapical radiography provides a high-resolution planar image of a limited region of the jaws.[19] Number 2 size dental film provides a 25 × 40-mm view of the jaw with each image. Periapical radiographs provide a lateral view of the jaws and no cross-sectional information. Even with adjacent periapical radiographs made with limited oblique orientations, three-dimensional information is of little use for the implant imaging (Box 3-3).

Periapical radiographs may suffer from distortion and magnification. The most accurate radiographic technique used for periapical radiology is the paralleling technique that necessitates placing the film or sensor parallel to the long axis of the implant, tooth, or osseous structure in question. These principles in positioning will allow for an intraoral image with minimal distortion and magnification. If improper positioning or the bisecting angle technique is used, vertical and horizontal measurements may be distorted and magnified.

The long cone paralleling technique eliminates distortion and limits magnification to less than 10%. Millimeter radiopaque grids, sometimes used in endodontics, may be superimposed over the film before it is exposed, but are of little quantitative value and provide misleading information because they lie on the film and obfuscate the underlying anatomy and do not compensate for magnification.

Image shape distortion occurs when unequal magnification of the object exists. This will occur when the total area in question (alveolar bone, implant) does not

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

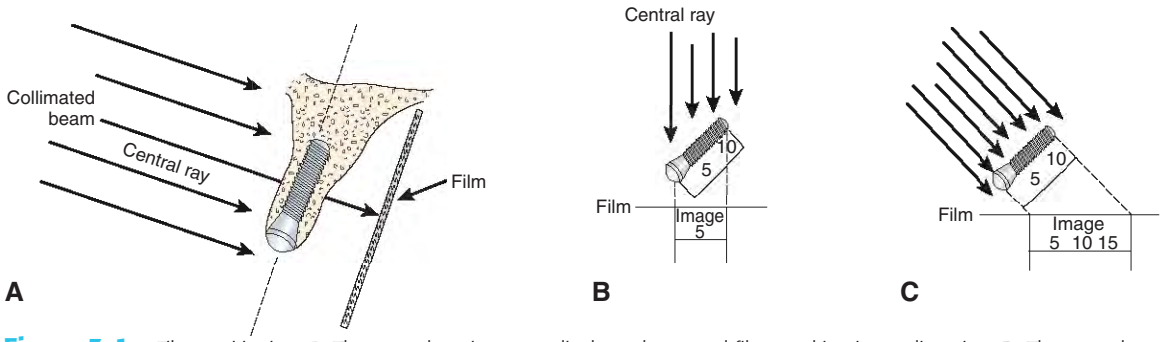


Figure 3-1 Film positioning. **A,** The central ray is perpendicular to bone and film resulting in no distortion. **B,** The central ray is perpendicular to film but not to the implant which will result in foreshortening. **C,** The central ray is perpendicular to the object but not the film and will result in elongation.

have the same focal spot-to-object distance. When the x-ray beam is perpendicular to the object, but the object is not parallel to the film, foreshortening will occur. If the x-ray beam is oriented perpendicular to the object but not the film, elongation will occur (Figure 3-1). These basic and important concepts will help minimize distortion and magnification when using intraoral radiographs.[20]

Image magnification may be assessed by placing a known-dimension radiographic marker (e.g., 5-mm ball bearing) at the crestal region of the desired implant location. When the marker is elongated, so is the implant site. For example, a ball bearing radiographic measurement of 8 mm relates to a 60% magnification. Therefore the image below the ball bearing may represent a 60% magnification of dimension.

The opposing landmark of available bone in implant dentistry is beyond lingual muscle attachments in the mandible or beyond the palatal vault in the maxilla. As such, the image most often must be foreshortened to visualize the opposing cortical plate. As a result, the actual available bone height may be difficult to determine.

The bone density at the crest is also a factor to evaluate crestal bone loss with radiographic indexes. In D4 bone, no cortical plate is present on the crest, and fine trabecular bone is primarily present. Burnout effects are common when standard kilovolt and milliampere settings are used, making crestal bone loss evaluation with digital intraoral systems of benefit in these situations.[21-23]

The dense cortical plates on the lateral aspect of the mandible and palatal aspect of the maxilla make bone quality difficult to assess with a periapical radiograph. In fact, a 40% change in trabecular bone density is necessary before a difference may be observed in the anterior mandible.

In terms of the objectives of presurgical imaging, periapical radiography is:

- A useful high-yield modality for ruling out local bone or dental disease
- Of limited value in determining quantity because the image is magnified, may be distorted, and does not depict the third dimension of bone width
- Of limited value in determining bone density or mineralization (the lateral cortical plates prevent accurate interpretation and cannot differentiate subtle trabecular bone changes)
- Of value in identifying critical structures but of little use in depicting the spatial relationship between the structures and the proposed implant site

In the preprosthetic phase, these films most often are used for single-tooth implants in regions of abundant bone width.

DIGITAL RADIOGRAPHY

One of the most recent significant advances in dental radiology is the advent of digital technology that has allowed numerous limitations of conventional intraoral radiography to be reduced. The advantages of digital radiography and the uses in oral implantology are well documented.[24,25] With the use of digital radiography, implant surgical procedures and prosthetics have been simplified with increased efficiency.

Digital radiology is an imaging process wherein the film is replaced by a sensor that collects the data. The analog information received is then interpreted by specialized software, and an image is formulated on a computer monitor. The resultant image can be modified in various ways, such as gray scale, brightness, contrast, and inversion. Color images may be formed to enhance the digital image for better evaluation. Computerized software programs (i.e., DexisImplant) are now available that allow for calibration of magnified images, thus ensuring accurate measurements (Figure 3-2).

When compared with conventional radiographs, the most current digital systems have significantly less radiation[26,27] with superior resolution.[25] However, with respect to oral implantology, the most significant advan-

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Figure 3-2 Digital radiographic system that includes digital sensor and computer. *(Courtesy Dexis, LLC.)*

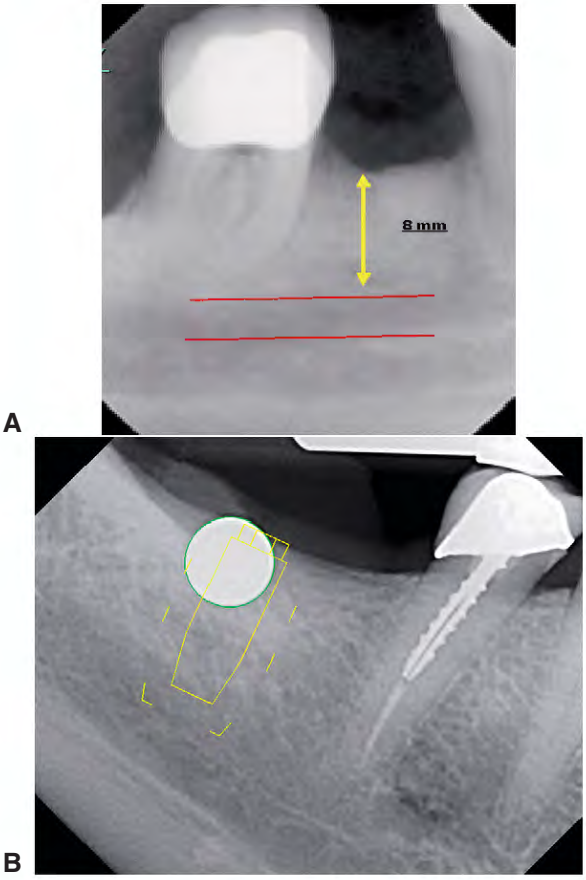


Figure 3-3 **A,** Direct measurements of vital structures may be made directly on the calibrated digital images. Studies have shown that on plain film intraoral radiographs, more than 50% of the time the mandibular canal and mental foramen are indistinguishable. However, this limitation has been drastically reduced with the advent of digital imaging. **B,** Image magnification can be determined by imaging a known-diameter radiographic marker, and the appropriate size implant (adjusted for magnification) may then be selected and placed into the edentulous area.

tage of digital radiography is the instantaneous speed in which images are formed that is highly useful during surgical placement of implants and the prosthetic verification of component placement (Figure 3-3).

Table 3-1 Comparison of Film versus Digital-Based Images

	FILM	DIGITAL
Image	Analog	Analog → Digital
Cost	Film, chemicals	Up-front
Radiation	High	50%-90% less
Viewing	Delayed	Immediate
Resolution	14-18 Ln/mm	12-20 Ln/mm
Grayscale	16 shades	256 shades
Film	Thin, flexible	Thin, cord
Enhancement	Unchangeable	Wide range
Storage	Chart	Computer

From Park ET, Williamson GF: Digital radiography: an overview, *J Contemp Dent Pract* 3:1-13, 2002.

Box 3-4 Occlusal Radiographic Images

Advantages
- Evaluation for pathology

Limitations
- Does not reveal true buccolingual width in mandible
- Difficulty in positioning

Indications
- None

A disadvantage of digital radiography is the size and thickness of the sensor and the position of the connecting cord. These features make the positioning of the sensor more difficult in some sites such as those adjacent to tori or a tapered arch form in the region of the canines (Table 3-1).[28]

OCCLUSAL RADIOGRAPHY

Occlusal radiographs are planar radiographs produced by placing the film intraorally parallel to the occlusal plane with the central x-ray beam perpendicular to the film for the mandibular image and oblique (usually 45 degrees) to the film for the maxillary image. Occlusal radiography produces high-resolution planar images of the body of the mandible or the maxilla.[19] Maxillary occlusal radiographs are inherently oblique and so distorted that they are of no quantitative use for implant dentistry for determining the geometry or the degree of mineralization of the implant site. In addition, critical structures such as the maxillary sinus, nasal cavity, and nasal palatine canal are demonstrated, but the spatial relationship to the implant site generally is lost with this projection (Box 3-4).

Because the mandibular occlusal radiograph is an orthogonal projection, it is a less-distorted projection

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

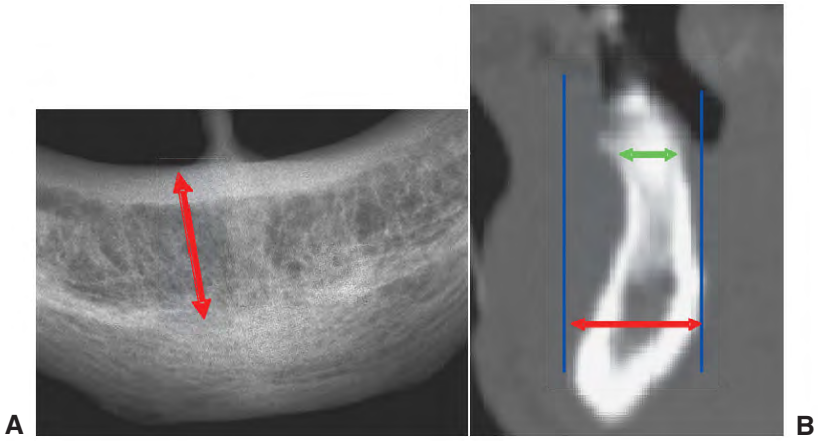


Figure 3-4 **A,** Occlusal radiographs have been postulated to show the width of bone in the anterior region. **B,** However, occlusal radiographs actually show the widest buccolingual distance *(red arrows)* not in the same plane. Actual width of bone *(green arrow).*

than the maxillary occlusal radiograph. However, the mandibular alveolus generally flares anteriorly and demonstrates a lingual inclination posteriorly, producing an oblique and distorted image of the mandibular alveolus, which is of little use in implant dentistry. In addition, the mandibular occlusal radiograph shows the widest width of bone (i.e., the symphysis) versus the width at the crest, which is where diagnostic information is needed most (Figure 3-4). The degree of mineralization of trabecular bone is not determined from this projection, and the spatial relationship between critical structures, such as the mandibular canal and the mental foramen, and the proposed implant site is lost with this projection. As a result, occlusal radiographs rarely are indicated for diagnostic presurgical phases in implant dentistry.

CEPHALOMETRIC RADIOGRAPHY

Cephalometric radiographs are oriented planar radiographs of the skull. The skull is oriented to the x-ray device and the image receptor using a cephalometer, which physically fixes the position of the skull with projections into the external auditory canal. The geometry of cephalometric imaging devices results in a 10% magnification of the image with a 60-inch focal object and a 6-inch object-to-film distance.[19]

A lateral cephalometric radiograph is produced with the patient's midsagittal plane oriented parallel to the image receptor. This radiograph demonstrates a cross-sectional image of the alveolus of the mandible and the maxilla in the midsagittal plane.[21] With a slight rotation of the cephalometer, a cross-sectional image of the mandible or maxilla can be demonstrated in the lateral incisor or in the canine regions. Unlike panoramic or periapical images, the cross-sectional view of the alveolus demonstrates the spatial relationship between occlusion and esthetics with the length, width,

Figure 3-5 A limited projection of the mandibular symphysis region is useful for preoperative evaluation of the symphysis.

angulation, and geometry of the alveolus and is more accurate for bone quantity determinations. Implants often must be positioned in the anterior regions adjacent to the lingual plate.

The lateral cephalometric radiograph is useful because it demonstrates the geometry of the alveolus in the mid-anterior region and the relationship of the lingual plate to the patient's skeletal anatomy (Figures 3-5 and 3-6; Box 3-5). The width of bone in the symphysis region and the relationship between the buccal cortex and the roots of the anterior teeth also may be determined before harvesting this bone for ridge augmentation. Together with regional periapical radiographs, quantitative spatial information is available to demonstrate the geometry of

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

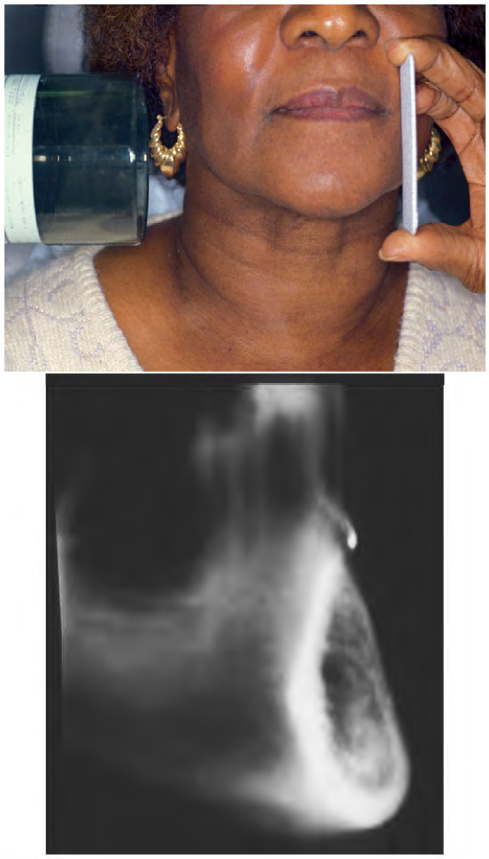


Figure 3-6 **A,** An alternative technique to the standard lateral cephalometric unit is the use of an occlusal film (size 2) held by the patient using a standard intraoral radiographic unit. **B,** Resultant image.

Box 3-5 Lateral Cephalometric Images

Advantages

- Height/width in anterior region
- Low magnification
- Skeletal relationship
- Crown-implant ratio (anterior)
- Tooth position in prosthesis
- Evaluation of the quantity of bone in anterior region prior to symphysis grafting

Limitations

- Availability
- Image information limited to midline
- Reduced resolution and sharpness
- Technique sensitive

Indications

- Used in combination with other radiographic techniques for anterior implants
- Symphysis bone graft evaluation

the implant site and the spatial relationship between the implant site and critical structures such as the floor of the nasal cavity, the anterior recess of the maxillary sinus, and the nasal palatine canal. The lateral cephalometric view also can help evaluate a loss of vertical dimension, skeletal arch interrelationship, anterior crown/implant ratio, soft tissue profile, anterior tooth position in the prosthesis, and resultant moment of forces. As a result, cephalometric radiographs are a useful tool for the development of an implant treatment plan, especially for the completely edentulous patient. However, this technique is not useful for demonstrating bone quality and only demonstrates a cross-sectional image of the alveolus where the central rays of the x-ray device are tangent to the alveolus.

Disadvantages of cephalometric radiographs include cross-sectional information limited to the midline area and difficulty in cephalometric machine accessibility. Any non-midline structure is superimposed on the contralateral side. This radiographic technique is operator technique sensitive and, if improperly positioned, will result in a distorted view. Because lateral cephalometric radiographs use intensifying screens, resolution and sharpness is compromised in comparison to intraoral radiographic techniques.

PANORAMIC RADIOGRAPHY

Panoramic radiography is a curved plane tomographic radiographic technique used to depict the body of the mandible, the maxilla, and the lower half of the maxillary sinuses in a single image. This modality is probably the most used diagnostic modality in implant dentistry. However, for quantitative presurgical implant imaging, panoramic radiography is not the most diagnostic. This radiographic technique produces an image of a section of the jaws of variable thickness and magnification. The image receptor traditionally has been radiograph film but may be a digital storage phosphor plate or a digital charge–coupled device receptor.[23,29,30] Nonetheless, panoramic images offer many advantages (Box 3-6).

The significant limitations of panoramic radiographs can be classified into two categories: (1) distortions inherent in the panoramic system and (2) errors in patient positioning. Panoramic radiography is characterized by a single image of the jaws that demonstrates vertical and horizontal magnification, along with a tomographic section thickness that varies according to the anatomical position. The x-ray source exposes the jaws from a negative angulation and produces a relatively constant vertical magnification of approximately 10%. The horizontal magnification is approximately 20% and variable depending on the anatomical location, the position of the patient and the focus object distance, and the relative location of the rotation center of the x-ray system. Clinical data have shown that nonuniform magnification may be in the range of 15% to 220%.[31-33] Structures of the jaws become magnified more as the object-film distance increases and the object–x-ray source distance decreases.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Box 3-6 Panoramic Radiographic Images

Advantages

- Easy identification of opposing landmarks
- Initial assessment of vertical height of bone
- Convenience, ease, and speed in performance in most dental offices
- Evaluation of gross anatomy of the jaws and any related pathologic findings*

Limitations

- Distortions inherent in the panoramic system
- Errors in patient positioning
- Does not demonstrate bone quality
- Misleading quantitate because of magnification and no third dimension
- No spatial relationship between structures

*Kircos LT: *Preprosthetic imaging in prospective,* Chicago, 1990, University of Chicago Press.

Structures that are located obliquely in relation to the implant receptor produce aspects of the structures that are magnified more when they are farther from the image receptor and less when they are closer to the image receptor.[34-36] Uniform magnification of structures produces images with distortion that cannot be compensated for in treatment planning. The posterior maxillary regions are generally the least distorted regions of a panoramic radiograph. The tomographic section thickness of panoramic radiography or trough of focus is thick, approximately 20 mm, in the posterior regions and thin, 6 mm, in the anterior region.[36]

Traditional panoramic radiography is a high-yield technique for demonstrating dental and bone disease. However, panoramic radiography does not demonstrate bone quality/mineralization, is misleading quantitatively because of magnification and because the third-dimension cross-sectional view is not demonstrated, and is of some use in demonstrating critical structures but of little use in depicting the spatial relationship between the structures and dimensional quantitation of the implant site. Because panoramic radiography is such a popular and widely available technique in dentistry, dentists have developed means to compensate for its shortcomings. Implant companies often market magnified overlays with a preset 25% magnification for evaluation of an implant size that are placed on a panoramic film for comparison with vital structure positions.

Because the concept of standardized magnification is impossible and unreliable, most clinical studies have observed the inaccuracies of direct measurements of panoramic radiographs[37,38] (Figure 3-7).

Inherent magnification is dependent upon patient positioning errors, which results in significant geometric distortion. With knowledge, most errors in patient positioning can be corrected (Table 3-2). However, in a given plane, horizontal distortion cannot be determined

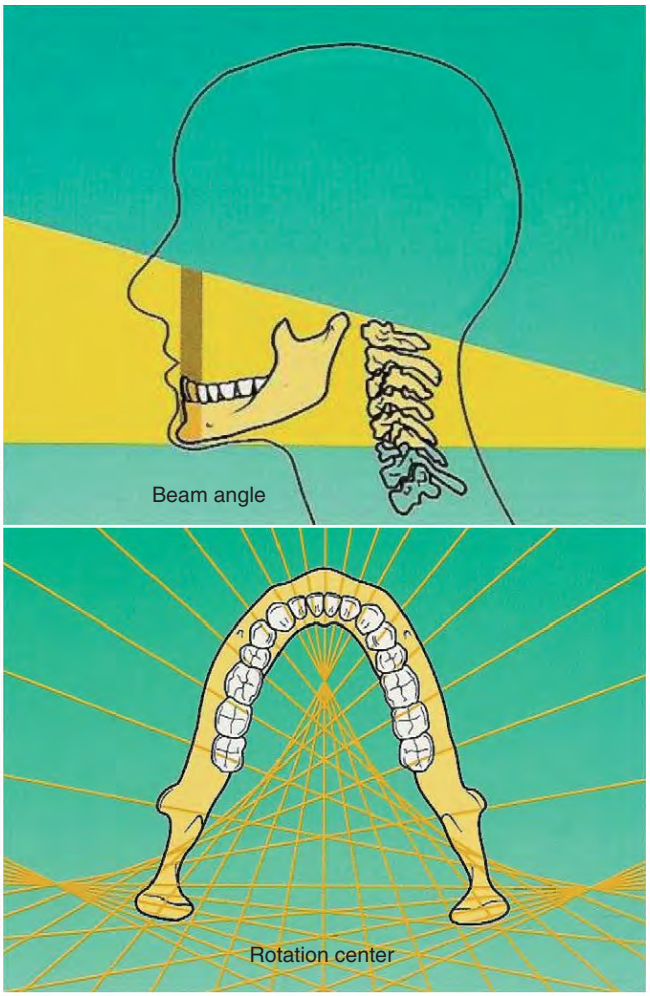


Figure 3-7 **A,** All panoramic beam angles are approximately at 8 degrees, which gives the image inherent magnification. **B,** Because of the curvature of the arch, panoramic machines have changing rotational centers.

and measurements are completely unreliable. The horizontal dimensions are affected by the rotation center of the beam that changes with relation to the object-film distance.[26]

The vertical dimensions are dependent upon the x-ray source as the focus with the amount of distortion determined by the distance of the patient's arch to the film. However, vertical magnification may be determined by imaging a known-diameter object close to the alveolar ridge. The magnification factor can be calculated at the given site by dividing the actual diameter of the object by the diameter measured on the radiographic image. Diagnostic templates that have 5-mm ball bearings or wires incorporated around the curvature of the dental arch and worn by the patient during the panoramic x-ray examination enable the dentist to determine the amounts of magnification in the radiograph (Figure 3-8). A technique for evaluating the panoramic radiograph for mandibular posterior implants and comparison with the clinical evaluation during surgery was developed by identifying the mental

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Table 3-2 Common Panoramic Positioning Problems and Corrections

PROBLEM	CAUSE	CORRECTION
Blurred Magnified	Patient positioned too far posterior	Make sure anterior teeth are properly in holder
Blurred Narrow anterior region	Patient positioned too far anterior	Make sure anterior teeth are in holder
Exaggerated curve of Spee Anterior foreshortening Condyles not seen Spine forms "gazebo" effect	Patient chin tipped down too far	Correctly align ala-tragus
Flattened curve of Spee Hard palate superimposed	Patient chin tipped too far upward	Correctly align ala-tragus
Radiopaque shadow over anterior region	Patient slumped too far forward	Straighten neck
Ramus larger on one side Uneven pattern of blurring	Patient head rotated in machine	Patient midsaggital plane should be perpendicular to floor
Large radiolucency over maxilla	Patient tongue not in floor of mouth	Patient places tongue in roof of mouth, swallows

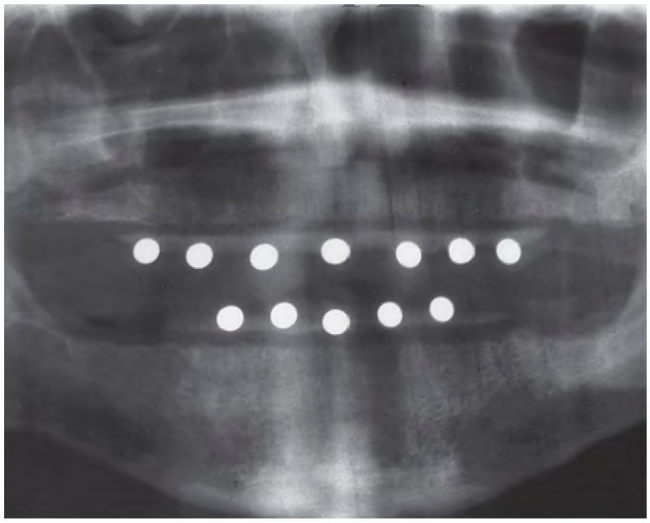


Figure 3-8 Panoramic radiograph with 5-mm ball bearings on the crest of a Division A mandible.

foramen and the posterior extent of the inferior alveolar canal.[39] However, studies have demonstrated that the mandibular foramen cannot be identified 30% of the time on the radiograph film and, when visible, may not be identified correctly.[40-43] The maxillary anterior edentulous region is generally oblique to the film and is often the most difficult area of a panoramic radiograph to evaluate because of the curvature of the alveolus and the inclination of the bone. The dimensions of inclined structures in panoramic radiographs are not reliable. Studies on panoramic x-ray units have demonstrated that objects in front of and behind the focal trough are blurred, magnified, reduced in size, or distorted to the extent of being unrecognizable.

Because the x-ray source comes from below the position of the mandible, the position of the mandibular canal in relation to the crest of the ridge is variable, depending on its buccolingual position in the mandibular body. In other words, when the canal runs lingual within the body, the position displayed on the film is more crestal compared with a nerve that is positioned more buccal, even though they are the same vertical distance from the crest of the ridge. As a result, the lingual-positioned canal may have enough vertical height to place an implant, but the panoramic film indicates inadequate height of bone.

A modification of the panoramic x-ray machine has been developed that has the capability of making a cross-sectional image of the jaws. These devices use limited-angle linear tomography (zonography) and a means for positioning the patient. The tomographic layer is approximately 5 mm. This technique enables the appreciation of spatial relationship between the critical structures and the implant site and quantification of the geometry of the implant site. The tomographic layers are thick and have adjacent structures that are blurred and superimposed on the image, limiting the usefulness of this technique for individual sites, especially in the anterior regions where the geometry of the alveolus changes rapidly. This technique is not useful for determining the differences in most bone densities or identifying disease at the implant site.

TOMOGRAPHY

Tomography is a generic term formed from the Greek words *tomo* (slice) and *graph* (picture) that was adopted in 1962 by the International Commission on Radiological Units and Measurements to describe all forms of body section radiography. Body section radiography is a special x-ray technique that enables visualization of a section of the patient's anatomy by blurring regions of the patient's anatomy above and below the section of interest.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Many ingenious tomographic methods and devices have been developed. However, the basic principle of tomography is that the x-ray tube and film are connected by a rigid bar called the *fulcrum bar*, which pivots on a point called the *fulcrum*. When the system is energized, the x-ray tube moves in one direction with the film plane moving in the opposite direction and the system pivoting about the fulcrum. The fulcrum remains stationary and defines the section of interest, or the tomographic layer. Different tomographic sections are produced by adjusting the position of the fulcrum or the position of the patient relative to the fulcrum in fixed geometry systems.[6]

Factors that affect tomographic quality are the amplitude and direction of tube travel. The greater the amplitude of tube travel, the thinner the tomographic section. Linear tomography is the simplest form of tomography in which the x-ray tube and film move in a straight line. This tomographic motion is one-dimensional and produces blurring of adjacent sections in one dimension, resulting in linear streak artifacts in the resulting image, which may obfuscate the section of interest. Complex-motion, high-quality tomography is described by two-dimensional motion of the tube and film and results in uniform blurring of the regions of the patient's anatomy adjacent to the tomographic motion. Circular, spiral, and hypocycloidal are tube motions used in complex tomography.

The diagnostic quality of the resulting tomographic image is determined by the type of tomographic motion, the section thickness, and the degree of magnification. The type of tomographic motion is probably the most important factor in tomographic quality. Hypocycloidal motion generally is accepted as the most effective blurring motion. Large-amplitude tube travel and 1-mm sections are preferred for high-contrast anatomical objects with geometry that changes in a short distance, such as the alveolus of the jaws. Magnification varies from 10% to 30%, with higher magnification generally producing higher-quality images. Dense structures such as teeth, exostoses, thick cortical plates, and dental materials/restorations are difficult to blur effectively when they are much more dense than the structures depicted in the tomographic section. Dense structures may persist in the tomographic image even though they are three or four times the tomographic layer thickness distant from the tomographic section and will serve to obfuscate the structures of interest in the tomographic section.

For dental implant patients, high-quality complex motion tomography demonstrates the alveolus and, taking magnification into consideration, enables quantification of the geometry of the alveolus.[44,45] This technique also enables determination of the spatial relationship between the critical structures and the implant site. Ideally, tomographic sections spaced every 1 or 2 mm enable evaluation of the implant site region and, with mental integration, enable appreciation of the quasi–three-dimensional appearance of the alveolus. The quantity of alveolar bone available for implant placement can be determined by compensating for magnification. Postimaging digitization of tomographic implant images enables use of a digital ruler to aid in the determination of alveolar bone for implant placement. Image enhancement can aid in identifying critical structures such as the inferior alveolar canal. Complex tomography is not particularly useful in determining bone quality or identifying dental and bone disease (Box 3-7).

Conventional tomograms will have a constant magnification that varies among different machines. Image magnification has been shown to be as much as 40% on some machines.[2] Additionally, this technique is very operator technique–sensitive with superimposition of structures outside of the plane of focus causing significant "blurring" of the image, making them very difficult to read (Figure 3-9). In addition, when looking for the position of a mandibular canal, a vascular space and the cross section of the canal appear similar, and a misinterpretation is possible. Studies have shown that more than 20% of tomographic images are undiagnostic.[46]

COMPUTED TOMOGRAPHY

The discovery and development of CT revolutionized medical imaging. CT is a digital and mathematical imaging technique that creates tomographic sections where the tomographic layer is not contaminated by blurred structures from adjacent anatomy. In addition, and probably most important, CT enables differentiation and quantification of soft and hard tissues. Thus for the first time in medical imaging, the radiologist could view hard and soft tissues on an image without performing

Box 3-7 Conventional Tomography Images

Advantages
- Cross-sectional views
- Constant magnification

Limitations
- Availability
- Cost
- Multiple images needed
- Technique sensitive
- Blurred images
- High radiation dose

Indications
- Single-site evaluation
- Vital structures evaluation

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

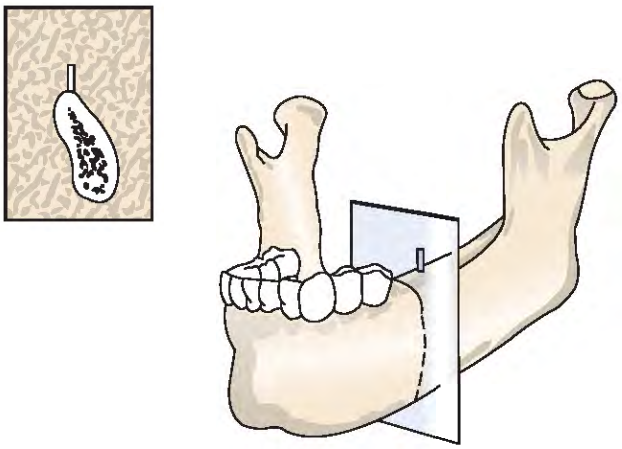


Figure 3-9 Conventional tomograms result in a cross-sectional (slice) image.

an invasive procedure on a patient, such as the injection of contrast media.

CT was invented by Hounsfield and was announced to the imaging world in 1972,[47] but it had its origins in mathematics (1917) and in astrophysics (1956). The first CT scanners appeared in medical imaging departments during the mid-1970s and were so successful that they largely replaced complex tomography by the early 1980s.

CT produces axial images of a patient's anatomy. Axial images are produced perpendicular to the long axis of the body. CT is a prospectively digital imaging technique. The x-ray source is attached rigidly to a fan-beam geometry detector array, which rotates 360 degrees around the patient and collects data. The image detector is gaseous or solid state, producing electronic signals that serve as input data for a dedicated computer. The computer processes the data using back-projection Fourier algorithm techniques first developed by Hounsfield to produce CT images. CT images are inherently three-dimensional digital images, typically 512×512 pixels with a thickness described by the slice spacing of the imaging technique. The individual element of the CT image is called a *voxel*, which has a value, referred to in Hounsfield units, that describes the density of the CT image at that point. Each voxel contains 12 bits of data and ranges from −1000 (air) to +3000 (enamel/dental materials) Hounsfield units. CT scanners are standardized at a Hounsfield value of 0 for water. The CT density scale is quantitative and meaningful in identifying and differentiating structures and tissues.

CT images are inherently three dimensional. Contiguous CT images describe a three-dimensional structure of voxels. The original imaging computer can create secondary images from almost any perspective by reprojecting or reformatting the original three-dimensional voxel data. When a secondary computer is used to perform reformatting or image processing of the original CT data, the system is referred to as a *workstation*.

The power and usefulness of CT for maxillofacial imaging and diagnosis were apparent as soon as high-resolution CT was introduced in the early 1980s. CT was used for imaging the temporomandibular joint, evaluating dental-bone lesions, assessing maxillofacial deformities, and preoperative and postoperative evaluation of the maxillofacial region.[48] CT provides a unique means of postimaging analysis of proposed surgery or implant sites by reformatting the image data to create tangential and cross-sectional tomographic images of the implant site. With current-generation CT scanners, reformatted images are characterized by a section thickness of 1 pixel (0.25 mm) and an in-plane resolution of 1 pixel by the scan spacing (0.5 to 1.5 mm), producing a geometric resolution similar to that of planar imaging. The density of structures within the image is absolute and quantitative and can be used to differentiate tissues in the region and characterize bone quality (Figure 3-10; Boxes 3-8 and 3-9).[10,11,49-55] CT enables the evaluation of proposed implant sites and provides diagnostic information that other imaging or combinations of imaging techniques cannot provide. The utility of CT for dental implant treatment planning[56-62] was evident, but the access to these imaging techniques was limited. Access to this diagnostic information required a radiologist to communicate with the referring doctors in detail about prospective surgery and then to sit at the imaging computer or a workstation for a considerable length of time to reformat the study, interpret the resulting images, and

Box 3-8 Bone Quality

Density	Hounsfield Units
D1	1250
D2	850-1250
D3	350-850
D4	150-350
D5	<150

Box 3-9 Tissue Characterization

Air	−1000
Water	0
Muscle	35-70
Fibrous tissue	60-90
Cartilage	80-130
Trabecular bone	150-900
Cortical bone	900-1800
Dentin	1600-2400
Enamel	2500-3000

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

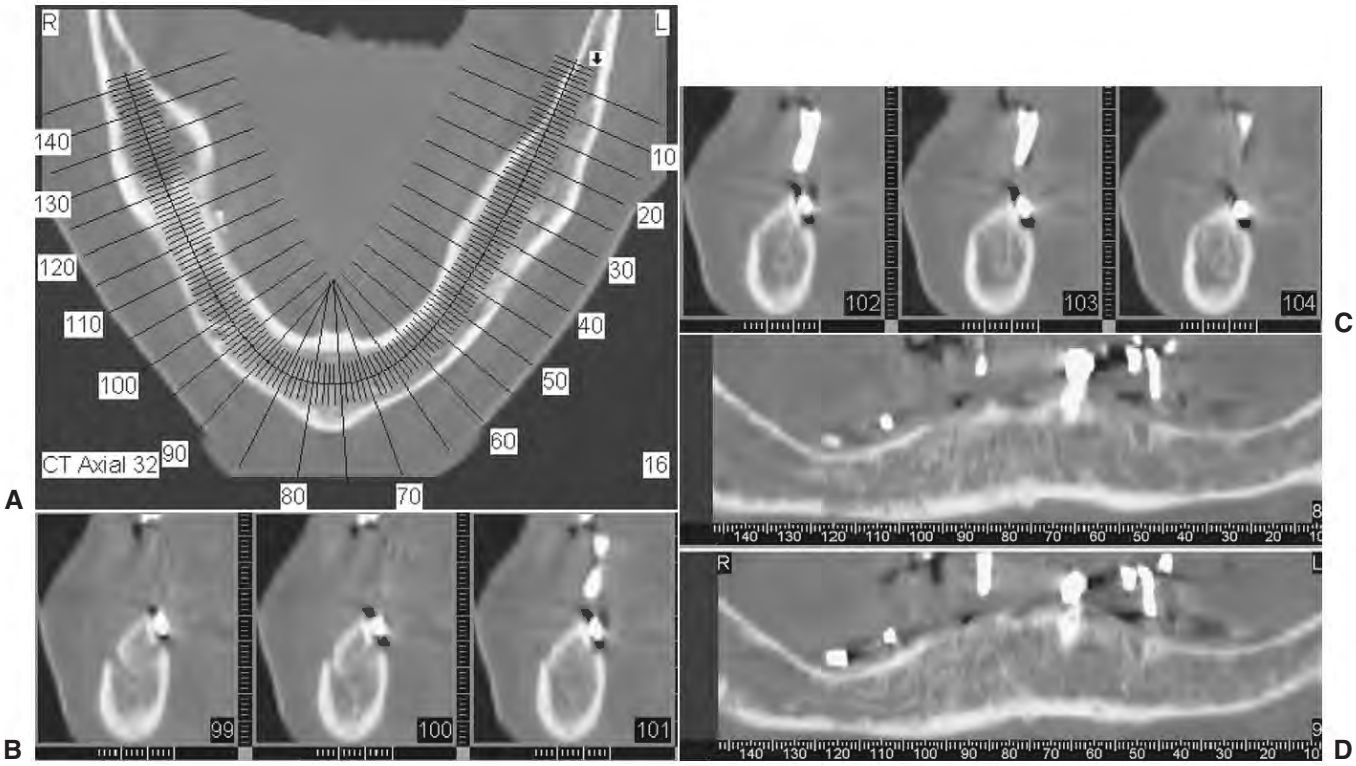


Figure 3-10 Programmed dental computed tomography. The radiologist identifies curvature of the mandible, which programs the computed tomography workstation to create reformatted cross-sectional and curved plane images. **A,** Axial view of mandibular alveolus with curvature of the mandible defined and location of the derived cross-sectional and curved plane images identified. **B, C,** Images 99 to 104 are cross-sectional images that are referenced to the axial and curved plane images. **D,** Images 8 and 9 are curved plane images that are referenced to the axial and cross-sectional images. Gutta-percha radiopaque cylinders identify prospective implant positions.

produce hard-copy images to send to the referring doctor. The advantages of this type of imaging were evident and the limitations of delivery clear, which spawned the development of a number of techniques referred to generically as *DentaScan imaging*.

DentaScan imaging provides programmed reformation, organization, and display of the imaging study[63-65] (see Figure 3-10). The radiologist or technologist simply indicates the curvature of the mandibular or maxillary arch, and the computer is programmed to generate referenced cross-sectional and tangential/panoramic images of the alveolus along with three-dimensional images of the arch. The cross-sectional and panoramic images are spaced 1 mm apart and enable accurate presurgical treatment planning.

Limitations of DentaScan imaging include images that may not be true to size and require compensation for magnification; determination of bone quality that requires use of the imaging computer or workstation; hard-copy DentaScan images that only include a limited range of the diagnostic grayscale of the study; and the tilt of the patient's head during the examination, which is critical because all the cross-sectional images are perpendicular to the axial imaging plane. This technique provides a wealth of diagnostic information that is accurate, detailed, and specific. Usually a diagnostic template is necessary to take full advantage of the technique. The diagnostic template enables the dentist to incorporate the three-dimensional treatment plan of the final prosthetic result into the imaging examination; evaluate the patient's anatomy relative to the proposed implant sites, esthetics, and occlusion; and record and transfer these findings to the patient at the time of surgery. CT enables identification of disease, determination of bone quantity, determination of bone quality, identification of critical structures at the proposed regions, and determination of the position and orientation of the dental implants. Thus CT is capable of determining all five of the radiologic objectives of presurgical implant imaging (Box 3-10; see Box 3-2).

Types of CT Scanners

Medical

In medical radiology departments, the CT scan is the most common diagnostic imaging modality to evaluate hard and soft tissues. Technology in medical CT imaging has advanced significantly since its introduction, and usage is increasing at a rate of 15% to 20% annually.[55] Advances in speed and image quality were apparent in the early 1990s with the advent of spiral/helical

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Box 3-10 Computed Tomography

Advantages
- Negligible magnification
- Relatively high-contrast image
- Various views
- Three-dimensional bone models
- Interactive treatment planning
- Cross-referencing

Limitations
- Cost
- Technique sensitive

Indications
- Interactive treatment planning
- Determination of bone density
- Vital structure location
- Subperiosteal implant fabrication
- Determination of pathology
- Preplanning for bone augmentation

CT scanners. However, since its introduction in 1998, multislice (multirow detector CT) has revolutionized the field of medical CT tomography. These CT scanning units are tomographic machines that are classified as 4-, 8-, 12-, 16-, 32-, and 64-slice machines. The number of slices corresponds to the number of times the x-ray beam rotates around the patient's head to acquire the CT data. The CT numbers, or Hounsfield units, are then reconstructed mathematically and formatted into images. However, because these images consist of a series of incremental images grouped together, CT spiral slices produce "average" reconstructed images based on multiple x-rays transversing the scanning area. With this reconstruction of images, a small gap between each slice is present, which contributes to an inherent error within medical scanners.

In the 1980s, cross-sectional reconstruction of the CT images dramatically improved the diagnosis and treatment planning in oral implantology. These reformatted images allowed three-dimensional evaluation of vital structures and related oral anatomy.[62-65] However, even though these advances enhanced diagnostic skills, there were inherent shortcomings to medical scanners used for dental purposes. Because medical scanners were not developed for dental reformatting, there existed inherent errors such as distortion, magnification, and positioning problems that led to inaccuracies when reformatted.[66] In addition, there existed no prosthetic information that could be gathered to predict the final prosthetic outcome. This was overcome with the advent of sophisticated scanning appliances, stereolithographic resin bone models,[67] interactive software, computer-generated surgical guides, and CT-based image-guided navigation systems, which allowed for ideal placement and prosthetic outcome to be established.

Although the clinical problems of medical scanners have been remedied, there still existed numerous disadvantages: radiation exposure and availability. The amount of radiation exposure of medical scans has been a controversial topic for many years and has been shown to be excessive and unnecessary. It has been postulated that radiation exposure for a scan involving the maxilla and mandible is equivalent to approximately 20 panoramic radiographs.[68] The availability, although dramatically improved over the years, is still a concern for practices in rural areas.

Cone Beam Volumetric Tomography

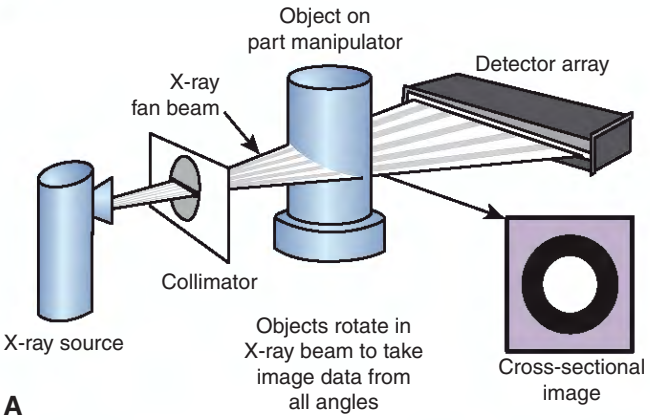
To overcome some of the disadvantages of conventional medical CT scanners, a new type of CT specific for dental applications has recently been developed.[69,70] This type of advanced tomography is termed *cone beam volumetric tomography* (CBVT). Because conventional CT is associated with such a high dose of radiation, this medical imaging technique has always been under significant criticism when used for implant treatment planning. However, with the advent of cone beam technology, the limitations of medical computerized tomography have been overcome.[71] Recently, with the U.S. Food and Drug Administration approval of cone beam technology, there are choices to provide more accurate diagnostic images along with a fraction of the radiation exposure, and adherence to the ALARA principle has been accomplished (Figure 3-11).[72] Scanners are made for "in-office" installation and use, which allows the doctor and patient the convenience of onsite scanning capabilities.

The first CBVT scanner approved for use in dentistry was the NewTom QR-DVT 9000 and most recently has been replaced by the NewTom 3G (Figure 3-12). Other brand names have also become readily available. The x-ray tube on these scanners rotates 360 degrees and will capture images of the maxilla and mandible in 36 seconds, in which only 5.6 seconds is needed for exposure. The positioning of patients is similar to medical scans in which the patient lies on a gurney with the head positioned in an open gantry. A scout film allows for proper positioning and calibrates for radiation dosage. The images recorded are placed onto a charge-coupled device chip with a matrix of 752×582 pixels[73] and are then converted into axial, sagittal, and coronal slices, and permit reformatting to view traditional radiographic images as well as three-dimensional soft tissue or osseous images (Figure 3-13).

Medical versus Cone Beam Technology Radiation Dosages. The average absorbed radiation dose from a CBVT scanner (NewTom 3G) is approximately 12.0 mSv (micro sieverts). This dose is equivalent to five D-speed dental x-rays or 25% of the radiation from a typical panoramic radiograph.[3] Additionally, medical scanners

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت



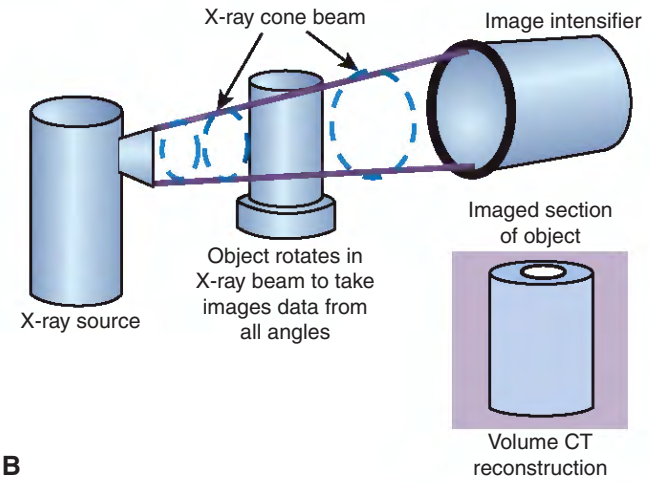


Figure 3-11 **A,** Conventional computed tomography (CT) scan uses a very narrow "fan beam" that rotates around the patient, acquiring one thin slice (image) with each revolution. Because of the numerous revolutions needed, radiation dose is increased. **B,** Cone beam volumetric tomography captures all data in one rotation, thus reducing radiation and avoiding distortion and errors in reformatting.

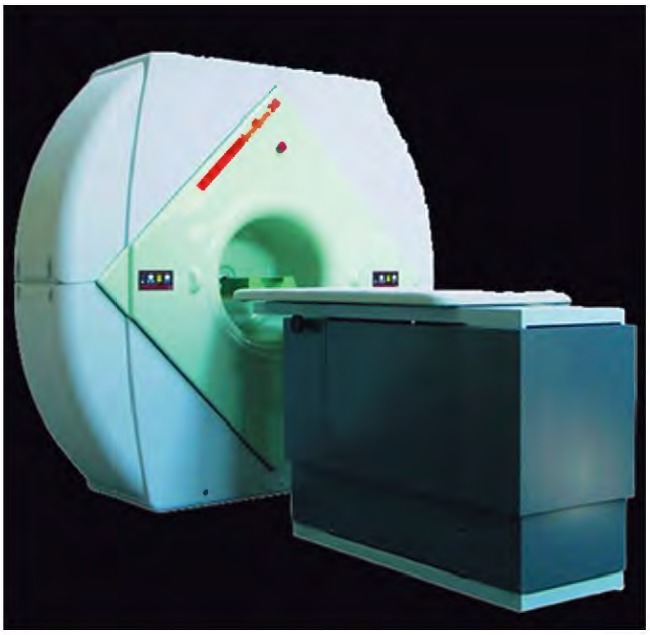


Figure 3-12 The NewTom 3G in-office cone beam computed tomography scanner. The scan is completed in just 36 seconds, with only 5.4 seconds of actual radiation exposure.

Table 3-3 Comparison of Medical Spiral Scanners and Cone Beam

	MEDICAL	CONE BEAM
Scanning time	Approximately 10 min	Approximately 36 sec
Radiation exposure	More	Less
Scan	Multiple slices	One rotation
Exposed field	One arch at a time	Both arches simultaneously
Scatter	More	Less
Positioning	Very technique sensitive	Not as critical

acquire images that use radiation doses of 40 to 60 times that of CBVT doses (Table 3-3).[3]

Image Acquisition of Medical versus Cone Beam Scanners. Medical CT scans produce images of transaxial planes by use of solid-state detectors and an x-ray source that rotates around the patient. When compared with a conventional dental x-ray that is a two-dimensional image of many planes superimposed on each other, the CT image has greater contrast with a reduction in spatial resolution.[74] These CT images are reconstructed mathematically from these incremental images that produce averaged reconstructed images. However, between each parallel slice exists a small "gap" that contributes to a built-in error within medical scanners. These gaps are adjusted within the software's algorithms that can result in errors of 1.0 to 1.5 mm.

Using CBVT avoids the errors in medical scanners by accumulating data from one 360-degree rotation around the patient's head. The algorithms on CBVT scanners are very predictable because they are void of any "gaps," thus eliminating distortion and magnification. Margins of error for CBVT are less than 0.1 mm. Numerous studies have shown cone beam technology to be more accurate than conventional medical CT.[75]

The advantages of CT images are numerous, with the magnification being almost 0% with no superimposition or overlapping of images and minimal distortion. The density of bone may also be assessed with all images being displayed as Hounsfield units. When comparing CT with other types of radiographic modalities, CT has been shown to be superior in identification of vital structures and calculation of distance measurements[76] (see Box 3-10).

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

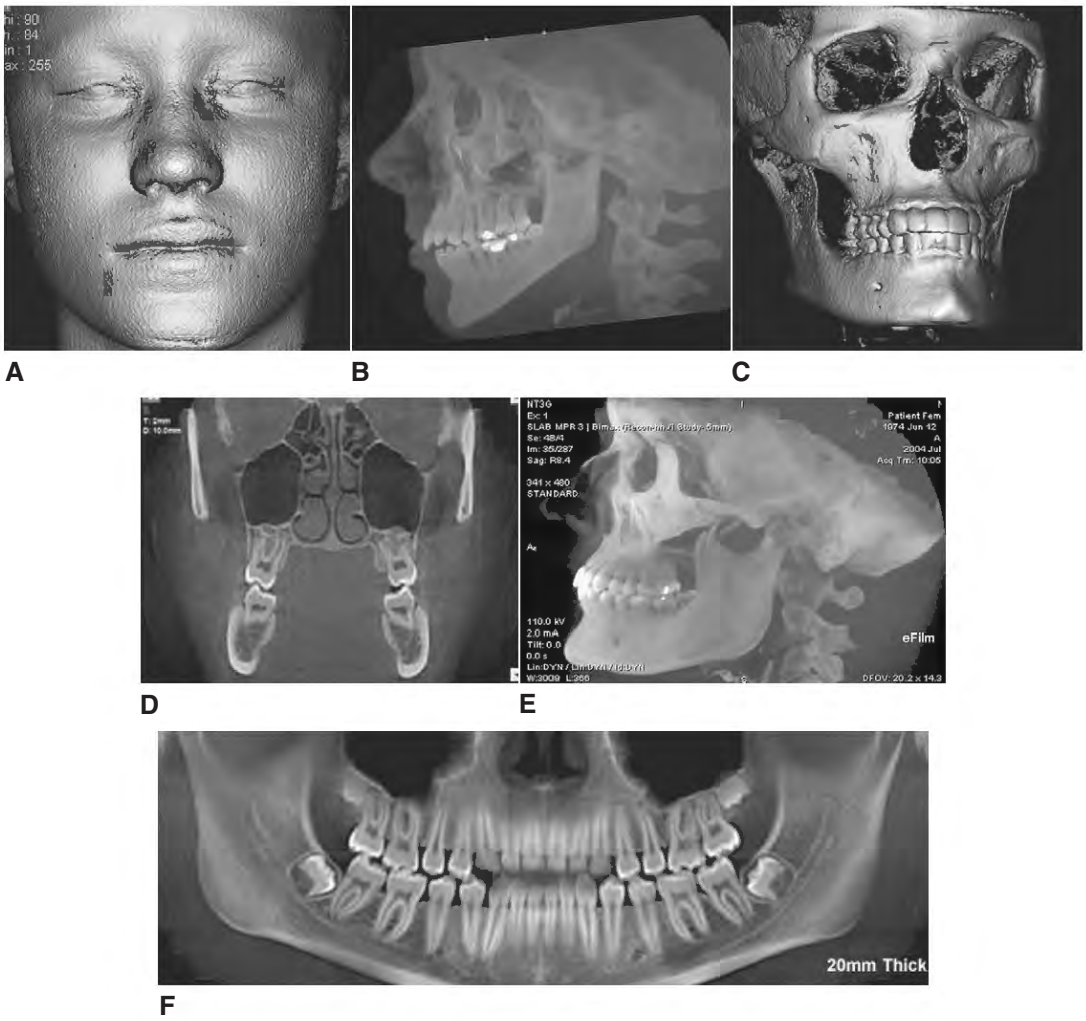


Figure 3-13 Cone beam images (NewTom 9000). **A,** Three-dimensional (3-D) soft tissue. **B,** Lateral cephalometric. **C,** 3-D osseous image. **D,** Coronal. **E,** Lateral osseous image. **F,** Panoramic.

INTERACTIVE COMPUTED TOMOGRAPHY

One of the most significant advances in CT is interactive ICT, which addresses many of the limitations of CT.[51,52,78,79] ICT is a technique that was developed to bridge the gap in information transfer between the radiologist and the practitioner. This technique enables the radiologist to transfer the imaging study to the practitioner as a computer file and enables the practitioner to view and interact with the imaging study on a personal computer. The dentist's computer becomes a diagnostic radiologic workstation with tools to measure the length and the width of the alveolus, measure bone quality, and change the window and level of the grayscale of the study to enhance the perception of critical structures. Axial, cross-sectional, and panoramic images and three-dimensional images are displayed and referenced so that the dentist can appreciate the same position or region within the patient's anatomy in each of the images. Regions of the patient's anatomy can be selected for display normally, with magnification, or with a number of grayscale depictions facilitating the appreciation of anatomy, structures, or disease.

An important feature of ICT is that the dentist and radiologist can perform electronic surgery (ES) by selecting and placing arbitrary-sized cylinders that simulate root form implants in the images. With an appropriately designed diagnostic template, ES can be performed to develop the patient's treatment plan electronically in three dimensions. Electronic implants can be placed at arbitrary positions and orientations with respect to each other, the alveolus, critical structures, and the prospective occlusion and esthetics. Electronic surgery and ICT enable the development of a three-dimensional treatment plan that is integrated with the patient's anatomy and can be visualized before surgery by members of the implant team and the patient for approval or modification. ICT enables the determination of bone quality adjacent to the prospective implant sites (Figures 3-14 to 3-16). With the number and size of implants accurately determined, along with the density of bone at the proposed implant sites, the dentist can

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

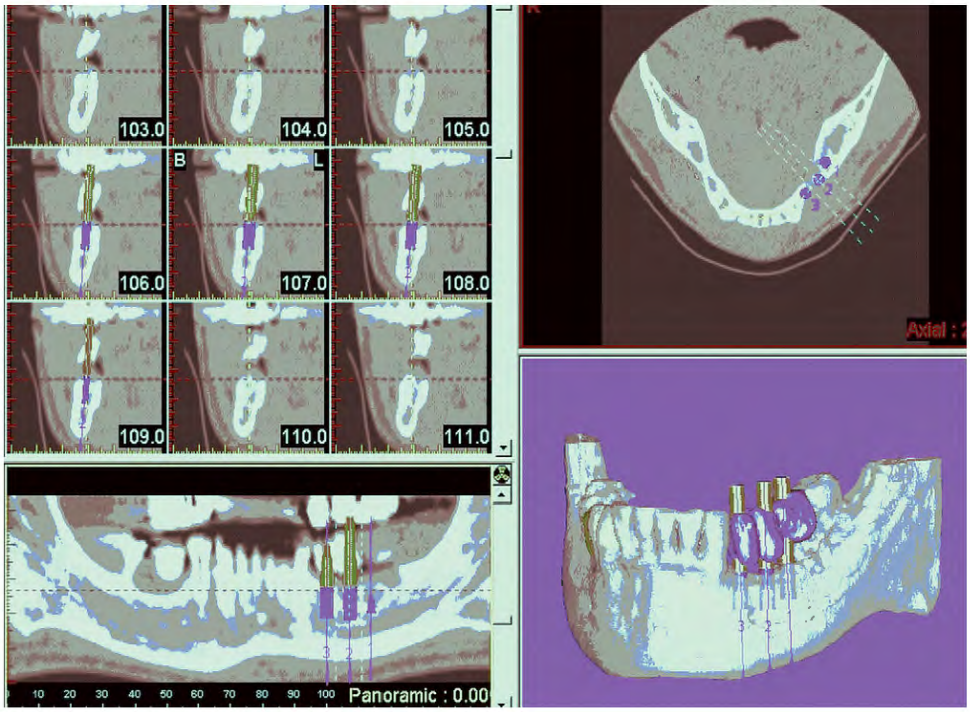


Figure 3-14 Reformatted computed tomography scan showing the interactive placement of implants in relation to the diagnostic wax-up fabricated radiopaque template. Note that each position is cross-referenced according to the tick marks.

determine the characteristics of the implants accurately before surgery.

The first step in the ICT process is the impressions for study casts. With the use of these casts, a diagnostic wax-up is completed according to the ideal position of missing teeth in question with emphasis on the final prosthesis. From the diagnostic wax-up, a radiopaque template is fabricated that the patient will wear during the scan. This diagnostic template will allow the transfer of the ideal positioning of the teeth that will be transferred into the radiographic examination.[79,80]

For conventional and CT, the positioning of the teeth is integrated into a scanning template by way of a radiopaque material. This can be accomplished by way of an acrylic template coated with barium sulfate, gutta percha markers, or radiopaque denture teeth.[81,82] These radiopaque templates may then be modified into use for surgical templates.

ICT is the most accurate imaging technique for implant imaging and surgery, but it some limitations. ES enables placement of electronic implants in the imaging study, but the refinement and exact relative orientation of the implant positions are difficult and cumbersome. For instance, three consecutive implants may require parallelism and interproximal spacing of 2.7 mm. The dentist may struggle in achieving the exact relative spacing and orientation with ES and ICT. Parallelism is difficult to appreciate in ICT using orthogonal rather than three-dimensional images. In this case, implant orientation must be deferred to the orientation developed in the diagnostic template rather than that from the images. After the treatment plan has been developed using ICT and ES and has been approved by the implant team and the patient, executing the plan may be difficult. The precision and accuracy of the treatment plan achieved using ICT and ES for the implant position, size, orientation, relative spacing, spatial relationship to the critical structures and proposed esthetics, and occlusion become a major challenge at the time of surgery. The treatment plan may require implant positions to an accuracy of a few tenths of a millimeter and orientation to a couple of degrees. Transfer of the plan to the patient at the time of surgery can be accomplished by simple visualization and comprehension by a skilled and experienced surgeon, using positions and orientations obtained from ICT and ES to convert the diagnostic template into a surgical template, or the production of a computer-generated, three-dimensional stereotactic surgical template from the digital ICT and ES data.

Surgical Guides

SurgiGuides (Materialise NV, Glen Burnie, Md.) are computer-generated drilling guides that are fabricated through the process of stereolithography. The SurgiGuide concept is based on the presurgical treatment planning using SimPlant software for ideal implant positioning. These successive diameter surgical osteotomy drill guides may be either bone-, teeth-, or mucosa-borne. SurgiGuides

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

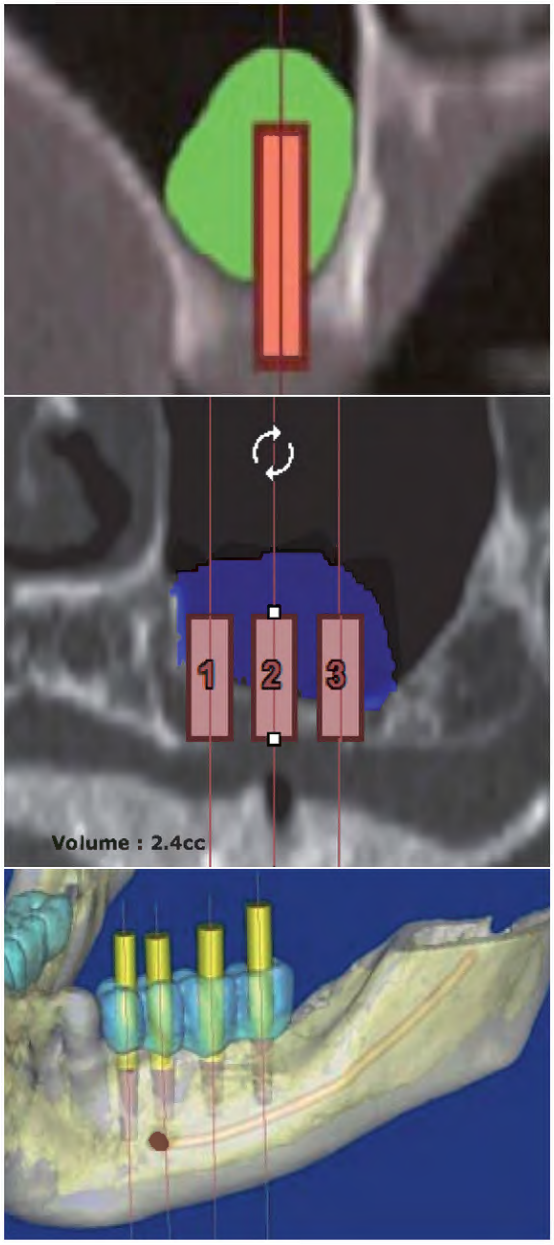


Figure 3-15 **A, B,** In areas where there is insufficient available bone such as the maxillary sinus, the amount of bone grafting needed may be determined. **C,** Implant placement in relation to the mandibular canal and mental foramen; all images are cross-referenced with each other. Three-dimensional analysis for the evaluation of proximity to vital structures may be generated from the same computed tomography images.

have metal cylindrical tubes that correspond to the number of desired osteotomy preparations and specific drill diameters. The diameter of the drilling tube is usually 0.2 mm larger than the corresponding drill, thus making angle deviation highly unlikely.

Clinical data and studies have shown that these computer-aided stereolithographic surgical guides have shown that implant placement is improved and that these guides allow precise translation of a predetermined treatment plan directly to the surgical field (see Chapter 13).[83,84]

CT-Based Surgical Guidance Templates and Navigation Systems

Advanced technology has introduced guidance systems to facilitate dental implant placement procedures during surgery (see Chapter 13). These systems allow the transfer of the presurgical plan to the patient, thus indicating when there is deviation from the predetermined drilling parameters. Therefore the depth and trajectory of the drilling sequence is made to the exact location of the preplanned position.

Numerous clinical studies and case reports have substantiated the use of the CT-based dental imaging with surgical guidance templates. The authors of these studies have shown enhanced accuracy and precision of the surgical procedure that facilitates surgery in anatomical locations.[85,86]

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging is a CT imaging technique that produces images of thin slices of tissue with excellent spatial resolution. This imaging modality developed by Lauterbur in 1972[87] uses a combination of magnetic fields that generate images of tissues in the body without the use of ionizing radiation.[88] MRI allows complete flexibility in the positioning and angulation of image sections and can reproduce multiple slices simultaneously. Digital MRI images are characterized by voxels with an in-plane resolution measured in pixels (512×512) and millimeters and a section thickness measured in millimeters (2 to 3 mm) for high-resolution imaging acquisitions. The image sequences used to obtain magnetic resonance images can be varied to obtain fat, water, or balanced imaging of the patient's anatomy. The images created by MRI are the result of signals generated by hydrogen protons in water or fat such that cortical bone will appear black (radiolucent) or as having no signal. Cancellous bone will generate a signal and will appear white because it contains fatty marrow. Metal restorations will not produce scattering and thus will appear as black images. Therefore MRI has been shown to be less prone to artifacts from dental restorations, prostheses, and dental implants than CT scans.[89] As with CT, MRI is a quantitatively accurate technique with exact tomographic sections and no distortion.[90]

Numerous authors have suggested the use of MRI for dental implant evaluation and treatment planning.[91,92] Additionally, vital structures are easily viewed, such as the inferior alveolar canal and the maxillary sinus. In cases where the inferior alveolar canal cannot be differentiated by conventional or computed tomography, MRI would be a viable alternative as the trabecular bone is easily differentiated with the inferior alveolar canal. In cases of nerve impairment or infection (osteo-

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

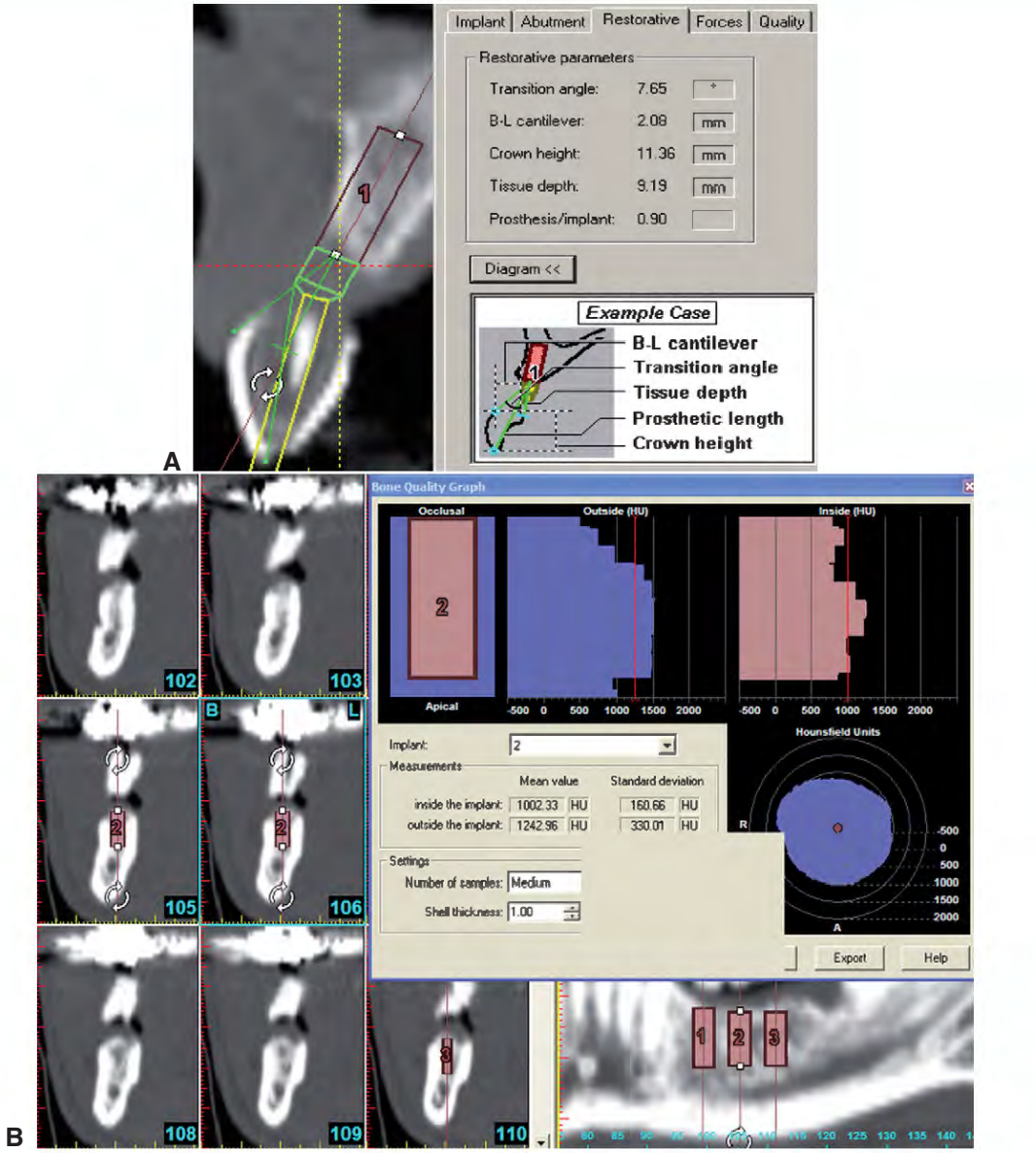


Figure 3-16 **A,** Valuable prosthetic information may be obtained from the positioning of the implants on the interactive computed tomography. **B,** Determination of bone density values are calculated inside and outside of the implant and can be correlated to various densities of bone.

myelitis), MRI may be used because of added advantages including differentiation of soft tissue with respect to CT. Studies have shown that the geometric accuracy of the mandibular nerve with MRI is comparable to CT and is an accurate imaging method for dental implant treatment planning.[93]

MRI may be used in implant imaging as a secondary imaging technique when primary imaging techniques such as complex tomography, CT, or ICT fail (Box 3-11).[92,94] Complex tomography fails to differentiate the inferior alveolar canal in 60% of implant cases, and CT fails to differentiate the inferior alveolar canal in about 2% of implant cases. Failure to differentiate the inferior alveolar canal may be caused by osteoporotic trabecular bone and poorly corticated inferior alveolar canal. MRI visualizes the fat in trabecular bone and differentiates the inferior alveolar canal and neurovascular bundle from the adjacent trabecular bone. Double-scout MRI protocols[90] with volume and oriented cross-sectional imaging of the mandible produce orthogonal quantitative contiguous images of the proposed implant sites. Oriented MRI of the posterior mandible is dimensionally quantitative and enables spatial differentiation between critical structures and the proposed implant site. However, there exist numerous disadvantages for the use of MRI for implant dentistry. MRI is not useful in characterizing bone mineralization or as a high-yield technique for identifying bone or dental disease. No commercially

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Box 3-11 Magnetic Resonance Imaging

Advantages

- No radiation
- Vital structures are easily seen (inferior alveolar canal, maxillary sinus)

Limitations

- Cost
- Technique sensitive
- No reformatting software
- Availability
- Nonsignal for cortical bone

Uses

- Evaluation of vital structures when computed tomography is not conclusive
- Evaluation of infection (osteomyelitis)

available reformatting programs are available to use as reference points. Additional disadvantages are listed (see Box 3-11).

RADIOGRAPHIC IMAGING OF VITAL STRUCTURES IN ORAL IMPLANTOLOGY

Mental Foramen and Mandibular Canal

When evaluating the posterior mandible for implants, the position of the mandibular canal and mental foramen must be identified to avoid trauma to the inferior alveolar nerve. In implant dentistry, two-dimensional radiographs such as periapical and panoramic images are still used routinely as the sole determinate of osseous measurements with respect to these vital structures. However, these imaging views have numerous disadvantages with the lack of buccolingual identification as the most significant. When evaluating distances around these anatomical structures with two-dimensional radiographs, positioning has a significant impact on the intraoral imaging of vital structures.[95] Because of the curvature of the mandible, great care must be given to the angulation of the x-ray beam for intraoral radiography. The x-ray beam must be perpendicular to the tangent of the area in question between the foramen and the most anterior tooth. If the image is taken from a mesio-oblique orientation, measurements will be foreshortened and if the orientation is from a distal-oblique, measurements will appear to be elongated (Figure 3-17).

When evaluating the correct position of the mental foramen using two-dimensional radiographs, care must be taken to evaluate the position of the actual foramen. Studies have shown that on some two-dimensional radiographs, there exists lack of identification because of too large of a radiographic density.[42] This study states that mental foramina are easily seen on light radiographs; however, as the density increases above 2.8, the foramina area becomes less apparent. Other studies show lack of identification because of a lack of cortical bone around the mandibular canal. When evaluating the mental foramen on periapical radiographs, studies have shown that on 50% of periapical radiographs, the mental foramen is not visible.[96] Many studies including dry skull evaluations conclude that the mental foramen is absent in approximately 12% of panoramic radiographs.[42]

In summary, the location of the mental foramen on periapical and panoramic radiographs are inaccurate. It has been shown that even though the mental foramen can be seen on Panorex more consistently than periapical radiographs, the radiograph position of this structure is dependent on the mandibular positioning in the panoramic unit. Additionally, the radiograph landmark depicted on these panoramic radiographs as the mental foramen is not the true foramen, however, represents a portion of the mental canal as it leaves the mandibular canal. In edentulous mandibles, the risk of error increases considerably where there is increased resorption of the alveolar crest.

Numerous studies have shown that the most accurate means of identification is with conventional and computerized tomography (Figure 3-18). The most accurate means of viewing the mandibular canal and mental foramen is with three-dimensional radiography. These images may be altered in contrast, brightness, and grayscale to help depict these structures. Therefore CT has been shown to be the most accurate and is highly recommended when exact location and measurements are needed for the inferior alveolar canal and mental foramen.[14,61,89]

When the inferior alveolar canal or mental foramen is not seen on a radiograph, usually superimposition

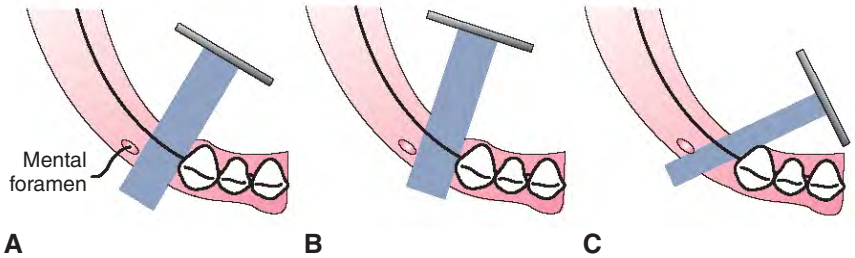


Figure 3-17 To have an accurate measurement between a tooth and a vital structure (mental foramen), a correct horizontal angulation of the x-ray beam is essential. **(A)** Correct, **(B)** improper positioning resulting in too large of a distance, and **(C)** improper positioning resulting in too small of a distance.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

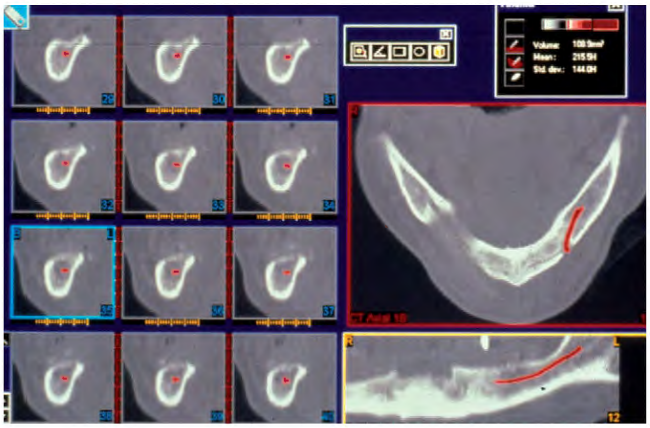


Figure 3-18 The inferior alveolar canal and mental foramen may be drawn, and the position is transferred to various views.

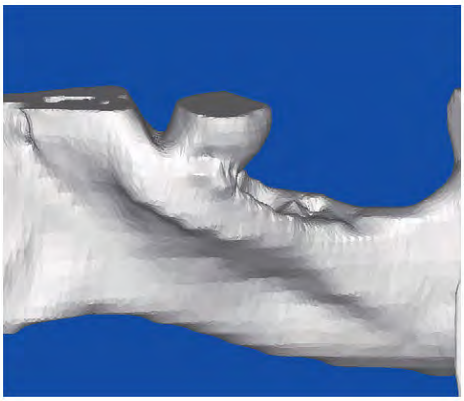


Figure 3-19 Three-dimensional view showing significant sublingual undercut.

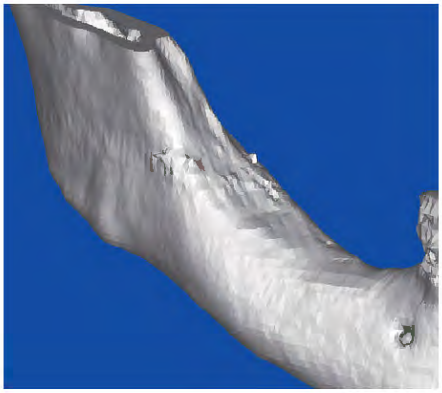


Figure 3-20 The more prominent the external oblique ridge, the better candidate for the ramus as a donor site. This can be best illustrated on a three-dimensional reformatted computed tomography image allowing 360-degree evaluation of the ramus area.

of structures on the contralateral side or lack of cortical bone around the canal is the cause.[97] Studies have shown that in cases where the canal or foramen cannot be seen, tilting the patient's head approximately 5 degrees downward in reference to the Frankfort horizontal plane allows these anatomical structures to be seen in 91% of radiographs.[98]

Mandibular Lingual Concavities

When relying on two-dimensional radiographs for evaluation of the amount of bone for implant placement, significant complications may occur because of overestimation of bone. When advanced atrophy in the posterior mandible is present, lingual concavities may be present (Figure 3-19). Even though there exists adequate bone on two-dimensional radiographs, this may be misleading. A study by Quirynen[99] showed a 2.4% prevalence of concavities with average depths of 6 mm (±2.6 mm). Within these concavities or submandibular gland fossa, branches of the facial artery may be present. Overestimation of the amount of bone may lead to perforation of the lingual plate when drilling the osteotomy. This may result in lingual bleeding problems that may even be life-threatening.[100-103] When assessment of the posterior mandible is needed, cross-sectional tomography is recommended.

Mandibular Ramus (Donor Site for Autogenous Grafting)

The mandibular ramus area has become a very popular donor site for autogenous onlay bone grafting. This area of the mandibular jaw is extremely variable in the amount of bone present. Standard radiographs for preassessment include panoramic images in which the location of the external oblique and the mandibular canal should be noted. However, two-dimensional evaluation of this area can be very difficult to use for adequate assessment of the amount of host bone present. A more accurate representation is the use of tomography, preferably computerized tomography. Accurate measurements may be made along with the use of three-dimensional bone models depicting the exact anatomy present (Figure 3-20).

Mandibular Symphysis

The mandibular symphysis area is a very critical anatomical area for oral implantology. Not only is this region a common position for implants in mandibular edentulous patients, it is also used as a donor site for autogenous grafting. When two-dimensional images are used, inherent errors may occur because of lingual concavities. It is not uncommon to overestimate the height of available bone in the anterior region on panoramic radiographs. For this reason, an imaging technique that will depict the true bucco-lingual amount of bone is recommended. Radiographs including lateral cephalometric and conventional CT, may be used (Figure 3-21).

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

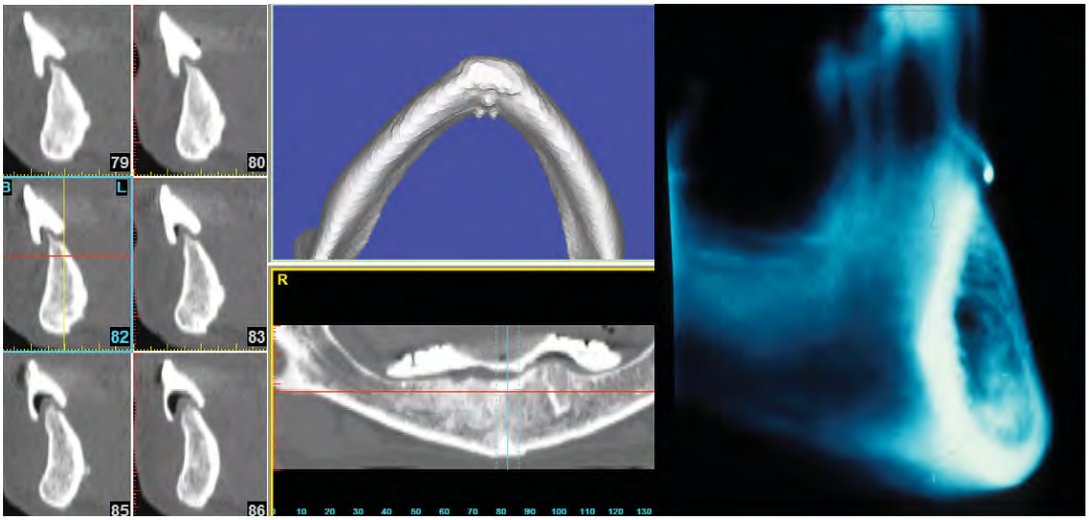


Figure 3-21 Mandibular symphysis. **A,** Computed tomography images illustrate various views of the mandibular anterior region, whereas a lateral cephalometric image **(B)** yields only midline osseous information.

Box 3-12 Recommended Imaging for Implant Treatment Planning

1. Division A: available bone with no approximate vital structures
   Panoramic radiograph
   Supplemental periapical radiographs if needed
2. Division A: available bone with approximate vital structures
3. Division B: available bone
4. Division C: available bone
5. Division D: available bone (allografts, autographs, sinus grafts)
   Panoramic radiograph
   Conventional or computed tomography
   Supplemental periapical radiographs
6. Division A, B, C, D: available bone in which computed tomography does not clearly distinguish exact location of mandibular canal or mental foramen
7. Infection (osteomyelitis)
   Magnetic resonance imaging

Maxillary Sinus

Today, no radiographic modality gives more information of the paranasal sinuses than CT, which is the gold standard for viewing the osseous structures and evaluating pathology in the sinuses. This type of radiography provides much more detailed information regarding prevalence and position of septa,[104] maxillary sinus anatomy, and detection of sinus pathology in comparison with plain film imaging.

RECOMMENDED PRESURGICAL TREATMENT PLANNING

In the past, panoramic radiology was the gold standard for dental implant treatment planning. Along with periapical radiographs, these imaging techniques have many shortcomings that have previously been discussed. The most significant drawback to these radiographs is that they are two-dimensional. For more complex cases or when accurate representation and location of vital structures is needed, CT should be integrated into the pretreatment imaging process.

With CT technology and interactive software, treatment planning has become a very accurate modality for dental implant surgery. Because optimal placement of dental implants can be challenging, special interactive software along with computer-generated templates have been developed to assist the implant surgeon in accurate positioning. From this information and evaluation of all imaging modalities available in implant dentistry, pretreatment imaging evaluation is recommended, as illustrated in Box 3-12.

INTRAOPERATIVE IMAGING

The use of surgical imaging has dramatically changed the way that surgical implantology is completed (Boxes 3-13 and 3-14). In the past, the disadvantage of periapical radiography perioperatively has been time inefficiency. To verify positioning and location of an osteotomy site or for identification of a vital structure, processing of standard radiograph film can take up to 6 minutes. Because of this, practitioners rarely verified positioning

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Box 3-13 Radiographic Sequence for Dental Implants

Pretreatment
Immediate postsurgical (baseline)
Healing period (if needed)
Second-stage surgery
Post prosthetic (baseline)
1 year postoperatively
After first year, every 2 years

Box 3-14 Advantages of Digital Radiography for Implant Surgery

- Fast
- Low radiation
- Calibration
- Magnification
- Excellent quality
- Measures depth, density, and neighboring structures
- Patient stays in surgical setting
- Keeps aseptic setting

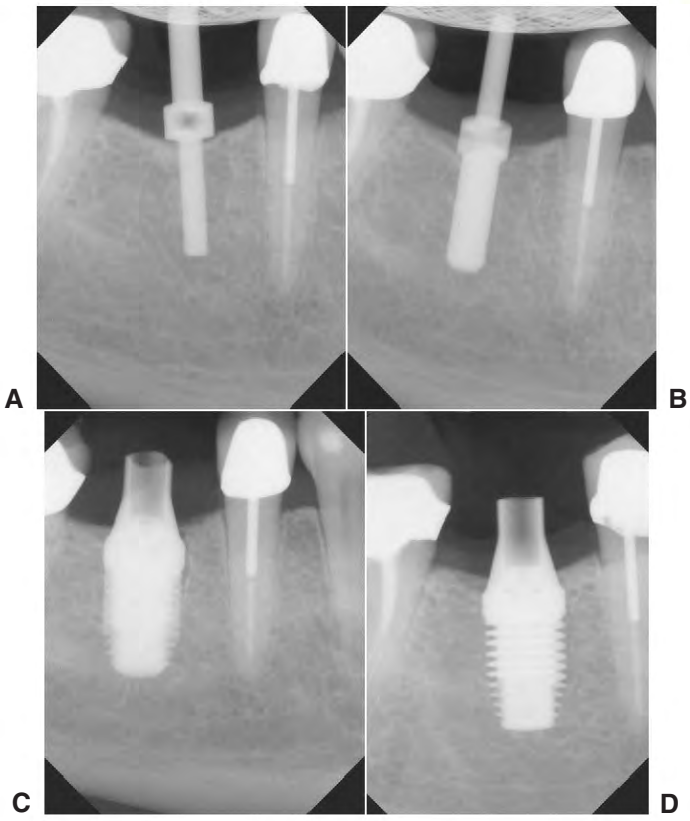


Figure 3-22 **A,** Initial pilot orientation with slight mesial inclination. **B,** Angulation corrected and verified with final depth indicator. **C,** Implant placement. Note poor angulation of radiograph leading to distorted measurements. **D,** Ideal implant placement radiograph. Note perpendicular orientation of x-ray beam as all threads are seen without distortion.

of anatomical structures during surgery. With new digital radiography technology, instantaneous images are achieved, allowing for multiple images to be completed in a fraction of the time. Additional advantages of digital intraoperative imaging include manipulation of images, calibration, accurate measurements and positioning, and maintenance of aseptic protocol (Figures 3-22 and 3-23).

Immediate Postsurgical Imaging

A plain film radiograph (periapical or panoramic) should be taken postsurgically so that a baseline image may be used to evaluate against future films (Figure 3-24). Additional imaging tools may be used to evaluate a zone of safety around vital structures.

Abutment and Prosthetic Component Imaging

When evaluating for transfer impressions along with two-piece abutment component placement, radiographs should be taken to verify secure adaptation. Intraoral radiographs should be used because of their high geometric resolution to evaluate for any fit discrepancy. However, care must be taken so that the x-ray beam is directed at a right angle to the longitudinal axis of the implant. Even a slight angulation may allow a slight gap to be unnoticed. When positioning is difficult for periapical radiographs, bitewing or panoramic radiographs may be used (Figures 3-25 and 3-26).

Postprosthetic Imaging

There exists no conclusive scientific evidence that low-level ionizing radiation has a detrimental effect on bone metabolism and healing. However, radiographs should not be indiscriminately taken. Only when there is a need for additional information or a problem is identified should additional imaging be used. When investigating complications after implant placement, a panoramic radiograph is the most ideal imaging technique for multiple implants. If single implants or if more detailed information concerning an implant viewed on a Panorex is needed, periapical radiographs are the image of choice. A postprosthetic radiograph needs to be taken to act as a baseline for future evaluation of component fit verification and also for marginal bone level evaluation. Care must be taken to ensure that adequate fit of all components is made. In addition, the marginal bone level must be determined for future evaluation.

Recall and Maintenance Imaging

For the evaluation of implant success, immobility and radiographic evidence of bone adjacent to the implant

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

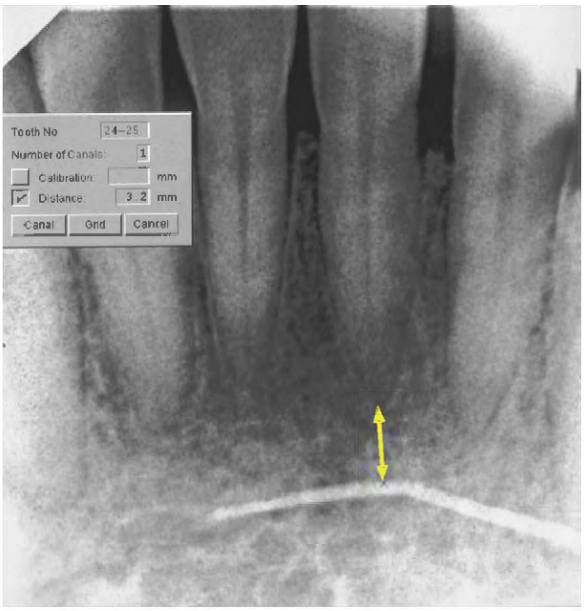


Figure 3-23 During symphysis bone harvesting, the benefits of digital radiography can be used by placing gutta percha in the superior osteotomy site for radiographic evaluation to confirm and measure adequate distance from tooth apices.

Figure 3-24 Inversion of the grayscale to evaluate osseous quality and location of vital structures.

body are the two most accurate diagnostic aids in evaluating success. Follow-up or recall radiographs should be taken after 1 year of functional loading and yearly for the first 3 years.[105] Multiple studies have shown that, in the first year, marginal bone loss and a higher rate of failure are seen.

Evaluation of Alveolar Bone Changes

Radiographically, lack or loss of integration is usually indicated as a radiolucent line around the implant. However, false-negative diagnoses may be made when the soft tissue surrounding an implant is not wide enough to overcome the resolution of the radiographic modality used. Also, false-positive diagnoses may be

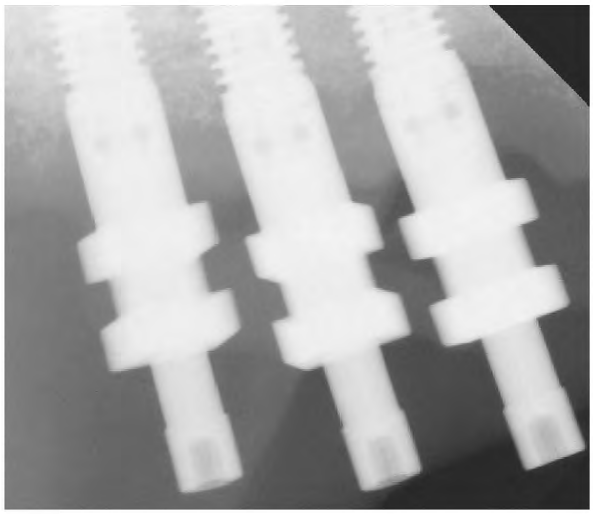


Figure 3-25 Verification of direct transfer coping placement before final impression. Note ideal angulation from thread alignment.

made when a "Mach band effect" results from an area of lower radiographic density adjacent to an area of high density (implant), which results in a more radiolucent area than is actually present.[106] However, studies have shown the possibility of the Mach band effect is significantly reduced with digital image processing. Additionally, digital radiography has been shown to have the advantage over conventional radiography with respect to "edge enhancement," which is the ability to detect space between the implant and the surrounding bone (Figure 3-27).[107] Because of the variability of operator-controlled problems, a strict quality assurance protocol should be used to maintain ideal image quality over time. Proper positioning along with documentation of kVp and mA settings should be documented for future reference.

Periapical Radiographs

In recall radiographic examinations, the marginal bone level is compared with the immediate postprosthetic films. Therefore radiographs similar in geometry, density, and contrast are paramount. To ensure accuracy, standardized periapical radiographs are essential.[108] However, reproducing positioning is very difficult. Numerous film-holding devices have been documented that attach to the implant, abutment, or prosthesis to standardize image geometry.[109-112] When proper projections are achieved, implant threads on both sides of the implant are clearly seen. If the threads are not clearly seen in the radiographs, modification of the beam angle needs to be made. If diffuse threads are present on the right side of the implant, then the beam angle was positioned too much in the superior direction. If the threads are diffuse on the left side, then the beam angle was from an inferior angulation (Figure 3-28). With digital enhanced radiographs, numerous techniques have been postulated to measure bone levels around implants.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

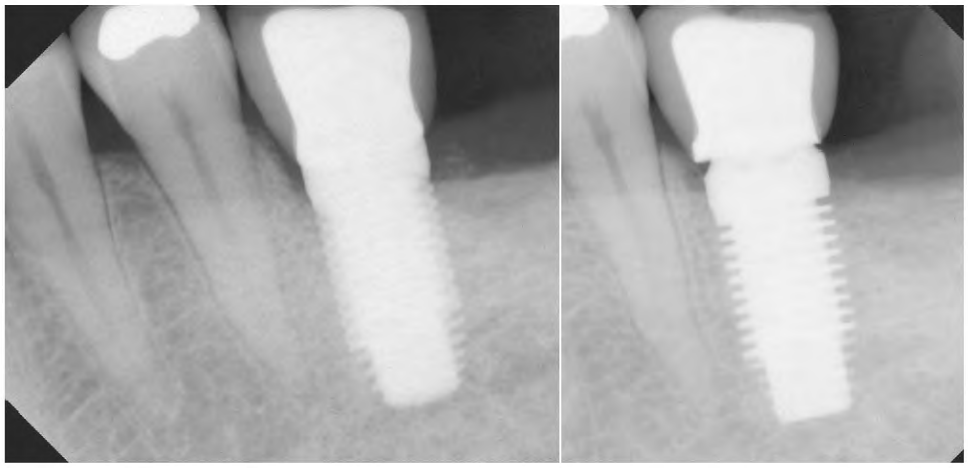


Figure 3-26 Seating of final prosthesis. **A,** Poor x-ray angulation showing a false-negative or complete seating of the prosthesis. Note the diffuse threads. **B,** A corrected angulation image exposes the seating problem.

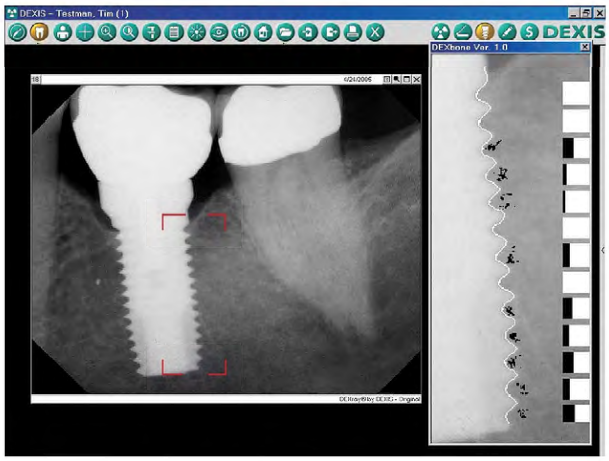


Figure 3-27 Specialized digital image modification programs (e.g., DexBone, Dexis) allow viewing of any subtle changes in bone density around the implant interface.

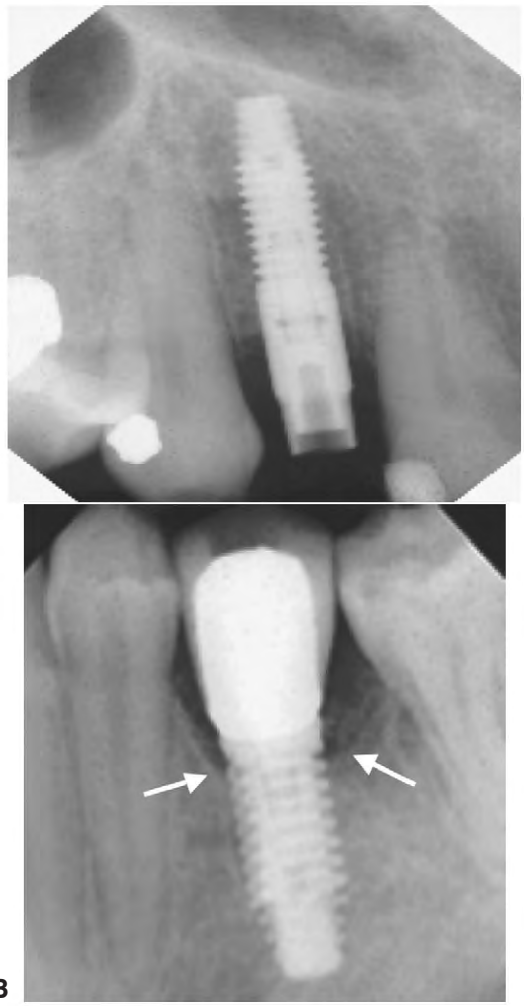


Figure 3-28 Alveolar bone level evaluation. **A,** Ideal positioning showing ideal thread orientation. **B,** Improper angulation showing diffuse thread orientation.

Computer-assisted measurements, rulers, calipers, and suprabony thread evaluation have been shown to have highly reproducible results.[113,114]

Bitewing Radiographs

In cases where the x-ray source cannot be positioned perpendicular to the implant because of oral anatomy or existing prosthesis, horizontal or vertical bitewings may be taken to evaluate the crestal bone area. With this projection, the central beam is perpendicular to the implant and alveolus and the object-film distance is relatively small, and very minor distortions are present. The only limitation of bitewing radiographs is that the apical portion cannot be seen.

Subtraction Radiography

Conventional radiographs are of limited value if minimal alveolar bone changes occur; therefore techniques have been developed to standardize the projection geometry of radiographs for evaluation. This technique, termed *digital subtraction radiography,* allows two radiographs taken at different times to be superimposed on one another, resulting in an image that exhibits the differences in the bone level.[115-117]

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Subtraction radiography requires the use of the same positioning and imaging technique between the two radiographs with respect to the x-ray source, patient and film position, exposure, and processing variables. After the images are subtracted, a subtraction image will be left that depicts the osseous changes between the radiographs.[118] This technique has been shown to be more accurate in accessing bone mineralization and volume changes in comparison to viewing original single radiographs. With the advent of in-office digital radiography systems, specialized software allows for digital radiography to be accomplished rather easily.

Panoramic Radiographs

Panoramic radiographs usually are not used routinely for evaluation of osseous bone levels and recall examinations. Because panoramic radiographs use intensifying screens, resolution is not as good as with intraoral radiographs. However, when film positioning or when multiple implants need to be evaluated, panoramic radiography is the imaging technique of choice.

Computed Tomography

Two-dimensional radiographs (periapical, panoramic) have limitations in that they give no buccolingual information about the present condition of alveolar bone. CT does allow three-dimensional information about the osseous status around an implant. Resolution and scattering has always been a problem in evaluation of implants; however, with the advent of cone beam technology, this is greatly improved.

CT can be of great benefit in the evaluation of sinus augmentation graft prognosis. With the advantage of bone density evaluation using Hounsfield units, important information on bone maturation may be determined. Also, this radiographic modality is the image of choice for evaluation of sinus infection or postsurgical sinusitis complications (Figure 3-29).

FABRICATION OF DIAGNOSTIC TEMPLATES

The purpose of diagnostic radiographic templates is to incorporate the patient's proposed treatment plan into the radiographic examination.[79-82] This practice requires the development of a tentative treatment plan before the imaging procedure. Ideally, mounted diagnostic casts, a diagnostic wax-up, agreement between the practitioners on the number and location of proposed dental implants, and prior authorization of the proposed treatment by the patient make the diagnostic template a useful tool and many times the determining factor in the final treatment plan of the patient. The preprosthetic imaging procedure enables evaluation of the proposed implant site at the ideal position and orientation identified by radiographic markers incorporated into the template.

Computed Tomography

The precision of CT enables use of a complex and precise diagnostic template. Although CT can identify the available bone height and width accurately for a dental implant at a proposed implant site, the exact position and orientation of the implant, which many times determine the actual length and diameter of the implant, often are dictated by the prosthesis. As such, a diagnostic template used during imaging is most beneficial. The surfaces of the proposed restorations and the exact position and orientation of each dental implant should be incorporated into the diagnostic CT template. Designs for diagnostic CT templates have evolved from a simple vacuform reproduction of the wax-up to one produced from a processed acrylic reproduction of the diagnostic wax-up and to more sophisticated types fabricated with specifically designed radiopaque denture teeth.[81,82] The processed acrylic template may be modified by coating the proposed restorations with a thin film of barium sulfate and filling a hole drilled through the occlusal surface of the restoration with gutta-percha. The surfaces of the proposed restoration then become radiopaque in the CT examination, and the position and orientation of the proposed implant may be identified by the radiopaque plug of gutta-percha within the proposed restoration.

The vacuform template has a number of variations. Another design involves coating the proposed restorations with a thin film of barium sulfate. Although the proposed restoration becomes evident in the CT examination, the ideal position and orientation of the proposed implant is not identified by this design. Another design involves filling the proposed restoration sites in the vacuform of the diagnostic wax-up with a blend of 10% barium sulfate and 90% cold-cure acrylic. This results in a radiopaque tooth appearance of the proposed restorations in the CT examination, which matches the density of enamel and dentin of natural teeth but does not identify the exact position and orientation of the proposed implant sites. The next design modifies the previous design by drilling a 2-mm hole through the occlusal surface of the proposed restoration at the ideal position and orientation of the proposed implant site with a twist drill. This procedure results in a natural toothlike appearance to the proposed restoration in the CT examination in which all the surfaces of the restoration are evident along with a 2-mm radiolucent channel through the restoration, which precisely identifies the position and orientation of the proposed implant.[81]

Recently, radiopaque teeth specifically designed for the fabrication of diagnostic templates for fixed and removable implant-supported restorations have been introduced. The radiopaque material (barium sulfate) is an integral component of the CT scan tooth (66% to 67%) (Figure 3-30) (Ivoclar, Orthotac, Schaan, Liechtenstein).

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

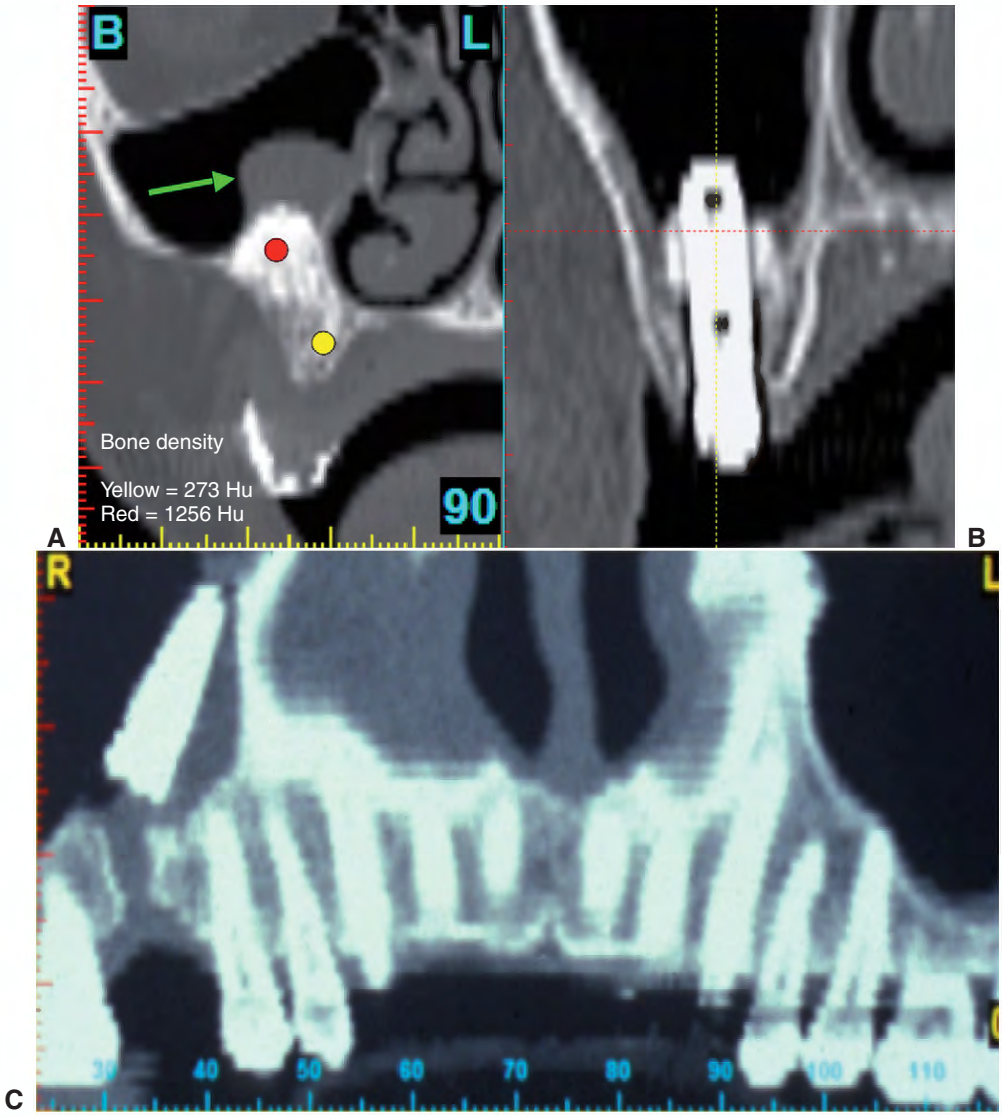


Figure 3-29 **A,** Post-sinus graft computed tomography (CT) scan showing differences in bone density of graft material (red = 1268 Hounsfield units, yellow = 273 Hounsfield units), and illustrating a post-sinus graft cyst blocking the ostium. **B,** CT image of failing implant with a lack of bone at implant interface. **C,** CT image of implant migration in the maxillary sinus.

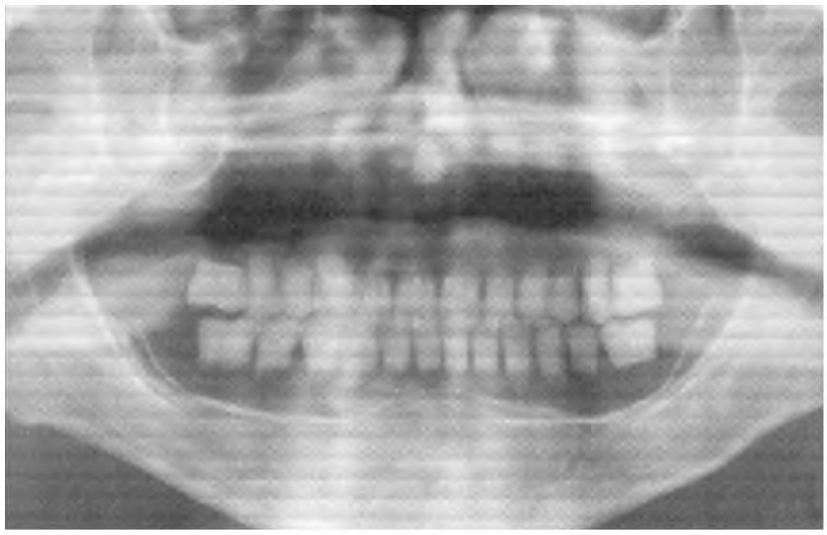
The advantages of prefabricated teeth are that they are time saving, are placed easily, provide consistently high radiopacity, have molds corresponding to prosthetic teeth used in the final restoration, and are bonded easily with the template-based material. The diagnostic template then can be modified into a surgical template.[59]

Tomography

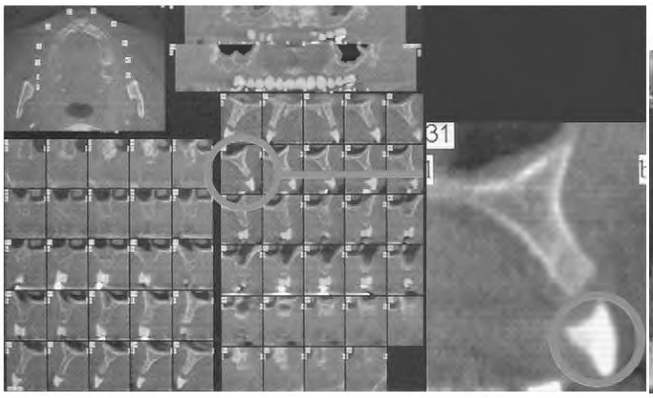
Diagnostic templates for tomography examinations are generally less precise than those required in CT examinations. The diagnostic information available from tomography examinations is not as detailed or as precise as that available from CT examinations. The simplest tomography template is produced by obtaining a vacuform of the patient's diagnostic cast with 3-mm ball bearings placed at the proposed implant positions. A number of tomograms of the implant region are produced with the implant site identified by the one in which the ball bearing is in sharp focus. The ball bearings additionally can serve as a measure of the magnification of the imaging system, although the magnification of most tomographic imaging systems is fixed and known. Templates that incorporate metal cylinders or tubes at the proposed implant sites also enable evaluation of tomograms for the orientation along with the position of the proposed implant. The diagnostic template used in CT examinations, which is produced from a vacuform of the patient's diagnostic cast with barium coating of the proposed restoration and orthodontic wires to indicate the position and orientation of the proposed implant, also can be used for tomography and provides the most diagnostic information of the templates described.

t.me/highdent www.highdentlab.com instagram.com/high_dent

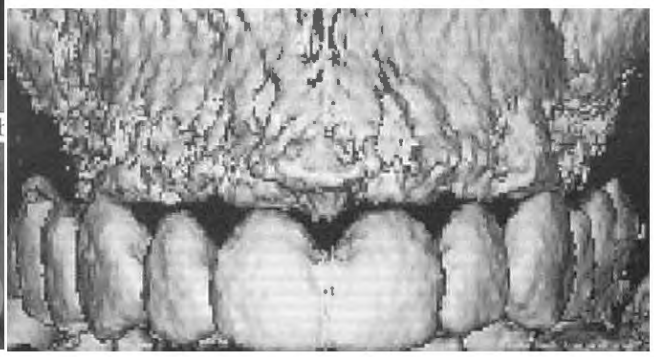
همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت



A



B



C

Figure 3-30 **A,** The patient wears the radiographic template during the computed tomography scanning process. **B,** The contours of the teeth are clearly visible and highly opaque to facilitate image interpretation. **C,** Interactive computed tomography programs provide three-dimensional imaging of the jaws with a full range of movements to view the surgical site and its relationship to the planned restoration, allowing an accurate determination of the hard tissue volume topography. *(Courtesy Ivoclar, OrthoTac, Scaan, Liechtenstein.)*

IMPLANT IMAGING IN PERSPECTIVE

The purpose of implant imaging is to assist the implant team in restoring the patient's occlusion and function by providing accurate and reliable diagnostic information on the patient's anatomy at the proposed implant sites. CT and ICT fulfill more of the objectives of preprosthetic imaging than periapical radiography, cephalometric radiography, panoramic radiography, and tomography. CT and ICT determine the absolute bone quantity, bone quality, and the relationship to critical structures at a level of precision considerably greater than that of tomography, periapical radiology, or panoramic radiology. For instance, bone quantity determination to an accuracy of ±1 mm occurs in about 95% of cases that use CT and only 30% of cases that use panoramic radiology. Furthermore, for mandibular cases, the inferior alveolar canal cannot be identified in more than 30% of cases that use periapical, panoramic, or tomographic techniques, whereas CT can identify the inferior alveolar canal in almost 100% of cases. With the use of ICT and ES, the implant team can determine bone quality directly at the prospective implant sites and develop an electronic three-dimensional treatment plan that can be converted into a stereotactic surgical guide to assist in executing the treatment plan at surgery. For surgical imaging, periapical or digital periapical radiography should be considered the modality of choice. Although digital periapical radiography offers many convenient alternatives to periapical film, neither modality appears to have a significant diagnostic advantage. However, temporal digital SR offers a considerable diagnostic advantage over periapical radiography for determining changes in bone volume or mineralization for post-prosthetic implant imaging. Unfortunately, SR is not conveniently available in general clinical practice, making periapical radiography the modality of choice for post-prosthetic imaging except in acute cases of implant failure, paresthesia, or infection, whereas CT has several advantages.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

References

1. International Commission on Radiological Protection: *Radiation protection*, ICRP publication 26, New York, 1977, Pergamon Press.
2. Richards A, Colquitt W: Reduction in dental x-ray exposure during the past 60 years, *J Am Dent Assoc* 103:713-718, 1981.
3. Clark DE, Danforth RA, Barnes RW et al: Radiation absorbed from dental implant radiography: a comparison of linear tomography, CT scan, and panoramic and intraoral techniques, *J Oral Implantol* 3:156-164, 1990.
4. Engelman MJ, Sorensen JA, Moy P: Optimum placement of osseointegrated implants, *J Prosthet Dent* 59:467-473, 1988.
5. Kircos LT: *Preprosthetic imaging in prospective*, Chicago, 1990, University of Chicago Press.
6. Curry TS, Dowdy JE, Murry RC: *Christensen's physics of diagnostic radiology*, Philadelphia, 1989, Lea & Febiger.
7. Doi K, Rossman K: Measurement of optical and noise properties of screen film systems in radiography, SPIE, medical x-ray photo optical systems evaluation, *Radiology* 56:45, 1975.
8. Rossman K, Lubberts G: Some characteristics of the line spread option and modulation transfer option of medical radiographic films and screen film systems, *Radiology* 86:235, 1966.
9. Sprawls P Jr: Digital imaging systems and image processing. In Daniel C, editor: *Physical principles of medical imaging*, Rockville, Md, 1987, Aspen.
10. Brooks RA: Comparative evaluation of CT scanner technology. In Fullerton GD, Zagzebski JA, editors: *Medical physics monograph no. 6. Medical physics of CT and ultrasound: tissue imaging and characterization*, New York, 1980, American Institute of Physics.
11. Hendee WR: *The physical principles of computed tomography*, Boston, 1983, Little, Brown.
12. Kantor ML, Zeichner SJ, Valachovic RW: Efficacy of dental radiographic practices: options for image receptors, examination selection, and patient selection, *J Am Dent Assoc* 119:259-268, 1989.
13. Sunden S, Grondahl K, Grundahl HG: Accuracy and precision in the radiographic diagnosis of clinical instability in Brånemark dental implants, *Clin Oral Implant Res* 6:220-226, 1995.
14. Zeichner SJ, Ruttiman UE, Webber RL: Dental radiography: efficacy in the assessment of intraosseous lesions of the face and jaws in the asymptomatic patients, *Radiology* 162:691-695, 1987.
15. Beason RC, Brooks SL: Preoperative implant site assessment in southeast Michigan, *J Dent Res* 80:137, 2001 (abstract 810).
16. Sakakura CE, Morais J, Loffredo LCM et al: A survey of radiographic prescription in dental implant assessment, *Dentomax Radiol* 32:397-400, 2003.
17. Tyndall DA., Brooks SL: Selection criteria for dental implant site imaging: a position paper of the American Academy of Oral Maxillofacial Radiology, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 89:630-637, 2000.
18. McCormack FW: A plea for a standardized technique for oral radiology with an illustrated classification of findings and their verified interpretations, *J Dent Res* 2:467-510, 1920.
19. Goaz PW, White SC: *Oral radiology: principles and interpretation*, St Louis, 1992, Mosby.
20. Resnik R: Digital implant plans, *Dental Products Report*, 42, October 2005.
21. Fernandes RJ, Azarbal M, Ismail YH: A cephalometric tomographic technique to visualize the buccolingual and vertical dimensions of the mandible, *J Prosthet Dent* 58:466-470, 1987.
22. Kircos LT, Foust RJ, Carlin M: Demonstration of facial anatomy using curved plane programmed zonography and computed radiography. Proceedings of the Thirty-ninth Annual Meeting of the Association of University Radiologists, Orlando, Fla, March 1991.
23. Grondahl K, Ekestubbe A, Grondahl HG: Technical considerations for intraoral radiography in postoperative examination, *Nobel Biocare Global Forum* 10:10-11, 1996.
24. Parks ET: Digital radiographic imaging: guidelines for implementation, *Pract Proceed Aesthet Dent* 18:173-173, 2006.
25. Van der Stelt PF: Filmless images: the uses of digital radiography in dental practice, *J Am Dent Assoc* 136: 1379-1387, 2005.
26. Wenzel A, Grondahl H-G: Direct digital radiography in the dental office, *Int Dent J* 45:27-34, 1995.
27. Borg E, Grondahl, HG: On the dynamic range of different x-ray photon detectors in intra-oral radiography. A comparison of image quality in film, charge-coupled device and storage phosphor systems, *Dentomaxillofac Radiol* 25:82-88, 1996.
28. Park ET, Williamson GF: Digital radiography: an overview, *J Contemp Dent Pract* 3:1-13, 2002.
29. Brettle DS, Workman A, Ellwood RP et al: The imaging performance of a storage phosphor system for dental radiography, *Br J Radiol* 69:256-261, 1996.
30. Dove SB, McDavid WD: Digital panoramic and extra oral imaging, *Dent Clin North Am* 37:541-551, 1993.
31. SanGiacomo TR: Topics in implantology III: radiographic treatment planning, *R I Dent J* 23:5-11, 1990.
32. Gratt B: Panoramic radiograph/oral radiology: principles and interpretation. In Goaz P, White S, editors: *Oral radiology*, St Louis, 1987, Mosby.
33. Sanfors K, Welander U: Angle distortion in narrow beam rotation radiology, *Acta Radiologica Diagn* 15:570-576, 1974.
34. Welander U, Tronje G, McDavid D: Theory of rotational panoramic radiography. In Langland OE, editor: *Panoramic radiology*, ed 2, Philadelphia, 1989, Lea & Febiger.
35. Langland OE, editor: *Panoramic radiology*, ed 2, Philadelphia, 1989, Lea & Febiger.
36. Lund TM, Manson-Hing LR: A study of the focal troughs of three panoramic dental x-ray machines. I. The area of sharpness, *Oral Surg Oral Med Oral Pathol* 39:318-328, 1975.
37. Bolin A, Eliaasson S: Radiographic evaluation of mandibular implant sites: correlation between panoramic and tomographic determinations, *Clin Oral Impl Res* 7:354-359, 1996.
38. Reddy MS, Mayfield-Donahoo T: A comparison of the diagnostic advantages of panoramic radiology and computed tomography scanning for placement of root form implants, *Clin Oral Impl Res* 5:29-238, 1994.
39. Misch CE, Crawford EA: Predictable mandibular nerve location: a clinical zone of safety, *Int J Oral Implantol* 7:37-40, 1990.
40. Klinge B, Petersson A, Maly P: Location of the mandibular canal: comparison of macroscopic findings, conventional radiography, and computed tomography, *Int J Oral Maxillofac Implants* 4:327-331, 1989.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

41. Sarment D, Sukovic P, Clinthorne N, Accuracy of implant placement with a stereolithographic surgical guide, *Int J Oral Maxillofac Impl* 18:571-577, 2003.
42. Yosue T, Brooks SL: The appearance of mental foramina on panoramic and periapical radiographs, *Oral Surg Oral Med Oral Pathol* 68:488-492, 1989.
43. Sonick M, Abrahams J, Faiella RA: A comparison of the accuracy of periapical, panoramic, and computerized tomographic radiographs in locating the mandibular canal, *Int J Oral Maxillofac Implants* 9:455-460, 1994.
44. Rosenberg HM: Laminography: methods and application in oral diagnosis, *J Am Dent Assoc* 74:88-96, 1967.
45. Eckerdal O, Kvint S: Presurgical planning for osseointegrated implants in the maxilla: a tomographic evaluation of available alveolar bone and morphological relations in the maxilla, *Int J Oral Maxillofac Surg* 15:722-726, 1986.
46. Kassenbaum DK, Nummikoski PV, Triplett RG et al: Cross-sectional radiography for implant site assessment, *Oral Surg Oral Med Oral Pathol* 70:674-678, 1990.
47. Hounsfield GN: Computerized transverse axial scanning (tomography), *Br J Radiol* 46:1016-1022, 1973.
48. Helms C, Morrish R, Kircos LT: Computed tomography of the TMJ: preliminary considerations, *Radiology* 141: 718-724, 1982.
49. Genant HK: Quantitative computed tomography: update, *Calcif Tissue Int* 41:179-186, 1987.
50. Cann CE: Quantitative CT for determination of bone mineral density: a review, *Radiology* 166:509-522, 1988.
51. Kircos LT: Quantitative implant imaging with a focus on interactive computed tomography: successful implant cases. Are they possible? For how long? Lecture, March 13-14, 1994, Loma Linda University.
52. Kircos LT: Implant imaging in perspective with a focus on interactive computed tomography and electronic surgery, Lecture, International Congress of Oral Implantologists, Winter Meeting, March 15-16, 1995.
53. Norton MR, Gamble C: Bone classification: an objective scale of bone density using the computerized tomography scan, *Clin Oral Implants Res* 12:79-84, 2001.
54. Shahlaie M, Gantes B, Schulz E et al: Bone density assessments of dental implant sites: quantitative computed tomography, *Int J Oral Maxillofac Implants* 18:224-231, 2003.
55. Vannier MW: Craniofacial computed tomography scanning: technology, applications, and future trends, *Orthod Craniofacial Res* 6(suppl 1):23-30, 2003.
56. Engstrom H, Svendsen P: Computed tomography of the maxilla in edentulous patients, *Oral Surg Oral Med Oral Pathol* 52:557-560, 1981.
57. McGivney GP, Haughton V, Strandt IE et al: A comparison of computer-assisted tomography and data-gathering modalities in prosthodontics, *Int J Oral Maxillofac Implants* 1:55-58, 1986.
58. Fjellstrom C-A, Strom C: CT of the edentulous maxilla intended for osseo-integrated implants, *J Craniomaxillofac Surg* 15:45-46, 1987.
59. Andersson J-E, Svartz K: CT-scanning in the preoperative planning of osseointegrated implants in the maxilla, *Int J Oral Maxillofac Surg* 17:33-35, 1988.
60. Casselman L, Quirynen M, Lemahieu S et al: Computed tomography in the determination of anatomical landmarks in the perspective of endosseous oral implant installation, *J Head Neck Pathol* 7:255-264, 1988.
61. Stella JP, Tharanon W: A precise radiographic method to determine the location of the inferior alveolar canal in the posterior edentulous mandible: implications for dental implants. 2. Clinical applications, *Int J Oral Maxillofac Implants* 5:23-29, 1990.
62. Andersson L, Kurol M: CT scan prior to installation of osseointegrated implants in the maxilla, *Int J Oral Maxillofac Surg* 16:50-55, 1987.
63. Swartz MS, Rothman SLG, Rhodes ML et al: Computed tomography. 1. Preoperative assessment of the mandible for endosseous implant surgery, *Int J Oral Maxillofac Implants* 2:137-141, 1987.
64. Schwartz M, Rothman S, Chaftez N et al: Computerized tomography. Part II. Preoperative assessment of the mandible for endosseous implant surgery, *Int J Oral Maxillofac Impl* 2:138-148, 1987.
65. Schwartz MS, Rothman SLG, Chafetz N et al: Computed tomography in dental implant surgery, *Dent Clin North Am* 33:555-597, 1989.
66. Kim KD, Jeong HG, Choi SH et al: Effect of mandibular positioning on pre-implant site measurement of the mandible in reformatted CT, *Int J Periodont Rest Dent* 23:177-183, 2003.
67. Sarment D, Sukovic P, Clinthorne N: Accuracy of implant placement with a stereolithographic surgical guide, *Int J Oral Maxillofac Impl* 18:571-577, 2003.
68. Mah JK, Danforth RA, Bumann A: Radiation absorbed in maxillofacial imaging with a new dental computed tomography device, *Oral Surg Oral Med Oral Pathol* 96:508-513, 2003.
69. Mozzo P, Procacci C, Tacconi A: A new volumetric CT machine for dental imaging based on the cone-beam technique: preliminary results, *Eur Radiol* 8:1558-1564, 1998.
70. Aral Y, Tammiasalo E, Iwai K: Development of a compact computed tomographic apparatus for dental use, *Dentomaxillofac Radiol* 28A:245-248, 1999.
71. Harris D, Buser D, Dula K: E.A.O. guidelines for the use of diagnostic imaging in implant dentistry, *Clin Oral Impl Res* 13:566-570, 2002.
72. Winter A, Pollack A: Cone beam volumetric tomography vs. medical CT scanners: expanding dental applications, *N Y State Dent J* 71:28-33, 2005.
73. Ziegler CM, Woertche R, Hasfeld S: Clinical indications for digital volume tomography in oral and maxillofacial surgery, *Dentomax Radiol* 31:126-130, 2002.
74. Michael G: X-ray computed tomography, *Physics Educ* 36:442-251, 2001.
75. Hashimoto K, Kawashima W: Comparison of image performance between cone-beam CT for dental use and four row multidetector helical CT, *J Oral Sci* 48:27-34, 2006.
76. Tal H, Moses O: A comparison of panoramic radiography with computed tomography in the planning of implant surgery, *Dentomaxillofac Radiol* 20:40-42, 1991.
77. Lee CY: Three dimensional CT in the evaluation of placement for dental implants, *Hawaii Dent J* 27:26-29, 1996.
78. Gher ME, Richardson AC: The accuracy of dental radiographic techniques used for evaluation of implant fixture placement, *Int J Periodontics Restorative Dent* 15:269-285, 1996.
79. Israelson H, Plemons JM, Watkins P et al: Barium-coated surgical stent and computer-assisted tomography in the preoperative assessment of dental implant patients, *Int J Periodontics Restorative Dent* 12:52-61, 1992.
80. Monson ML: Diagnostic and surgical guides for placement of dental implants, *J Maxillofac Surg* 52:642-645, 1994.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

81. Fondriest JF, McClenahan DC: Fabricating radiographic stents in implant treatment planning, *Chicago Dent Soc Rev* 90:40-43, 1997.
82. Borrow JW, Smith JP: Stent marker materials for computerized tomography assisted implant planning, *Int J Periodontics Restorative Dent* 16:60-67, 1996.
83. Giovanni AP, Di Giacomo P, Cury P et al: Clinical application of stereolithographic surgical guides for implant placement: preliminary results, *J Periodontol* 76:503-507, 2005.
84. Sarment DP, Al-Shammari, K, Kazor CE: Stereolithographic surgical templates for placement of dental implants in complex cases, *Int J Periodontics Restorative Dent* 23: 287-295, 2003.
85. Siesseger M, Schneider BT, Mischkowski RA: Use of an image-guided navigation system in dental implant surgery in anatomically complex operation sites, *J Craniomaxillofac Surg* 29:276-281, 2001.
86. Fortin T, Champleboux G, Bianchi S: Precision of transfer of preoperative planning for oral implants based on cone-beam CT scan images through a robotic drilling machine, *Clin Oral Implants Res* 13:651-656, 2002.
87. Lauterbur PC: Image formation by induced local interactions: example employing nuclear magnetic resonance, *Nature* 242:190, 1973.
88. Stark DD, Bradley WG: *Magnetic resonance imaging,* St Louis, 1988, Mosby.
89. Gray CF, Redpath TW, Smith FW et al: Advanced imaging: magnetic resonance imaging in implant dentistry, *Clin Oral Implants Res* 14:18-27, 2003.
90. Kircos LT: Magnetic resonance imaging of the temporomandibular joint. In del Baso A, editor: *Advances in maxillofacial imaging,* Philadelphia, 1990, WB Saunders.
91. Gray CF, Redpath TW, Smith FW et al: Advanced imaging: magnetic resonance imaging in implant dentistry, *Clin Oral Implants Res* 14:18-27, 2003.
92. Zabalegui J, Gil JA, Zabalegui B: Magnetic resonance imaging as an adjunctive diagnostic aid in patient selection for endosseous implants: preliminary study, *Int J Oral Maxillofac Implants* 5:283-288, 1991.
93. Eggers G, Rieker M, Fiebach J et al: Geometric accuracy of magnetic resonance imaging of the mandibular nerve, *Dentomaxillofac Radiol* 34:285-291, 2005.
94. Kircos LT: Magnetic resonance imaging of the mandible utilizing a double scout technique for preprosthetic imaging, *J Magn Reson Med* 7:190-194, 1993.
95. Wical KF, Swoope CC: Studies of residual ridge resorption: part 1: use of panoramic radiographs for evaluation and classification of mandibular resorption, *J Prosthet Dent* 32:7-12, 1974.
96. Fishel D, Buchner A, Hershkowith A et al: Roentgenologic study of the mental foramen, *Oral Surg Oral Med Oral Pathol* 41:682-686, 1976.
97. Rosenquist B: Is there an anterior loop of the inferior alveolar nerve? *Int J Periodont Rest Dent* 16:41-45, 1996.
98. Dharmar S: Locating the mandibular canal in panoramic radiographs, *Int J Oral Maxillofac Implants* 12:113-117, 1997.
99. Quirynen M, Mraiwa N, van Steenberghe D et al: Morphology and dimensions of the mandibular jaw bone in the interforaminal region in patients requiring implants in the distal areas, *Clin Oral Implants Res* 14:280-285, 2003.
100. Darriba MA, Mendonca-Caridad JJ: Profuse bleeding and life-threatening airway obstruction after placement of mandibular dental implants, *Int J Oral Maxillofac Surg* 55:1328-1330, 1997.
101. Laboda G: Life-threatening hemorrhage after placement of an endosseous implant: report of case, *J Am Dent Assoc* 121:599-600, 1990.
102. Niamtu J: Near-fatal airway obstruction after routine implant placement, *Oral Surg Oral Med Oral Path Oral Radiol Endo* 92:597-600, 2001.
103. Ten Bruggenkate CM, Krekeler G, Kraajenhagen HA et al: Hemorrhage of the floor of the mouth resulting from lingual perforation during implant placement: a clinical report, *Int J Oral Maxillofac Implants* 8:329-334, 1993.
104. Kasabah S, Slezak R, Simunek A et al: Evaluation of the accuracy of panoramic radiograph in the definition of maxillary sinus septa, *Acta Medica (Hradec Kralove)* 45:173-175, 2002.
105. Grondahl K, Lekholm U: The predictive value of radiographic diagnosis of implant instability, *Int J Oral Maxillofac Implants* 12:59-64, 1997.
106. Sunden S, Grondahl K: Accuracy and precision in the radiographic diagnosis of clinical instability in Brånemark dental implants, *Clin Oral Implant Res* 6: 220-226, 1995.
107. Borg E, Grondahl K., Persson LG: Marginal bone level around implants assessed in digital and film radiographs: in vivo study in the dog, *Clin Implant Dent Related Res* 2:10-17, 2000.
108. De Smet E, Jacobs R, Gijbels F et al: The accuracy and reliability of radiographic methods for the assessment of marginal bone level around oral implants, *Dentomaxillofac Radiol* 31:176-181, 2002.
109. Cox JF, Pharoah M: An alternate holder for radiographic evaluation of tissue integrated prostheses, *J Prosthet Dent* 56:338-341, 1986.
110. Duckworth JE, Judy PF, Goodson JM et al: A method for geometric and densitometric standardization of intraoral radiographs, *J Periodontol* 54:435-440, 1983.
111. Meijer HJA, Steen WHA, Bosman F: Aiming device for standardized intraoral radiographs of the alveolar crest around implants in the lower jaw, *J Prosthet Dent* 68: 318-321, 1992.
112. Jeffcoat MK, Reddy MS, Webber RL et al: Extraoral control of geometry for digital subtraction radiography, *J Periodontol Res* 22:396-402, 1987.
113. Wouters FR, Jon-And C, Frithiof L et al: A computerized system to measure interproximal alveolar bone levels in epidemiologic, radiographic investigations. I. Methodologic study, *Acta Odontol Scand* 46:25-31, 1988.
114. Reddy MS, Mayfield-Donahoo TL, Jeffcoat MK: A seminal automated computer assisted method for measuring bone loss adjacent to dental implants, *Clin Oral Implants Res* 3:28-31, 1992.
115. Jeffcoat MK, Reddy MS: Digital subtraction radiography for the longitudinal assessment of periimplant bone change: method and validation, *Adv Dent Res* 7:196-201, 1993.
116. Bragger U, Burgin W, Long WP et al: Digital subtraction radiography for the assessment of changes in peri-implant bone density, *Int J Maxillofac Implants* 6:160-166, 1991.
117. Nicopoulou-Karayianni K, Bragger U, Lang NP: Subtraction radiography in oral implantology, *Int J Periodont Rest Dent* 17:221-230, 1997.
118. Jeffcoat MK, Reddy MS, Van Den Berg HR et al: Quantitative digital subtraction radiography for the assessment of periimplant bone change, *Clin Oral Implants Res* 3:22-27, 1992.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

Chapter 4

Stress Treatment Theorem for Implant Dentistry

Carl E. Misch

Dentistry is a unique aspect of medicine, blending science and art form. Some aspects of the dental field emphasize the art form, as in dental esthetics, which deals with tooth color and shape to enhance a patient's smile and overall appearance. However, the primary reason the term *doctor* is applied to the dental profession is because of the dental sciences. These may be separated into a biological component and a biomechanical component. For general dentists, the biological aspects of oral health are emphasized. Common complications related to the natural dentition are primarily of biological origins, with periodontal diseases, caries, and endodontic problems as examples.[1-4]

A combination of biological and biomechanical factors is responsible for the failure of tooth-supported fixed prostheses. The four most common complications for three-unit fixed prostheses are (1) caries, (2) endodontic problems, (3) unretained prosthesis, and (4) porcelain fracture.[5,6] The biological complications occur with greater frequency (11% to 22%), compared with the biomechanical (7% to 10%), but both aspects should be understood by the clinician.

Implant dentistry always involves the replacement of teeth. When implant complications are reported, the vast majority of problems are related to the implant sciences, rather than esthetics.[7] But, unlike natural teeth, the biological aspects of implant dentistry have relatively few complications. For example, the development of a direct bone-implant interface is largely biological. Most recent reports indicate the surgical phase of implants form a successful interface more than 95% of the time, regardless of the implant system used.[7] Hence, the biological aspect of the field is very predictable.

The most common implant-related complications are biomechanical problems that occur after the implant is loaded. A literature review focusing on implant failure indicated these problems primarily occur within 18 months of initial implant loading. These early implant loading failures occur most often in the softest bone types (16% failure) or the shortest implant lengths (17% failure). These two failure groups are typically caused by biomechanical factors. Soft bone is too weak for the occlusal forces applied to the implants, or short implants have higher stresses at the bone-implant interface[8-20] (Figure 4-1).

The most common complications that do not lead to the failure of the implant are also biomechanical problems. Implant overdentures have problems of attachment fracture or complication (30%) and removable prosthesis fracture (12%). In implant-supported fixed prostheses, acrylic resin veneer fracture (22%), abutment or prosthetic screw loosening (7% to 10%), porcelain fracture (7%), and prosthesis metal fracture (3%) are examples of common complications.[7,15] In addition, implant components (2% to 4%) and even implant bodies may fracture (1% to 2%) (Box 4-1). In other words, mechanical complications far outnumber biological implant problems.[7,20]

Any complex engineering structure will fail at its "weakest link," and dental implant structures are no exception. A general concept in engineering is to determine the causes of complications and develop a system to reduce the conditions that cause the problems. The most common causes for implant-related complications

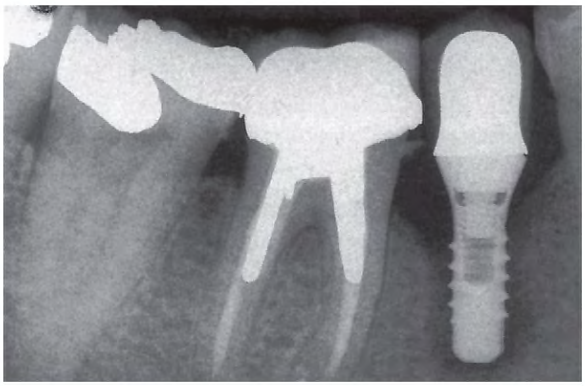


Figure 4-1 The majority of implant failures occur within 18 months after prosthetic loading and are related to short implants or implants placed in poor-quality bone.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

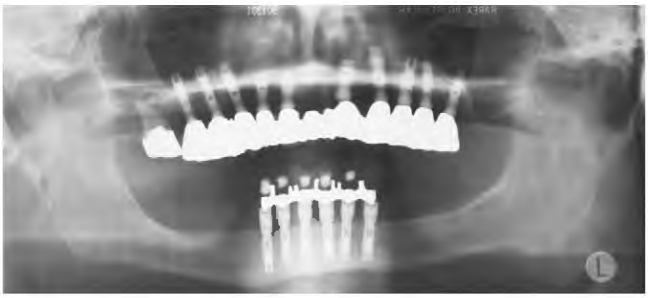


Figure 4-2 Excessive heat during osteotomy preparation can cause surgical failure of a healing implant.

Box 4-1 Stress Treatment Theorem: Biological versus Biomechanical

Biological	Biomechanical
Implant Failure	
Surgical failure	Early loading failure
Healing	Micromovement
Crestal Bone Loss	
Periosteal reflection	Cellular biomechanics
Osteotomy	Engineering
Autoimmune (bacteria)	Bone mechanics
Biological microgap	Animal studies
	Clinical reports
Prosthetic Complications: Mechanics	
Screw loosening	Attachment wear
Component fracture	Attachment fracture
Implant body fracture	Denture tooth fracture
Acrylic veneer/porcelain fracture	Acrylic base fracture
Framework fracture	Opposing prosthesis fracture

are centered around stress. Thus, the overall treatment plan should (1) assess the greatest force factors in the system and (2) establish mechanisms to protect the overall implant-bone-prosthetic system.

SURGICAL FAILURE

There are many reasons for the failure of an implant to integrate initially with the bone. The primary causes of failure relate to excessive heat during the preparation of the osteotomy or excessive pressure at the implant-bone interface at the time of implant insertion[21] (Figure 4-2). The excessive pressure at implant insertion is observed most often with tapered screw-type body designs. The insertion torque force on a tapered screw implant design may place excessive forces on the bone, which leads to resorption and implant failure.

An additional cause of surgical failure is micromovement of the implant while the developing interface is established (Figure 4-3). A fractured arm is immobilized to prevent movement at the fracture site to decrease

Figure 4-3 Micromovement of a developing bone-implant interface may cause fibrous tissue to form around an implant rather than a bone-implant interface. Excessive stresses to an implant may cause overload and failure. This implant had occlusal overload, which resulted in fibrous tissue formation around the implant *(From Isidor F: Loss of osseointegration caused by occlusal load of oral implants: a clinical and radiographic study in monkeys,* Clin Oral Implants Res *7:143-152, 1996.)*

the risk of a fibrous nonunion. Movement as little as 20 microns has been reported to cause a fibrous interface to form at the fracture site. Brunski observed a fibrous tissue interface development when a dental implant moved more than 100 microns during initial healing.[22] The original Brånemark protocol used a two-stage surgical approach.[23,24] One of the main reasons for this concept was to place the implant at or below the crestal bone region to decrease the risk of implant movement during initial bone healing. Schroeder also suggested an unloaded healing period on implants, although the implant was placed at or slightly above the gingival tissues.[25]

Occlusal forces applied to a removable prosthesis over a healing implant may also cause incision line opening of the soft tissue and delay soft tissue healing.[26] These occlusal forces may also affect the marginal bone around the developing implant site. Transferring these forces to an overlying soft tissue-borne prosthesis may cause micromovement of the implant-bone interface, whether the implant is healing below or above the gingival tissues. Stresses applied to a healing implant increase the risk of complications. On the other hand, multicenter clinical reports indicate an experienced surgeon may obtain rigid fixations after surgical placement 99% of the time.[27] The surgical component of implant failure is often the least risk associated with the overall implant treatment.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

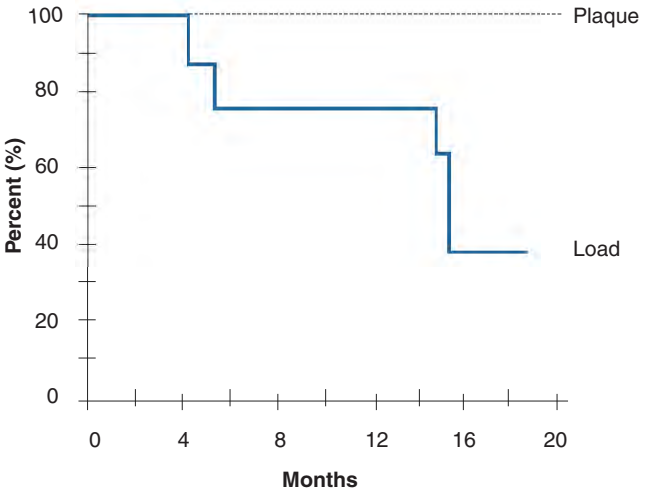


Figure 4-4 Premature occlusal contacts caused six of eight integrated implants to fail within 18 months. Strings in the sulcus and excessive plaque accumulation caused no failure during this period. *(From Isidor F: Loss of osseointegration caused by occlusal load of oral implants: a clinical and radiographic study in monkeys,* Clin Oral Implants Res *7:143-152, 1996.)*

Figure 4-5 The risk of screw loosening is greatest for single-implant crowns. To tighten the abutment screw, the implant crown may need to be cut off to gain access.

EARLY LOADING FAILURE

On occasion, an implant may fail shortly after it has initially "integrated" to the bone. Before failure, the implant appears to have rigid fixation, and all clinical indicators are within normal limits. However, once the implant is loaded, the implant becomes mobile within 6 to 18 months (see Figure 4-1). This has been called *early loading failure* by Misch and Jividen.[9] The cause of early loading failure is usually excessive stress for the bone-implant interface. Isidor allowed eight implants to integrate in monkey jaws.[28] Crowns were attached to the healed implants with excessive premature occlusal contacts. Over a 20-month period, six of eight implants failed (Figure 4-4). In these same animals, eight integrated implants with no occlusal loads had strings placed in the marginal gingiva to increase the amount of plaque retention. None of these implants failed over the following 20 months. The authors concluded that in this animal model, biomechanical occlusal stress was a greater risk factor for early implant failure than the biological component of bacterial plaque.[28,29]

Early loading failure is worse for the implant clinician than when a surgical failure occurs, because the patient may blame the restoring dentist. Although this is bad enough, in addition the restoring dentist spent two to five appointments restoring the implant and has a laboratory expense. Early loading failure is related to the amount of force applied to the prosthesis[8,22,30-33] and the density of the bone around the implants,[7,10-14,34] and it may affect 15% of implant restorations.[6-11] Early implant failure from biomechanical overload, as high as 40%, has been reported in the softest bone types.[13] No reports in the literature correlate such high incidence extreme with early implant failure rates related to the biological width-related complications observed in the field.

IMPACT OF OCCLUSAL OVERLOAD ON MECHANICAL COMPONENTS

Screw Loosening

Abutment screw loosening has been detected in an overall average of 6% of implant prostheses.[7] Single-tooth crowns exhibited the highest rate of 25% in early screw designs and concepts (and as high as 45%). Recent studies indicate this ratio has been reduced to an overall 8% average, with multiple-unit fixed prostheses at a 5% average and implant overdentures at 3% (Figure 4-5). The greater the stress applied to the prostheses (single tooth versus overdentures), the greater the risk of abutment screw loosening. Cantilevers also increase the risk of screw loosening, as they increase the forces in direct relationship to the length of the cantilever.[35] The greater the crown height attached to the abutment, the greater the force applied to the screw, and the greater the risk of screw loosening.

The height or depth of an antirotational component of the implant body also can affect the amount of the force applied to the abutment screw. The higher (or deeper) the hex height, the less stress applied to the screw and a corresponding lower risk for abutment screw loosening.[35,36] The platform dimension upon which the abutment is seated is even more important than the hex height dimension. Larger-diameter implants, with larger platform dimensions, reduce the forces applied to an abutment screw and change the arc of displacement of the abutment on the crest module. Screw loosening also may be decreased by a preload force with a torque wrench on the screw. Therefore, prosthetic methods to decrease stress to the abutment

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

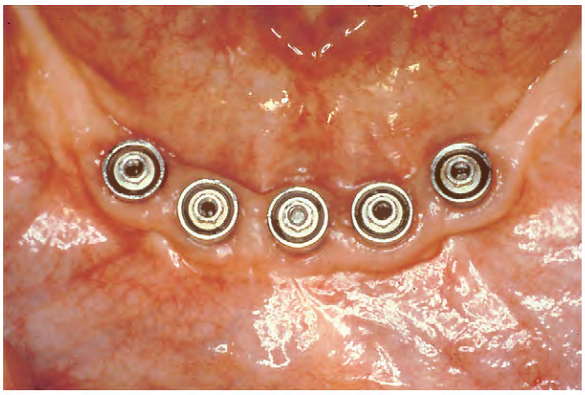


Figure 4-6 Prosthesis screw fracture has been observed for screw-retained fixed prostheses.

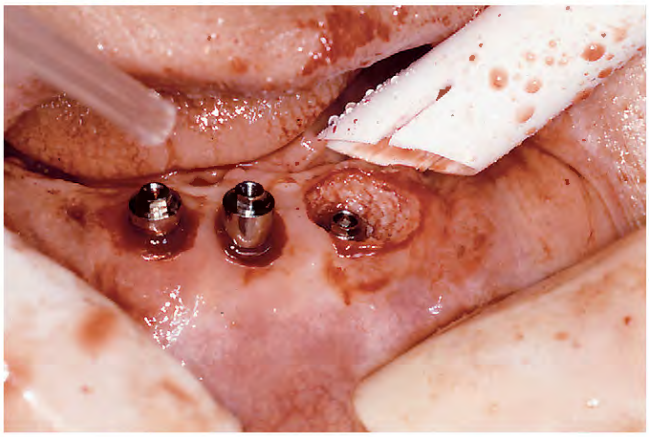


Figure 4-7 Abutment screw fracture occurs less often than prosthetic screw fracture, as they are larger in diameter.

screw or engineering approaches to decrease stress or increase thread tightening may be used to decrease the incidence of complications related to screw loosening.

Fatigue Fractures

Materials follow a fatigue curve, which is related to the number of cycles and the intensity of the force. There is a force so great that one cycle causes a fracture (e.g., karate blow to a piece of wood). However, if a lower force magnitude repeatedly hits an object, it will still fracture. The wire coat hanger that is bent does not break the first time, but repeated bends will fracture the material—not because the last bend was more forceful, but because of fatigue. Indeed, when the patient says he soaked his bread in coffee and then placed it in his mouth prior to the porcelain/abutment screw/cement seal/cantilevered prostheses fracture, it may have been "the straw that broke the camel's back."

Prosthesis screw fracture has been noted in both fixed partial and complete fixed prostheses, with a mean incidence of 4% and a range of 0% to 19%[7] (Figure 4-6). Abutment screws are usually larger in diameter and therefore fracture less often, with a mean incidence of 2% and a range of 0.2% to 8%

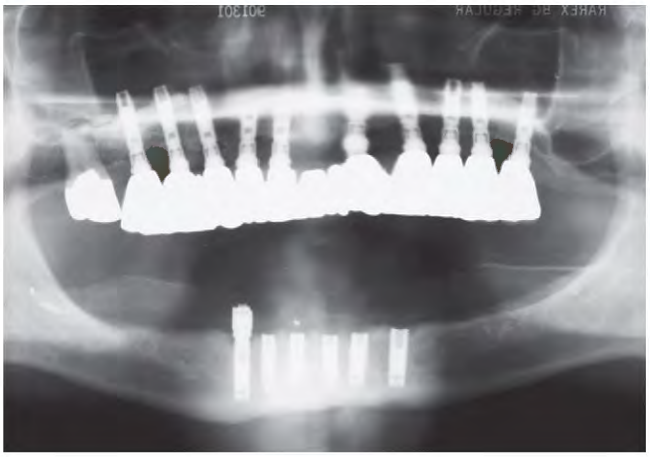


Figure 4-8 Metal framework fracture has been reported with an average of 3% with implant-fixed prostheses.

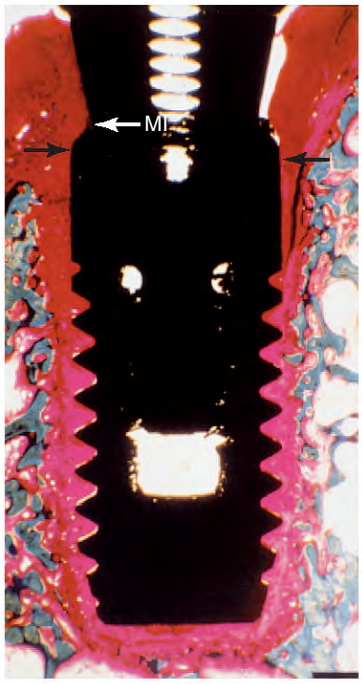


Figure 4-9 Implant body fracture may occur. It is most often related to parafunction and occurs with greater frequency for CP grade 1 titanium implants.

(Figure 4-7). Metal framework fractures also have been reported in an average of 3% of fixed complete and overdenture restorations, with a range of 0% to 27% (Figure 4-8). Implant body fracture has the least incidence of this type of complication, with an occurrence of 1% (Figure 4-9). This condition is reported with more frequency in long-term fixed prostheses and may even account for the majority of long-term failures. Resin veneer fractures of fixed implant prostheses averaged 22%, overdentures clip/attachment fractures averaged 17%, porcelain veneer fractures averaged 14%, overdenture fractures averaged 12%, and acrylic-base resin fractures averaged 7%. Prostheses-related fractures far outnumber implant component fractures.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Box 4-2 Effects of Crestal Bone Loss

1. Early implant failure (especially in soft bone or short implants)
2. Crestal bone loss may have an occlusal stress component
3. Prosthetic screw loosening
4. Abutment screw loosening
5. Acrylic resin veneer fracture
6. Porcelain fracture
7. Unretained cemented restoration
8. Prosthetic framework fracture
9. Overdenture attachment adjustments
10. Acrylic base fracture of overdentures
11. Overdenture attachment fracture
12. Abutment screw fracture
13. Implant body fracture
14. Esthetic complications
15. Peri-implant disease

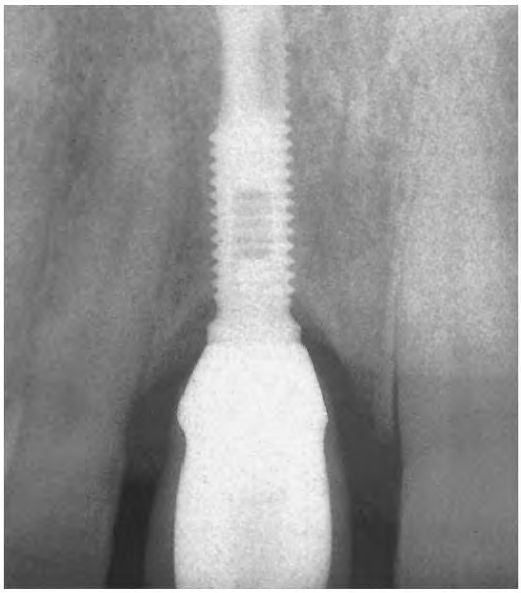


Figure 4-10 Marginal bone loss around the crestal portion of an implant often occurs during the first year of occlusal loading.

Uncemented restorations (or worse, partially uncemented prostheses) occur most likely when chronic loads are applied to the cement interface, or when shear forces are present (as found with cantilevers). Cement strengths are weakest in shear loads. Zinc phosphate cement may resist a compressive force of 12,000 psi but can only resist a shear force of 500 psi. It is interesting to note that bone is also strongest to compression and 65% weaker to shear forces. A similar scenario relative to shear load is found with porcelain or other occlusal materials. As a consequence, the evaluation, diagnosis, and modification of treatment plans related to stress conditions are of considerable importance. Therefore, once the implant dentist has identified the sources of additional force on the implant system, the treatment plan is altered in an attempt to minimize their negative impact on the longevity of the implant, bone, and final restoration.

MARGINAL BONE LOSS

Crestal bone loss has been observed around the permucosal portion of dental implants for decades. It has been described in the crestal region of successfully osteointegrated implants regardless of surgical approaches. It can range from loss of marginal bone to complete failure of the implant[17,23,37,38] and dramatically decreases after the first year (Box 4-2). For the one-piece blade implants, this phenomenon was described as a "saucerization" and occurred after implant loading.[37]

Occlusal Trauma: Bone Loss

Adell et al.[23] were the first to quantify and report marginal bone loss. The study also indicated greater magnitude and occurrence of bone loss during the first year of prosthesis loading, averaging 1.2 mm during this time frame, with a range of 0 to 3 mm. This report measured bone loss from the first thread as the 0-mm baseline, not from the original level of crestal bone at insertion, which was 1.8 mm above this baseline point. Thus the actual first-year crestal bone loss averaged 3.3 mm around the implants observed (Figure 4-10). Years subsequent to the first showed an average of 0.05 to 0.13 mm bone loss per year. Other studies report an average first-year bone loss of 0.93 mm, with a range from 0.4 to 1.6 mm and a mean loss of 0.1 mm after the first year.[31,32] The early crestal bone loss has been observed so frequently that proposed criteria for successful implants often do not even include the first-year bone loss amount.[39]

The initial transosteal bone loss around an implant forms a V- or a U-shaped pattern, which has been described as *ditching* or *saucerization* around the implant. The current hypotheses for the cause of crestal bone loss have ranged from reflection of the periosteum during surgery, preparation of the implant osteotomy, the position of the microgap between the abutment and implant body, micromovement of the abutment components, bacterial invasion, the establishment of a biological width, and factors of stress.[23,17,38-42]

An understanding of the causes of marginal crestal bone loss around dental implants and early implant failure is critical in preventing such occurrences, fostering long-term periimplant health, and improving long-term implant success rates and, foremost, implant prosthesis success. Marginal crestal bone loss may influence esthetics, as the height of the soft tissue (e.g., interdental papilla) is directly related to the marginal bone. If the tissue shrinks as a consequence of the bone loss, the emergence profile of the crown elongates and the papilla may disappear next to the adjacent tooth or

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

implant. If the soft tissue does not shrink, the increase in pocket depth may be related to the presence of anaerobic bacteria and peri-implantitis.

Over the years, the cause of marginal bone loss has kept the implant community busy with academic debates and clinical studies. However, clinical consequences are such that all phases of implant dentistry, from diagnosis and treatment planning to the final stages of occlusion and prosthesis delivery, must focus on its reduction or elimination.

Periosteal Reflection Hypothesis

Periosteal reflection causes a transitional change in the blood supply to the crestal cortical bone. Ninety percent of the arterial blood supply and 100% of the venous return are associated with the periosteum in the long bones of the body.[43] When the periosteum is reflected off the crestal bone, the cortical bone blood supply is affected dramatically, causing osteoblast death on the surface from trauma and lack of nutrition. These events have fostered the periosteal reflection theory as a cause for early bone loss around an endosteal implant.

Although crestal bone cells may die from the initial trauma of periosteal reflection, the blood supply is reestablished once the periosteum regenerates. Cutting cones develop from monocytes in the blood and precede new blood vessels into the crestal regions of bone. Osteoblasts then are able to remodel the crestal bone anatomy.[44] Composite bone forms rapidly on periosteal surfaces to restore its original condition. In addition, the underlying trabecular bone is also a vascular source because its blood supply often is maintained in spite of crestal periosteal reflection. The greater the amount of trabecular bone under the crestal cortical bone, the less crestal bone loss is observed.[45] To place the implant in sufficient available bone, an implant ridge is usually 5 mm or wider at the crest. As a result, trabecular bone is readily available to assist in cortical blood supply and remodeling around the implants. The cortical bone is remodeled to its original contour, without significant loss of height.

The periosteal reflection theory would lead to a generalized horizontal bone loss of the entire residual ridge reflected, not the localized ditching pattern around the implant that typically is observed. In addition, generalized bone loss already would be directly noticeable at the second-stage uncovery of the implant body, 4 to 8 months after Stage I implant placement surgery. Yet generalized bone loss rarely is observed at the second-stage uncovery surgery (Figure 4-11). Therefore, the periosteal reflection hypothesis does not appear as a primary causal agent of marginal crestal bone loss around an implant.

Implant Osteotomy Hypothesis

Preparation of the implant osteotomy has been reported as a causal agent of early implant bone loss. Bone is a labile organ and is sensitive to heat. The implant osteotomy causes trauma to the bone in immediate contact with the implant, and a devitalized bone zone of about 1 mm is created around the implant. A renewed blood supply and cutting cones are necessary to remodel the bone at the interface. The crestal region is more susceptible to bone loss during initial repair because of its limited blood supply and the greater heat generated in this denser bone, especially with the less efficient cutting of countersink drills used in this region.[45-47] This condition supports implant osteotomy preparation as a causal agent for marginal crestal bone loss around the implant.

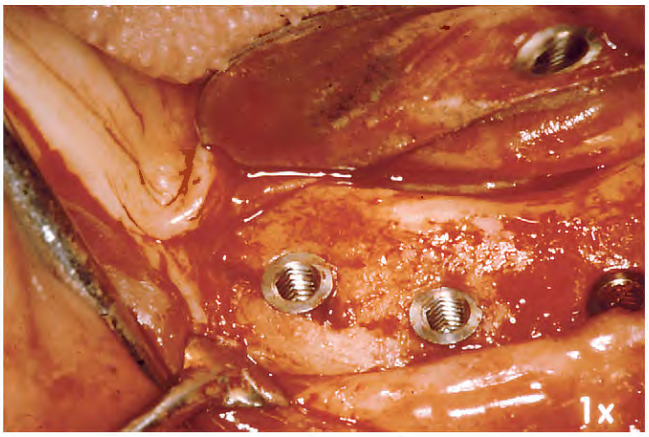


Figure 4-11 At Stage II uncovery, the marginal bone level most often is similar to the initial day of surgery and may even have grown over the top of the implant. Because the periosteum is reflected and bone is prepared to insert the implants, the cause of frequently occurring early crestal bone loss may not be related to the reflection of the periosteum or the osteotomy preparation.

However, if heat and trauma during implant osteotomy preparation were responsible for marginal crestal bone loss, the effect would be noticeable at the second-stage uncovery surgery 4 to 8 months later. The average bone loss of 1.5 mm from the first thread is not observed at Stage II uncovery. In fact, bone often has grown over the first-stage cover screw, especially when level or slightly countersunk below the bone (see Figure 4-11). Reports in the literature indicate different surgical trauma causes and numbers for bone loss. For example, Manz[48] observed that bone loss at second-stage surgery ranged from 0.89 to 0.96 mm regardless of the bone density. Hoar et al.[49] reported only 0.2-mm bone loss at Stage II uncovery. The surgical system or approach may influence these data, but usually this bone loss remains minimal.

One should remember that these are averages of bone loss reported. Therefore if 2 mm of bone loss is found on one implant, and the next nine implants exhibit no bone loss, the average bone loss would be 0.2 mm. Most implants at Stage II uncovery do not demonstrate any bone loss. Therefore the implant osteotomy hypothesis for marginal crestal bone loss cannot be primarily responsible for this routinely observed phenomenon.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Autoimmune Response of Host Hypothesis

The primary cause of bone loss around natural teeth is bacteria induced. Repeat studies demonstrate that bacteria are the causative element for vertical defects around teeth. Occlusal trauma may accelerate the process, but trauma alone is not deemed a determining factor.[50] The implant gingival sulcus in the partially edentulous implant patient exhibits a bacterial flora similar to that of natural teeth.[1] A logical assumption is that if implants are similar to teeth, the marginal implant bone loss is caused primarily by bacteria, with occlusal factors playing a contributing or accelerating role.

In a prospective study of 125 implants, Adell et al.[38] reported 80% of implant sulcular regions were without inflammation. Lekholm et al.[51] found that deep gingival pockets around implants were not associated with crestal bone loss. Yet the marginal crestal bone loss to the first thread of screw-type implants is a common radiologic finding. If bacteria were the causal agent for the initial bone loss, why does most bone loss occur the first year (1.5 mm) and less (0.1 mm) each successive year? The implant sulcus depth progressively increases from the early bone loss, impairing hygiene and making anaerobic bacteria more likely as the cause of bacteria-related bone loss. If bacteria are responsible for 1.5-mm early crestal bone loss, what local environmental changes occur to reduce their effect by 15 times after the first year?[23] The bacteria autoimmune theory cannot explain the marginal bone loss condition when it follows the pattern most often reported.

Although the bacteria theory does not explain adequately the marginal crestal bone loss phenomenon, this does not mean that bacteria are not a major contributor to bone loss around an implant. Threads and porous implant surfaces exposed to bacteria are reported to cause a more rapid loss of bone around an implant.[51] Poor hygiene also is reported to accelerate the bone loss observed around endosteal implants[52,53] (Figure 4-12). To state that bacteria are never involved in marginal bone loss around an implant would be incorrect. Bone loss often is associated with bacteria as a causal agent. However, when most bone loss occurs in the first year and less bone loss is observed afterward, the hypothesis of bacteria as the primary causal agent for the early crestal bone loss cannot be substantiated.

Figure 4-12 Exudate around an implant is more likely to be present when the probing depth is greater than 5 mm and an aerobic environment exists around the implant.

Biological Width Hypothesis

The sulcular regions around an implant and around a tooth are similar in many respects. The rete peg formation within the attached gingiva and the histologic lining of the gingiva within the sulcus are similar in implants and teeth. A free gingival margin forms around an implant with nonkeratinized sulcular epithelium, and the epithelial cells at its base are similar to the functional epithelial cells described with natural teeth.[54] However, a fundamental difference characterizes the base of the gingival sulcus.

For a natural tooth, an average biological width of 2.04 mm exists between the depth of the sulcus and the crest of the alveolar bone (Figure 4-13). It should be noted the biological "width" is actually a height dimension with a greater range in the posterior region compared with the anterior, and may be greater than 4 mm in height. In teeth, it is composed of a connective tissue (CT) attachment (1.07 mm average) above the bone and a junctional epithelial attachment (0.97 mm average) at the sulcus base, with the most consistent value between individuals being the CT attachment.[55-57] The biological width allows gingival fibers and hemidesmosomes to establish direct contact with the natural

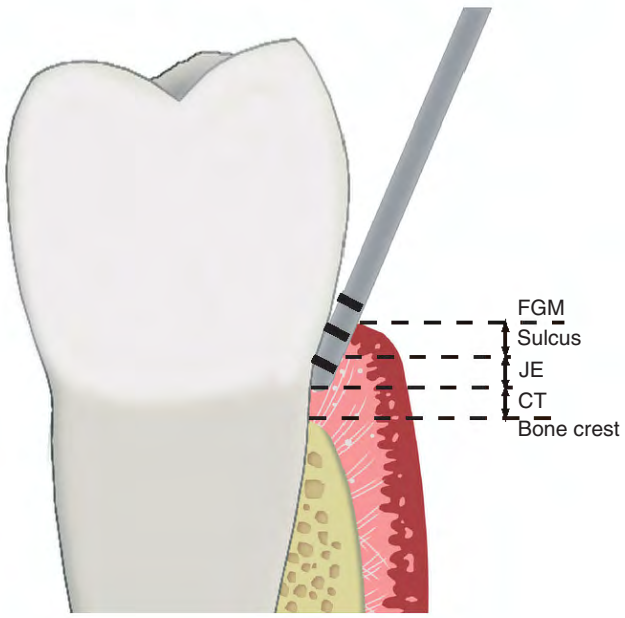


Figure 4-13 The biological width of a natural tooth has a connective tissue zone that inserts into the cementum of the tooth. A periodontal probe will penetrate the sulcus and the junctional epithelial attachment. *FGM,* Free gingival margin; *JE,* junctional epithelium; *CT,* connective tissue.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

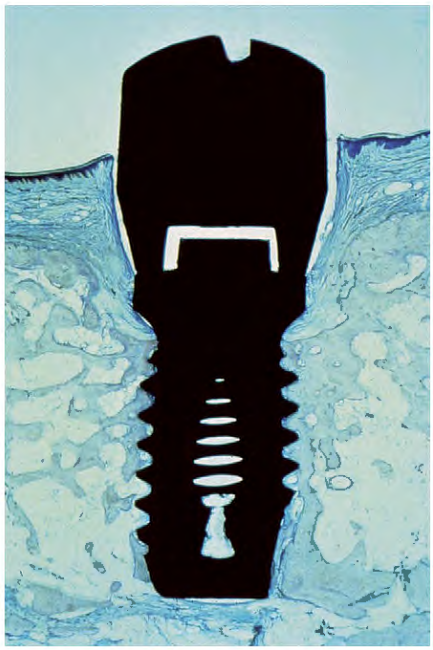


Figure 4-14 The bone levels recede to at least 0.5 mm around an abutment to implant connection after the implant extends through the soft tissue, regardless of whether the implant is loaded. *(Courtesy Steve Wallace.)*

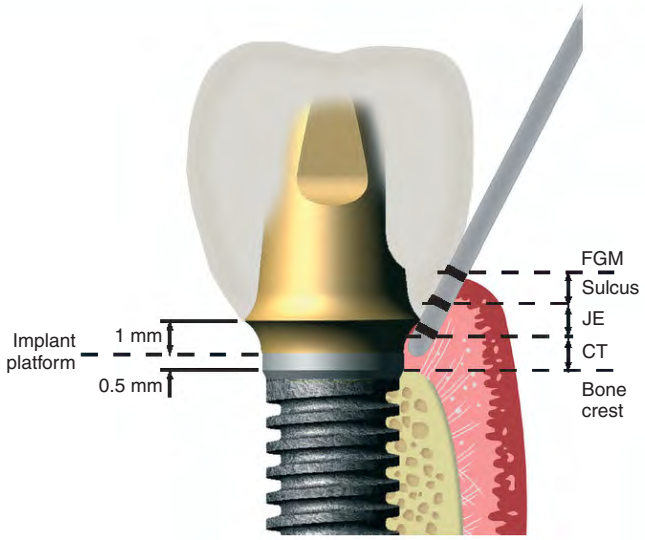


Figure 4-15 There are primarily two soft tissue fiber groups around an implant: circular fibers and crestal bone fibers. Neither of these fiber types insert into the implant or the abutment. The peri-implant probe penetrates the sulcus, junctional epithelial attachment, and most of the connective tissue zone. *FGM,* Free gingival margin; *JE,* junctional epithelium; *CT,* connective tissue.

tooth and acts as a barrier to the bacteria in the sulcus to the underlining periodontal tissues. When a crown margin invades the biological width, the crestal bone recedes to reestablish a favorable environment for the gingival fibers.[58,59]

Many surgical protocols recommend the placement of endosteal implants at or below the crest of the ridge during the first-stage surgery. The abutment-to-implant body connection may be compared with a crown margin. Berglundh et al.[60] observed 0.5 mm of bone loss below the implant-abutment connection within 2 weeks after stage II uncovery and abutment connection in dogs (Figure 4-14). Lindhe et al.[61] reported an inflammatory connective tissue extending 0.5 mm above and below this implant abutment connection. Wallace and Tarnow[62,63] stated that the biological width also occurs with implants and may contribute to some of the marginal bone loss observed. The biological width theory seems attractive to explain the lack of bone loss from the first stage of surgery and the early bone loss seen within the first year after the second-stage abutment placement. However, it should be noted that the biological "width" in implants, as reported, often includes the sulcus depth, whereas the natural tooth biological width does not include the sulcus depth.

Eleven different gingival fiber groups are observed around a natural tooth: dentogingival (coronal, horizontal, and apical), alveologingival, intercapillary, transgingival, circular, semicircular, dentoperiosteal, transseptal, periosteogingival, intercircular, and intergingival. At least six of these gingival fiber groups insert into the cementum of the natural tooth: the dentogingival (coronal, horizontal, and apical), dentoperiosteal, transseptal, circular, semicircular, and transgingival fibers. In addition, some crestal fibers from the periodontal fiber bundles also insert into the cementum above the alveolar bone.[57] However, in a typical implant gingival region, only two of these gingival fiber groups and no periodontal fibers are present (Figure 4-15). These fibers do not insert into the implant body below the abutment margin as they do into the cementum of natural teeth.[56,64] Instead, the collagen fibers in the CT attachment around an implant run parallel to the implant surface, not perpendicular, as with natural teeth.[65,66] The gingival and periosteal fiber groups are responsible for the connective tissue attachment component of the biological width around teeth, and these are not present around the transosteal region of an implant. Therefore the CT attachment around the abutment-implant connection cannot be compared with the CT attachment of a tooth.

James and Keller[64] were first to begin a systematic scientific study to investigate the biological seal phenomenon of the soft tissue around dental implants. Hemidesmosomes help form a basal lamina–like structure on the implant, which can act as a biological seal. However, collagenous components of the linear body cannot physiologically adhere to or become embedded into the implant body as they do in the cementum of the tooth.[67] The hemidesmosomal seal only has a circumferential band of gingival tissue to provide mechanical protection against tearing.[68] Therefore the biological seal around dental implants can prevent the migration of bacteria and endotoxins

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

into the underlying bone. It is unable, however, to constitute a junctional epithelial attachment component of the biological width similar to the one found with natural teeth. The amount of early crestal bone loss therefore seems unlikely to be solely the result of the remodeling of the hard and soft tissues to establish a biological width below an abutment connection. No connective tissue attachment zone or components of the linear body are embedded into an implant. The importance, amount, and mechanism for these anatomical structures require further investigation.

The crevice between the cover screw and the implant body during initial healing is similar to the crevice of the abutment-implant connection. Yet bone can grow over the cover screw, and therefore the crevice, in and of itself, may not be the cause of bone loss. The crevice between the implant and the abutment connection has been called a "microgap." The actual dimension of this connection is usually 0 µm and has a direct metal to metal connection. However, when the crevice is exposed to the oral environment, bone loss is usually observed for at least 0.5 mm below the connection.[68-70]

The biological width hypothesis cannot fully explain the several millimeters of marginal crestal bone loss, which also has been observed readily with one-stage implants that extend through the tissue at the initial implant placement surgery and have no abutment-implant connections. For example, plate form (blade) implants, transosteal implants, pins, one-piece screw implants, and even subperiosteal implants demonstrate the marginal crestal bone loss phenomenon.

It is true that, bone loss does occur around an exposed abutment-implant connection placed below the bone and is observed within 2 to 4 weeks, once the connection is exposed to the oral environment. The bone loss often occurs before the implant is loaded with the prosthesis. It is logical to call this marginal bone loss the *biological width*.

The primary question remains, when the surgeon places the implant abutment connection below the bone, how much bone loss is from the implant biological width, and therefore out of the influence of the dental practitioner? Several reports in the literature note implant macro- and microgeometry may affect the biological width dimensions or the amount of early crestal bone loss.[31,32,49,71-74]

The bone loss to the first thread observation implies the amount of bone loss is similar for different implant designs. However, the first thread is a different distance from the abutment margin for several implant designs. A smooth polished 4-mm collar below the bone has been associated with greater bone loss than a smooth 2-mm collar below the bone. The implant biological width concept does not explain completely the total amount of vertical bone loss observed. In addition, the amount of bone loss from the biological width occurs within 1 month, whether the implant is loaded or not, and is related to the crest module implant design and the position of the abutment-implant connection in relation to the bone but is unrelated to the density of the bone. The concept does not explain why greater crestal bone loss often is observed in soft bone compared with denser bone after loading, nor does it explain the higher implant failure rates in lesser-quality bone after loading.

Occlusal Trauma

Marginal bone loss on an implant may be from occlusal trauma.[40] *Occlusal trauma* may be defined as an injury to the attachment apparatus as a result of excessive occlusal force.[1] A controversy exists as to the role of occlusion in the bone loss observed after an implant prosthesis delivery.[8] Some articles state that peri-implant bone loss without implant failure is primarily associated with biological formations or complications.[16-18] Other authors suggest a correlation of crestal bone loss to occlusal overload.[8,40,41,75,76] The determination of the etiology of bone loss around dental implants is needed in order to minimize its occurrence and foster long-term peri-implant health that may ultimately determine implant prosthesis survival.

The association of occlusal trauma and bone loss around natural teeth has been debated since Karolyi claimed a relationship in 1901.[76] A number of authors conclude trauma from occlusion is a related factor in bone loss, although bacteria is a necessary agent.[77-82] On the other hand, Waerhaug and many others state there is no relationship between occlusal trauma and the degree of periodontal tissue breakdown.[83-85] According to Lindhe et al., "trauma" from occlusion cannot induce periodontal tissue breakdown.[86] However, occlusal trauma may lead to tooth mobility that can be transient or permanent. By extrapolation of this rationale, several authors have also concluded occlusal trauma is not related to marginal bone loss around a dental implant.[16-18]

To establish further a correlation between marginal bone loss and occlusal overload, related articles from cellular biomechanics, engineering principles, mechanical properties of bone, physiology of bone, implant design biomechanics, animal studies, and clinical reports were procured.[40]

Cellular Biomechanics

Bone remodeling at the cellular level is controlled by the mechanical environment of strain.[87] *Strain* is defined as the change in length divided by the original length, and the units of strain are given in percentages. The amount of strain in a material is directly related to the amount of stress applied.[88] Occlusal stress applied through the implant prosthesis and components can transmit stress to the bone-implant interface.[87] The amount of bone strain at the bone-implant interface is directly related to the amount of stress applied through

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

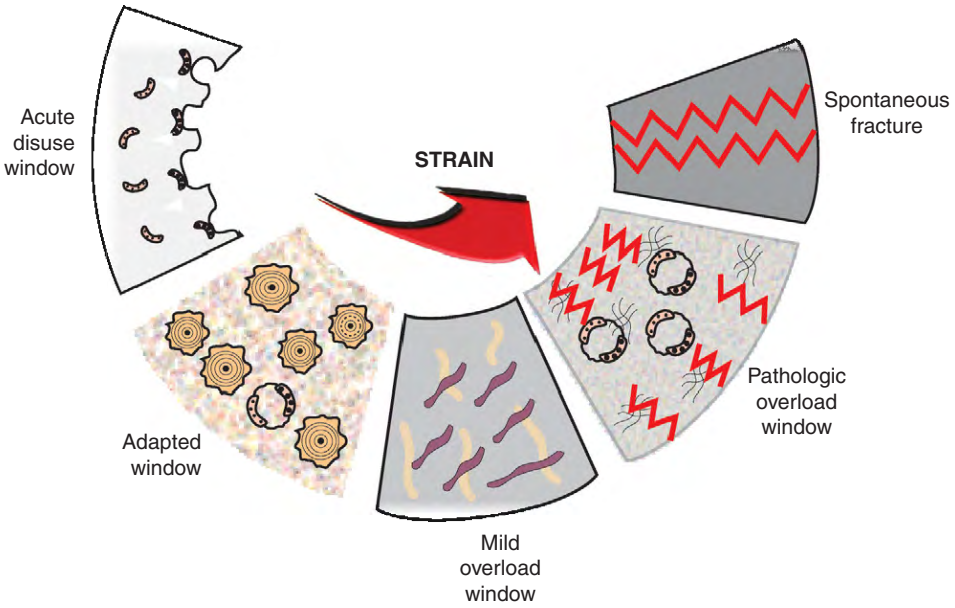


Figure 4-16 Mechanical stress applied to bone cells causes a change in shape or strain. The microstrains may trigger the release of cytokines and bone resorption.

the implant prosthesis. Mechanosensors in bone respond to minimal amounts of strain, and microstrain levels 100 times less than the ultimate strength of bone may trigger bone remodeling[89] (Figure 4-16).

One of the earliest remodeling theories for a direct relationship between stress and the magnitude of bone remodeling was proposed by Kummer in 1972.[90] More recently, Frost reported on the cellular reaction of bone to different microstrain levels.[91,92] He observed that bone fractures at 10,000 to 20,000 microstrain units (1% to 2% deformation). However, at levels 20% to 40% of this value (4000 units), bone cells may trigger cytokines to begin a resorption response. In other words, excessive bone strain may not only result in physical fracture, but may also cause bone cellular resorption. Therefore the hypothesis that occlusal stresses beyond the physiologic limits of bone may result in strain in the bone significant enough to cause bone resorption is plausible from a cellular biomechanics standpoint. To date, bone cellular studies have not replicated this bone condition next to a dental implant. However, cytokines in the bone-implant interface tissue obtained from failed hip replacement devices leading to bone loss have been reported in humans.[93]

Engineering Principles

The relationship between stress and strain determines the modulus of elasticity (stiffness) of a material.[88] The modulus conveys the amount of dimensional change in a material for a given stress level. The modulus of elasticity of a tooth is similar to cortical bone. Dental implants are typically fabricated from titanium or its alloy. The modulus of elasticity of titanium is five to ten times greater than that of cortical bone (Figure 4-17). An engineering principle called the *composite beam analysis* states that when two materials of different elastic moduli are placed together with no intervening material and one is loaded, a stress contour increase will be observed where the two materials first come into contact.[94] In an implant-bone interface, these stress contours are of greater magnitude at the crestal bone region. This phenomenon was observed in both photoelastic and three-dimensional finite element analysis (Figure 4-18) studies when implants were loaded within a bone simulant.[95-98] These authors note that the marginal bone loss observed clinically and radiographically around implants follows a similar pattern as the stress contours in these reports.

Bone Mechanical Properties

Bone density is directly related to the strength and elastic modulus of bone.[99] In denser bone, there is less strain under a given load compared with softer bone. As a result, there is less bone remodeling in denser bone compared with softer bone under similar load conditions.[91] A decrease in bone remodeling can result in a reduction of bone loss. In a prospective human study, Manz observed the amount of marginal bone loss next to an implant was related to the density of bone.[48] The initial peri-implant bone loss from implant insertion to uncovery was similar for all bone qualities. However, 6 months after prosthesis delivery, the additional radiographic-observed peri-implant bone loss ranged from 0.68 mm for quality 1 to 1.1 mm for quality 2, 1.24 mm for quality 3, and 1.44 mm for quality 4-type bone (Figure 4-19). In other words, the more dense the bone, the less peri-implant bone loss was observed after prosthesis delivery. A clinical report by Appleton

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

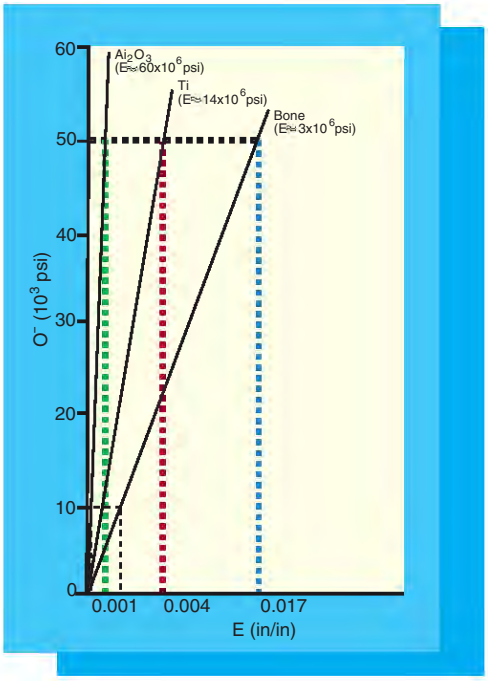


Figure 4-17 The modulus of elasticity is greater for titanium (Ti) compared with bone. When stress is plotted on the Y axis and strain on the X axis, the modulus of elasticity can be obtained. Titanium is five to ten times more rigid than cortical bone.

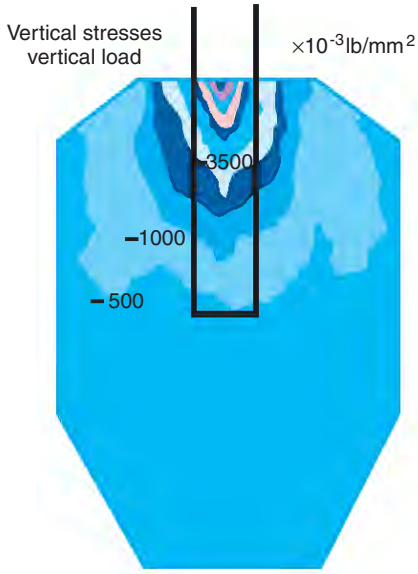


Figure 4-18 A three-dimensional finite element analysis of a titanium implant in a bone model after axial loading. The V-shape pattern of strain is greatest at the crestal region and decreases in intensity as the stress is dissipated throughout the implant length.

et al.[100,101] demonstrated that progressively loaded single-tooth implants in the first premolar region of human beings exhibited greater bone density increase in the crestal half of the implant interface and less marginal bone loss compared with nonprogressively loaded implants in the same jaw region and even the same patient on the contralateral side without progressive loading. Because an increase in bone density is related to bone strength, elastic modulus, bone remodeling, and a decrease in marginal bone loss, these entities may be related to each other.

Animal Studies

Several animal studies in the literature demonstrate the ability of bone tissue to respond to a dental implant. For example, Hoshaw et al. inserted dental implants into a dog femur perpendicular to the axis of the long bone and perpendicular to the direction of the osteons.[102,103] After applying a tensile load to the implants for only 5 days, the bone cells reorganized to follow the implant thread pattern and resist the load. This unique bone pattern was only observed for 3 to 4 mm around the implants. Crestal bone loss was also noted around these implants and explained as stress overload. To rearrange its osteal structure, bone must remodel.

Miyata placed crowns on integrated dental implants with no occlusal contacts (control group), and premature interceptive occlusal contacts of 100 µm, 180 µm, and 250 µm in a monkey animal model.[104-106] After 4 weeks of premature occlusal loads, the implants were removed in a block section and evaluated. The crestal bone levels for 100 µm and control implants with no loading were similar. However, statistically significant crestal bone loss was observed in the 180-µm group (Figure 4-20). The 250µm group experienced two to three times the bone loss of

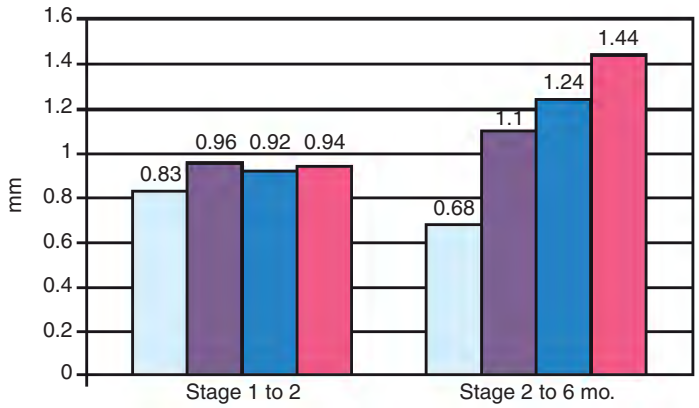


Figure 4-19 Mean peri-implant vertical bone change for study intervals by bone quality score. Many observed the amount of bone loss from Stage 1 to Stage 2 was similar, regardless of bone quality. However, after 6 months of loading, the amount of marginal bone loss was directly related to the quality of the bone, with type 4 (the softest bone) exhibiting the greatest bone loss. *(Reproduced from Manz MC: Radiographic assessment of peri-implant vertical bone loss: DIRG Implant Report No 9,* J Oral Maxillofac Surg *55 (Suppl):62-71,1997.)*

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Figure 4-20 Miyata et al. loaded integrated implants for 4 weeks with premature contacts on crowns of 100 μm, 180 μm, and 250 μm. The implants with 180 μm premature contacts demonstrate a V-shaped crestal bone loss. *(From Miyata T, Kobayashi Y, Araki H et al: The influence of controlled occusal overload on periimplant tissue: 3, a histologic study in monkeys,* Int J Oral Maxillofac Implants *15:425-431, 2000.)*

Figure 4-21 The implants with 250-μm premature contacts demonstrated greater bone loss than the 180-μm group. The 250-μm premature contact for 4 weeks lost the most bone. The higher stresses resulted in more crestal bone loss. *(From Miyata T, Kobayashi Y, Araki H et al: The influence of controlled occusal overload on periimplant tissue: 3, a histologic study in monkeys,* Int J Oral Maxillofac Implants *15:425-431, 2000.)*

the crowns with moderate prematurities (Figure 4-21). Duyck used a dog model to evaluate the crestal bone loss around screw-type dental implants with no loads (controls), static loads, and dynamic loads.[107] The dynamic-loaded implants were the only group to demonstrate crestal bone loss. As the only variable in these two studies was the intensity or type of occlusal load applied to the implants, these animal reports imply dynamic occlusal loading may be a factor in crestal bone loss around rigid fixated dental implants.

Clinical Reports

Clinical reports have shown an increase in marginal bone loss around implants closest to a cantilever used to restore the lost dentition[108-110] (Figure 4-22). Cantilever length and an increase in occlusal stress to the nearest abutment are directly related[111] and point to the fact that the increase in marginal bone loss may be related to occlusal stress. Quirynen et al. evaluated 93 implant patients with various implant restorations and concluded that the amount of crestal bone loss was definitely associated with occlusal loading.[31] These authors also reported increased crestal bone loss around implants in patients with no anterior occlusal contacts and parafunctional habits in full-arch fixed prostheses in both jaws.[31,32] These clinical reports do not provide statistical analyses to demonstrate a clear link between occlusal stress and bone loss. However, they indicate a consensus by some authors that occlusal overload

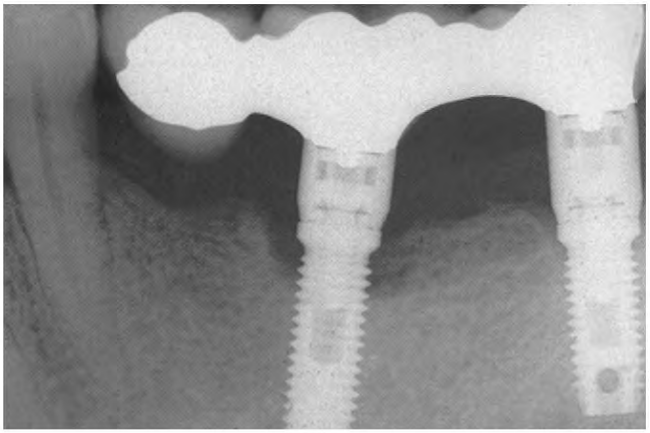


Figure 4-22 Rangert et al. observed that cantilevers on fixed partial dentures increase the marginal bone loss on the implant next to the cantilever. Note the more distal implant with less bone loss. *(From Rangert B, Krogh PHJ, Langer B et al: Bending overload and implant fracture: a retrospective clinical analysis,* Int J Oral Maxillofac Implants *10:326-334, 1995.)*

may be related to the incidence of peri-implant bone loss around the cervical aspect of an implant. In fact, in a study of 589 consecutive implants, Naert et al. suggested overload from parafunctional habits may be the most probable cause of implant loss and marginal bone loss after loading.[112]

Rangert et al. have noted that occlusal loads on an implant may act as a bending moment, which increases

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان
لابراتوار دندانسازی های دنت

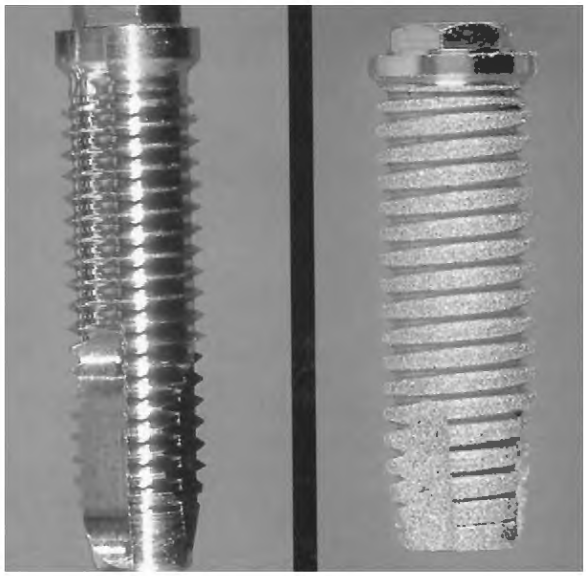


Figure 4-23 Researchers have compared crestal bone loss for the implant design on the *left* (a V-shaped machined thread) with the implant design on the *right* (a roughened square thread) in the anterior mandible for 3 to 7 years.

stress at the marginal bone level and can cause implant body fracture.[33] Before the fracture of the implant body, marginal bone loss was noted in this retrospective clinical evaluation. The same stress that caused implant fracture is the logical cause of the peri-implant bone loss before the event.

Rosenberg et al. found microbial differences in implant failures from both overload and biological complications.[75] Uribe et al. presented the case of a mandibular implant crown with a marginal peri-implantitis and osseous defect.[113] Histologic analysis revealed an infiltrate and a central zone of dense fibroconnective tissue with scanty inflammatory cells. According to the authors, this finding differs from chronic inflammatory tissue associated with infectious peri-implantitis and can be directly related to occlusal overload.

A clinical report by Leung et al. observed radiographic angular crestal bone loss to the 7th thread around one of two implants supporting a fixed prosthesis in hyper-occlusion 2 weeks after prosthesis delivery.[114] The prosthesis was removed and over the next few months, radiographic observation showed the crestal defect was repaired to almost the initial level, without any surgical or drug intervention. The prosthesis was then seated with proper occlusal adjustment. The bone levels stabilized at the second thread of the implant and remained stable over the next 36 months. This report indicates bone loss from occlusal overload is not only possible, but may even be reversible when found early in the process. Therefore, although no prospective clinical study to date has clearly demonstrated a direct relationship between stress and bone loss without implant failure, several practitioners agree a causal relationship may exist.

Implant Design Biomechanics

Different amounts of marginal bone loss have been reported for different implant body designs. The design and surface condition of the implant body may affect the amount of strain distributed to an implant-bone interface. A report by Zechner et al. evaluated the peri-implant bone loss around functionally loaded screw-type implants with machined surfaced V-threads or a sandblasted/acid-etched square-thread design[115] (Figure 4-23). Both these implant designs had a similar crest module and external hex connection. Four interforaminal implants were placed in the mandible in 36 patients and followed for 4 years. Over this period, the average bone loss was 2.4 mm (V-thread) versus 1.6 mm (square thread). However, the range of bone loss in the study was 0.1 to 8.5 mm for machined V-threaded implants and 0.2 to 4.8 mm for rough-surfaced square-threaded implants. Twenty-two V-threaded implants lost more than 4 mm of bone of less than 1 mm was reported for 16 rough-surface square-threaded implants compared with only two machined-surfaced V-threaded implants (Figure 4-24). There were no clinical findings of inflammation or exudate. The range of bone loss with the different implant surface conditions and designs in a clinical report suggests that more than the biological width, microgap position, and/or surgical causes are involved in the individual implant marginal bone loss process. The three most probable factors that influenced the amount of crestal bone loss in this report are the amount of force applied to the prosthesis, the quality of the bone to resist these forces, and the implant body design. All three of these conditions implicate occlusal overload as the cause of marginal bone loss around an implant.

A prospective study by Karousis et al. also indicated that different implant designs and surface conditions correspond to different incidences of crestal bone loss.[116] Three different implant designs from the

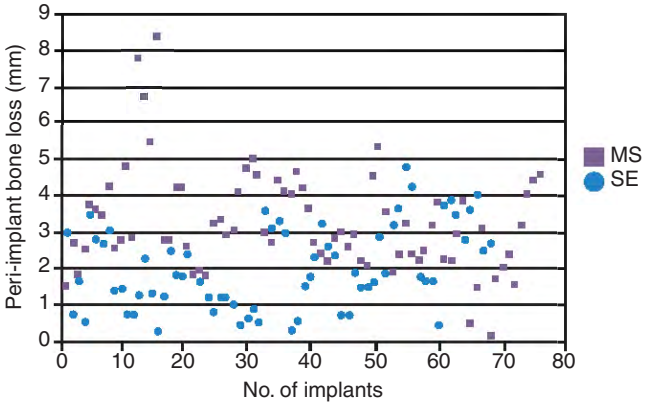


Figure 4-24 Most of the implants that lost more than 4 mm of bone were of the machined V-shaped-thread design. The implants that lost less than 1 mm of bone were primarily roughened square-thread designs.

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

same manufacturer were evaluated over 10 years in a prospective report. One implant body design lost more than 5 mm of bone 26% of the time, whereas the other two designs reported 37% and 39% incidence. More than 6 mm of marginal bone loss occurred in 22% of the implants with the first design, compared with 35% and 33% for the other two designs. These results indicate that one implant design may result in less marginal bone loss than another and point to the fact that clinical reports with similar healing and loading protocols, but of variable implant body designs and surface conditions, may yield different amounts of crestal bone loss. Because the implant design and surface condition affect the amount of stress transferred to the bone, one of the reasons for a different amount of bone loss for different implant designs may be related to the stress transmitted to the bone.

In the field of orthopedics, hip joint replacement has several complications, including wound infection, periprosthetic fracture, dislocation, mechanical failure, and osteolysis.[117] *Osteolysis* refers to the bone resorption that occurs around both cemented and uncemented orthopedic implants (Figure 4-25). Aseptic loosening from osteolysis of the bone-implant interface is the leading cause of late joint replacement failure (10% within 10 years). Mechanical loading factors primarily are associated with this condition.[118-120] Patient factors that increase loading failure include body weight and activity level.[120] An animal model and human report have linked the resorption of bone at the interface to mechanical overload.[121] Treatment of the disorder, if the patient is asymptomatic with a large osteolytic defect but no implant mobility, includes curettage of the osteolytic membrane and bone grafting.[122] These orthopedic reports accept that mechanical overload can cause bone resorption at the bone-implant interface. The metal most often used in hip replacement therapy is titanium alloy, and the bone-implant interface is very similar to a dental implant. In addition, potential causative elements encountered intraorally, such as oral bacteria contamination, microgap position, and microbial-related bone loss, are eliminated in this aseptic environment. It is logical to assume these studies further support a relationship between marginal bone loss around implants and biomechanical stress.

Discussion

Limited marginal bone loss during the first year of function after Stage II surgery has been observed around the permucosal portion of dental implants for decades.[23,38] Hypotheses for the causes of crestal bone loss have included the reflection of the periosteum during surgery, preparation of the implant osteotomy, level of the microgap between the abutment and implant body, bacterial invasion, the establishment of a biological width, the implant crest module design, and occlusal overload.[8,40,41,123]

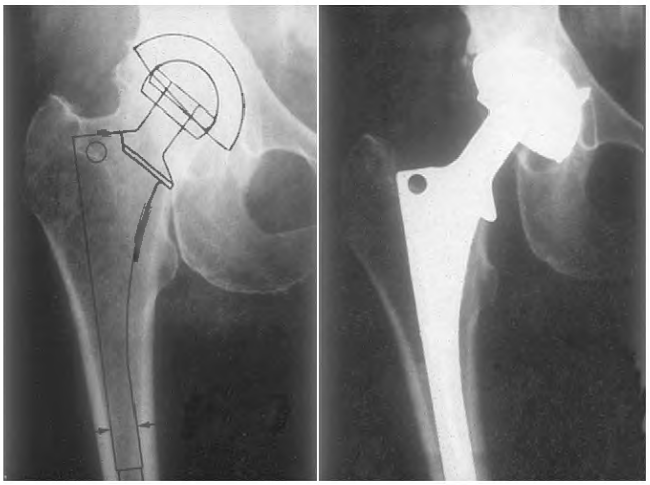


Figure 4-25 Bone loss around orthopedic implants (osteolysis) is primarily caused by mechanical stress to the bone-implant interface.

The fact that occlusal overload may be an etiology for crestal bone loss does not mean other factors are not present. For example, the microgap position of the implant platform and abutment and the biological width often affect the marginal bone during the first month after the implant becomes permucosal.[48] However, the clinician has certain variables under their control that may influence the amount of peri-implant bone loss. The position of the microgap in relation to the bony crest and the implant crest module design are primarily under the control of the implant surgeon. On the other hand, the autoimmune or bacterial response of the patient, the biological width, and the patient response to the surgical trauma of implant placement are variables often escaping the control of the dentist. Once the final prosthesis is delivered to the patient, many events responsible for marginal bone loss have already occurred, whereas others such as occlusal overload and its relationship to the quality of bone persist. Occlusal overload is one factor most in control of the restoring dentist. If a relationship between occlusal overload and crestal bone loss exists, approaches to decrease stress to an implant interface appear appropriate.

A puzzling element in the relationship between occlusal force and peri-implant bone loss is the lack of continued bone loss until the implant fails. Implant crown height may be measured from the occlusal plane to the crest of the bone. The crown height is a vertical cantilever, which may magnify the stresses applied to the prosthesis. As a result of the greater crown height from the vertical bone loss, occlusal overload will be increased after crestal bone loss occurs. Therefore, if occlusal loading forces can cause crestal bone loss, the resulting increased moment forces should further promote the loss of bone until the implant fails. Yet most clinical studies indicate the rate of bone loss decreases after the first year of loading and is minimal thereafter. There are

t.me/highdent www.highdentlab.com instagram.com/high_dent

همیار دندانسازان و دندانپزشکان لابراتوار دندانسازی های دنت

Figure 4-26 In evaluating the bone around an implant after healing in a monkey model, a fine trabecular pattern is noted. *(From Piatelli A, Ruggeri A, Franchi M et al: A histologic and histomorphometric study of bone reactions to unloaded and loaded non-submerged single implants in monkeys: a pilot study,* J Oral Implantol *19:314-319, 1993.)*

Figure 4-27 Once the implant was loaded, the fine trabecular bone became coarse trabecular bone, especially at the crestal region. When the stresses are too great, bone loss occurs. When the stresses are within the physiologic range, the bone density increases. *(From Piatelli A, Ruggeri A, Franchi M et al: A histologic and histomorphometric study of bone reactions to unloaded and loaded non-submerged single implants in monkeys: a pilot study,* J Oral Implantol *19:314-319, 1993.)*

two reasons why the bone levels may become stable after initial marginal bone loss, even when the cause is from occlusal overload: bone physiology and implant design mechanics.

Bone Physiology

The bone is less dense and weaker at Stage 2 implant surgery than it is 1 year later after prosthetic loading.[124] Bone is 60% mineralized at 4 months and takes 52 weeks to complete its mineralization.[125] Partially mineralized bone is weaker than fully mineralized bone. In addition, the microscopic organization of bone progresses during the first year. Woven bone is unorganized and weaker than lamellar bone, which is organized and more mineralized. Lamellar bone develops several months after the woven bone repair has replaced the devitalized bone caused by the surgical insertion trauma around the implant.[124] The occlusal stress levels may be high enough to cause woven bone microfracture or overload during the first year, but the increase in bone strength achieved after complete mineralization and organization may be able to resist the same stress levels during the subsequent years.

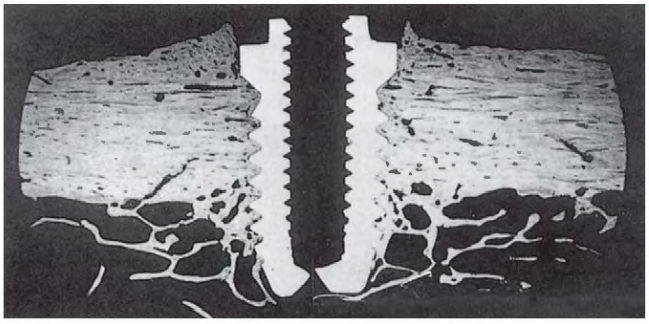
As functional forces are placed on an implant, the surrounding bone can adapt to the stresses and increase its density, especially in the crestal half of the implant body during the first 6 months to 1 year of loading.[125] In a histologic and histomorphometric study of bone, Piatelli et al. reported reactions to unloaded and loaded nonsubmerged implants in monkeys (Figures 4-26, 4-27). The bone changed from a fine trabecular pattern after initial healing to a more dense and coarse trabecular pattern after loading, especially in the crestal half of the implant interface.[126] Hoshaw loaded threaded implants in dogs with a tensile load and noted that the fine trabecular bone pattern became coarse trabecular bone around the implant.[102,103] In addition, the bone reorganized to a more favorable condition to assist the direction and type of occlusal load (Figure 4-28).

Fine trabecular bone is less dense than coarse trabecular bone.[124] Because the density of bone is directly related to its strength and elastic modulus, the crestal bone strength and biomechanical mismatch between titanium and bone may diminish gradually during the functional loading phase. In other words, the stresses applied to the peri-implant bone may be great enough to cause bone resorption during the first year, because bone strains are greatest at the crest. However, the stresses applied below the crest of bone are of less magnitude and may correspond to the physiologic strain that allows the bone to gain density and strength. As a result, the occlusal load that causes bone loss initially (overload) is not great enough to cause continued bone loss once the bone matures and becomes more dense.

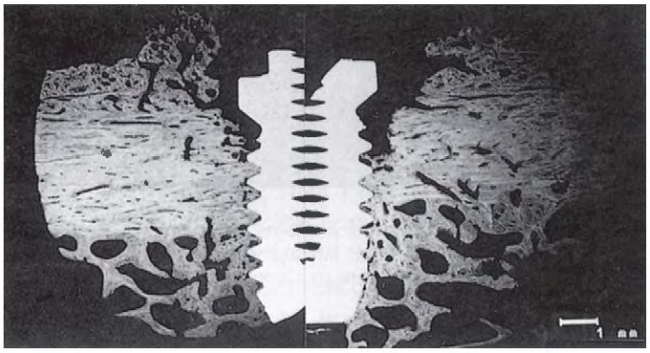
A clinical report by Appleton et al. demonstrated that progressively loaded single-tooth implants in the first premolar region of humans exhibited less bone loss and greater bone density increase in the crestal half of the implant interface, compared with nonprogressively loaded implants in the same jaw region, and even

t.me/highdent www.highdentlab.com instagram.com/high_dent

WWW.HIGHDENT.IR



A



B

Figure 4-28 Researchers loaded threaded implants in dog tibiae and noted the fine trabecular bone in the apical region **(A)** became coarse trabecular after loading. In addition, crestal bone loss was observed on the loaded implant. *(From Hoshaw SJ, Brunski JB, Cochran GVB: Mechanical loading of Brånemark fixtures affects interfacial bone modeling and remodeling,* Int J Oral Maxillofac Implants *9:345-360, 1994.)*

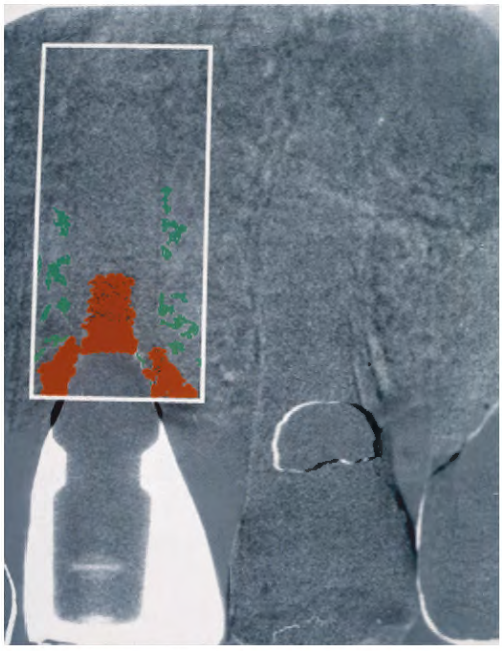


Figure 4-29 Researchers observed in humans that less crestal bone loss and an increase in bone density were present around implants progressively loaded in the maxillary first premolar region. *(From Appleton RS, Nummikoski PV, Pigno MA et al: A radiographic assessment of progressive loading on bone around single osseointegrated implants in the posterior maxilla,* Clin Oral Implants Res *16:161-167, 2005.)*

in the same patient on the contralateral side[100,101] (Figure 4-29). Marginal bone loss is less in the mandible compared with the maxilla in several clinical reports. The bone is denser in the mandible than the maxilla. The reduced crestal bone loss that has been reported in the mandible, in greater bone densities, and in progressively loaded implants point to the fact that stress/strain is a primary etiology of crestal bone loss after the implant is loaded. Therefore the stresses at the crest of the ridge may cause microfracture or overload during the first year, and the change in bone strength after loading and mineralization is complete alters the stress/strain relationship and reduces the risk of microfracture during the following years.[127]

Implant Design Biomechanics

Implant design may affect the magnitude or type of forces applied to the bone-implant interface. A smooth collar at the crest module may transmit shear forces to the bone. Bone is strongest under compressive forces, 30% weaker under tensile loads, and 65% weaker to shear forces.[128] Bone may heal to the smooth metal collar of the implant crest module from the time of implant insertion to implant uncovery; but placed under loading conditions, the weaker shear interface is more likely to overload the bone. The first thread

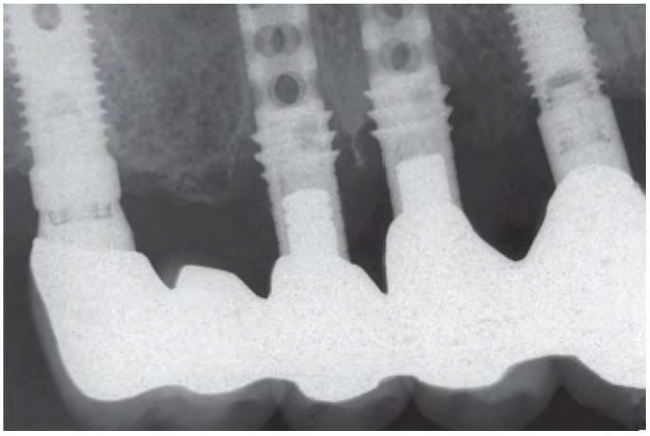


Figure 4-30 This prosthesis is supported by four implants. The center two implants with a long smooth crest module have 5 mm of bone loss. The anterior implants with a moderate smooth crest module have 3 mm of bone loss; the distal implant with the smallest smooth crest module has the least bone loss. The crestal bone loss is related in part to implant design.

or a roughened surface condition of the implant is where the type of force changes from primarily shear to compressive or tensile loads. Therefore, in many situations the 35% to 65% increase in bone strength, through changes from shear to compressive and/or tensile loads, is sufficient to halt the bone loss process. This may be one of the reasons why implant designs with a 2-mm smooth collar above the first thread and a 4-mm smooth collar above the first thread lose bone to this "first thread" landmark[129] (Figure 4-30). A previous

WWW.HIGHDENT.IR

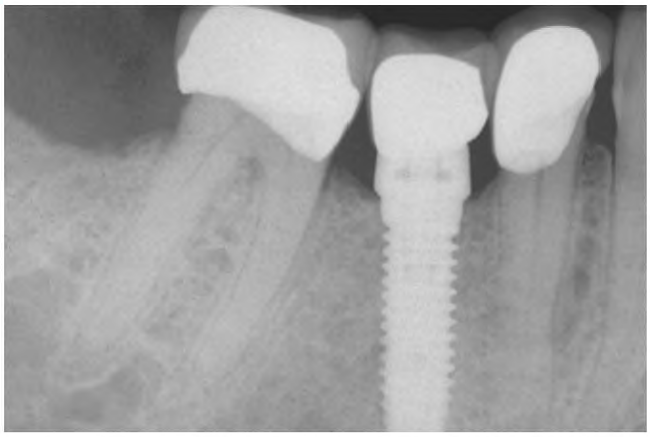


Figure 4-31 Crestal bone loss during the first year varies and is related to bone density and the amount of occlusal stresses. Some implant designs lose less bone than others. The BioHorizons Maestro dental implant is shown with no radiographic bone loss after 2 years.

review addressed the range of bone loss with different implant designs.[115,116,130] Because implant crest module designs may affect the amount of bone loss, and the implant design contributes to the force transfer of the bone-implant interface, the stress-related theory for one of the etiologies of crestal bone loss is further enhanced (Figure 4-31).

Literature from cellular biomechanics, engineering principles, differences in bone loss related to bone density, animal studies and clinical reports all substantiate that occlusal overload may be an etiology of peri-implant bone loss. Literature related to orthopedic joint replacement devices clearly indicate biomechanical stress and overload contribute to bone loss at the implant interface. The increase in bone mineralization and organization during the first year, the increase in bone density at the implant interface, and the type of force changes at the first thread of the implant body all are factors that may halt the bone loss phenomenon after the initial marginal loss. Although this occlusal overload concept does not negate other factors related to marginal bone loss, it is more clinician dependent than most other parameters. Treatment plans that emphasize occlusal stress reduction to the prosthesis are therefore mandated.

EFFECT ON TREATMENT PLANNING

Understanding the relationships of stress and related complications provides a basis for a consistent treatment system. A general concept in engineering is to determine the cause of a complication and develop a method to reduce the conditions that cause the problem. The clinical success and longevity of endosteal dental implants as load-bearing abutments are controlled largely by the biomechanical milieu in which they function.[129,131] The Stress Treatment Theorem, developed by the author, states that most all treatment related to the science of implant dentistry should be centered around the biomechanical aspects of stress.[131]

Stress-related conditions that affect the treatment planning in implant dentistry include the bone volume lost after tooth loss, bone quality decrease after tooth loss, complications of surgery, implant positioning, initial implant interface healing, initial loading of an implant, implant design, occlusal concepts, prosthesis fixation, marginal bone loss, implant failure, component fracture, prosthesis fracture, and implant fracture. Biomechanical parameters are excellent predictors of increased risks because they are objective and can be measured. One can not only predict which condition presents greater stress, and therefore greater risk, but also how much the risk is increased. A risk factor is not an absolute contraindication, but it significantly increases the complication rate. With so many variables, success or failure in implant dentistry is often a complex subject and not necessarily an exact science. But this does not mean a method cannot be established to decrease the risk. For example, smoking is a risk factor for longevity. A long-term smoker has a 75% less chance of living past 75 years of age. This does not mean a long-term smoker always dies before the average lifespan of a nonsmoker—smoking is not a poison that kills immediately. Several years ago, the oldest person on earth, who was 116 years old, was a smoker. However, this does not negate the fact that smoking is a considerable risk factor for longevity. Likewise, force factors are a considerable risk factor. Greater forces in one aspect of treatment do not always equal implant failure or complications, especially as so many factors are involved, including the density of bone around the implant. Yet the risks may be considerably reduced by decreasing the overall stress to the overall system. To assess the increase in risk factors, each factor is considered separately. The goal is to decrease the overall risk.

Understanding the relationships of stress and related complications provides a basis for a consistent treatment system. The Stress Treatment Theorem has evolved into a particular sequence of treatment planning (Box 4-3).

Box 4-3 Stress Treatment Theorem Sequence of Treatment Planning

1. Prosthesis design
2. Patient force factors
3. Bone density in implant sites
4. Key implant positions and number
5. Implant size
6. Available bone
7. Implant design

WWW.HIGHDENT.IR

Prosthesis Design

Partially and completely edentulous patients want teeth, not implants. The final result (the prosthesis) should be visualized prior to the selection of the foundation (the implants). The design of the prosthesis is related to a number of factors. Nearly all treatments for partially edentulous patients are planned as a fixed restoration. The fixed restoration must consider the soft tissue and whether it requires a surgical or prosthetic replacement approach.

Completely edentulous patients may be treated with either a removable overdenture or a fixed prosthesis. In either approach, the loss of bone from tooth loss and methods to reduce future bone loss is part of the treatment plan. Most all patients should eventually have a completely implant-supported restoration (see Chapter 5).

Patient Force Factors

An implant team should evaluate more than 60 items before developing a treatment plan. Of all these conditions, the factors that influence the amount of patient stress may influence treatment more than several other factors combined.[132,133] Because stress equals force divided by the area to which the forces are applied, the amount of force is directly related to the amount of stress. There are several force factors to consider, including: (1) bruxism, (2) clenching, (3) tongue thrust, (4) crown height, (5) masticatory dynamics, and (6) the opposing arch. The forces applied to the restoration also differ by their (1) magnitude, (2) duration, (3) type, and (4) predisposing factors (e.g., cantilevers).

Some patient force factors are more important than others. For example, severe bruxism is the most significant factor and, on a risk scale from 1 to 10, is a 10.[132] Forces from bruxism are often the most difficult forces to contend with on a long-term basis.[132,133] As a result of this condition, marginal implant bone loss, unretained abutments, and fatigue fractures of implants or prostheses are more likely. The increase in force magnitude and duration is a significant problem. A bruxing patient is at higher risk in two ways. The magnitude of the force increases because the muscles become stronger and the number of cycles on the prosthetic components is greater as a result of the parafunction. Eventually "something" will break if the occlusal disease cannot be reduced in intensity or duration. No long-term prosthetic result without complications can be expected in patients with severe bruxism.

The second highest risk factor is severe clenching, which is a 9 on the risk scale. Cantilevers, including crown height, are next on the list, followed by masticatory muscle dynamics. The position of the implant in the arch is followed by the direction of load, with a risk of 5. These numbers are arbitrary, as they are influenced by the other force factors. For example, angled forces greater than 30 degrees to the implant body are more damaging than a crown height of 20 mm with a long axis load.[134-136] The clinician should evaluate the number of force conditions and their influencing severity factors. As the overall number increases, the risks increase, and the overall treatment plan should be modified to decrease the increased force or by increasing the area of support (see Chapter 6).

Bone Density

The density of bone is directly related to the strength of the bone.[99,137] Misch et al. have reported on the biomechanical properties of four different densities of bone in the jaws.[99] Dense cortical bone is 10 times stronger than the soft, fine trabecular bone. D2 bone is approximately 50% stronger than D3 bone. In addition, the stiffness of the bone is affected by the bone density. Young's modulus for compact bone is 10 times larger than cancellous bone. The denser the bone, the stiffer the bone, and the less biomechanical mismatch to titanium during loading.

Progressive bone loading changes the amount and density of the implant-bone contact. The bone is given time to respond to a gradual increase in occlusal load. This increases the quantity of bone at the implant interface, improves the bone density, and improves the overall support system mechanism.

Key Implant Positions and Implant/Abutment Number

Key Implant Positions

In any prostheses, there are implant positions that are more important from a stress management perspective. In one- or two-unit prostheses, an implant should be placed in each prospective tooth position, without a cantilever crown contour in any direction (e.g., facial, lingual, mesial, or distal). In a three- to four-unit restoration, the most important abutments are the terminal abutments. If a terminal abutment is not present, a cantilever is created, which magnifies the stress to the rest of the support system. Cantilevers are a force magnifier and represent a considerable risk factor in implant support, screw loosening, crestal bone loss, fracture, and any other item negatively affected by force.[134,136,138] Therefore position should aim at the elimination of cantilevers whenever possible, especially when other force factors are increased.

In a 5- to 14-unit prosthesis, intermediary abutments are also important in order to limit the edentulous spans to less than three pontics. A three-pontic prosthesis flexes 18 times more than a two-pontic prosthesis, whereas a two-pontic restoration flexes eight times more than a one-pontic prosthesis. It is suggested that multiple missing adjacent teeth be replaced in a staggered position (tripod effect),[139,140] or a larger intermediary implant be inserted.

WWW.HIGHDENT.IR

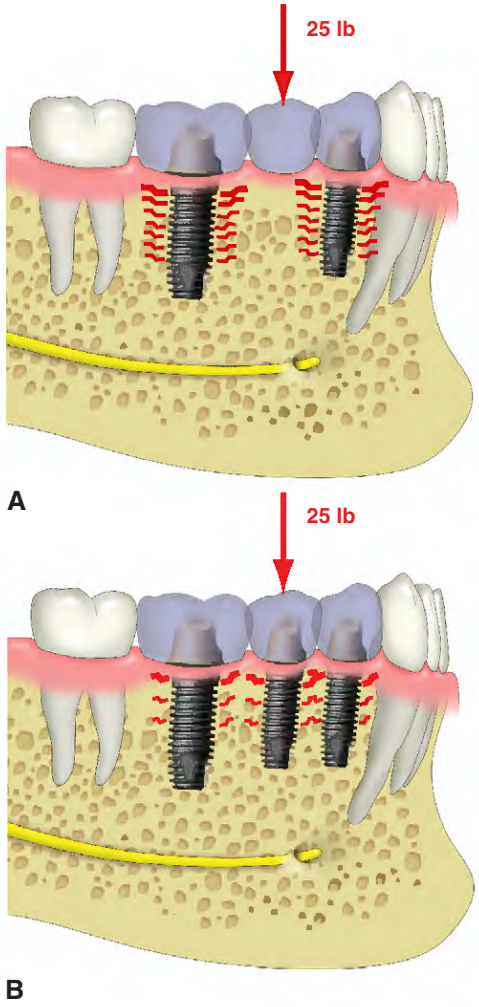


Figure 4-32 Increasing the implant number is an effective method to decrease the stress to each component in the system. Two implants supporting a three-unit prosthesis **(A)** have more stress to the implant interface than three implants supporting a similar restoration **(B)**. The additional implant also decreases the pontic number and increases the retention of the prosthesis.

The canine is an important implant position whenever the canine and two adjacent teeth are missing. Therefore, when the two premolars, a first premolar and lateral, or a lateral and a central, are missing next to a canine, a canine implant is warranted.

An edentulous mandible may be divided into three sections from a biomechanical perspective: the anterior (canine to canine) and the bilateral posterior regions (premolar and molars). A key implant position is one implant in each region, or at least three key implants.

An edentulous maxilla is divided in five regions: the anterior region (laterals and centrals), bilateral canines, and the bilateral posterior (premolar and molars). A key implant position is one implant in each region, or at least five key implants.

Treatment plans should incorporate methods to reduce stress and minimize its initial and long-term complications. Several parameters are in the doctor's control to improve the environment of the transosteal region to manage stress around and within endosteal implants. The definition of *stress* is force divided by the functional area over which it is applied. One biomechanical approach to decrease stress is to increase the surface area of the implant support system[134] (see Chapter 8).

The overall stress to the implant system may be reduced by increasing the area over which the force is applied. The most effective method to increase the surface area of implant support is by increasing the number of implants used to support a prosthesis (Figure 4-32). For example, previous studies by Bidez and Misch demonstrated that force distributed over three abutments results in less localized stress to crestal bone than two abutments.[140] This study applies only to implants that are splinted together. Therefore the number of pontics should be reduced and the number of implant abutments should be increased whenever forces are increased, compared with a treatment plan for an ideal patient with minimal force factors.[136,138,139,141,142]

The retention of the prosthesis is also improved with a greater number of splinted abutments. This approach also decreases the incidence of unretained restorations. Splinted implants also decrease porcelain fracture. The overall amount of stress to the system is reduced, and the marginal ridges on the implant crowns are supported by the connectors of the splinted crowns, with resulting compressive forces rather than shear loads on the porcelain.

Common clinical sense indicates that it is better to err with one extra implant than to err with too few. One implant too few, and the whole treatment may fail. One implant too many, and rarely is a problem noticed. Placing one implant for each buccal root is rarely indicated, regardless of the total number of risk factors. However, one implant for each tooth missing may be indicated in the posterior regions of the mouth for a large, young, male patient with severe parafunction. Rarely are more than 10 implants required in the completely edentulous arch, and more rarely are fewer than five implants suggested.

Implant Size

An excessive implant length is not critical at the crestal bone interface, but rather for initial stability and the overall amount of bone-implant interface. The increased length also provides resistance to torque or shear forces when abutments are screwed into place. However, the extra length does little to decrease the stress that occurs at the transosteal region around the implant at the crest of the ridge during occlusal loading.[143-145] Excessive implant length is not as effective a method to decrease stress from force factors.

On the other hand, with improper biomechanical management, shorter implants may have higher failure rates after loading.[146,147] Therefore, the initial treatment plan should use implants at least 12 mm in length. Ideally, softer bone types require longer implants than denser bone. The surface area of each implant is directly

WWW.HIGHDENT.IR

related to the width of the implant. Wider root form implants have a greater area of bone contact than narrow implants (of similar design), resulting from their increased circumferential bone contact areas. Each 0.25-mm increase in implant diameter may increase the overall surface area approximately 5% to 10% in a cylinder implant body. Bone augmentation in width may be indicated to increase implant diameter by 1 mm when force factors are greater than ideal. In addition, it has been suggested that an increase in implant diameter may be more effective than implant staggering to reduce stress.[148,149]

It is interesting to note that the natural teeth are narrower in the anterior regions of the mouth, where the amount of force generated is less. The natural teeth increase in diameter in the premolar region and again in the molar region as the amount of force increases, with a total 300% surface area increase from the lower anterior teeth to the maxillary molars. The length of natural teeth roots do not increase from anterior to posterior regions of the arch, but their cross section does. The supplemental implant support gained from the greater diameter not only decreases stress, but also decreases the likelihood of implant fracture and reduces the force to the abutment screw, which results in less screw loosening (see Chapter 9).

Available Bone

Once the previous steps to the treatment plan sequence have been determined, the available bone in the potential implant sites is evaluated. If adequate bone is present to position the preselected implant number, size, and design, the treatment sequence proceeds to the next factor. If available bone is not present, bone augmentation or modification is required. If these options are not possible, the sequence of treatment is begun again, starting from the prosthesis design.

In the past, the available bone was the first condition evaluated and the treatment would proceed, based upon the number and position of implants, with little regard to size or design. This approach often led to the high complication rates related to increased stress conditions (see Chapter 10).

Implant Design

Implant macrodesign may affect surface area even more than an increase in width. A cylinder (bullet-shaped) implant provides 30% less surface area than a conventional threaded implant of the same size. Strong et al. have identified 11 different variables that affect the overall functional surface area of an implant.[150] A threaded implant with 10 threads for 10 mm has more surface area than one with five threads. A thread depth of 0.2 mm has less surface area than an implant with 0.4 mm. Therefore, implant design may be the easiest method to increase surface area significantly and decrease overall risk to the implant interface (see Chapter 11).

In addition to the stress theorem and relative sequence of treatment plan, corollaries have been developed to facilitate the selection of the most appropriate therapy (Box 4-4).

SUMMARY

An understanding of the etiology of the most common implant complications has led to the development of a stress-based treatment plan theorem. Once the implant dentist has identified the sources of forces on the implant system, the treatment plan can be designed to minimize their negative impact on the implant, bone, and final restoration. Under these conditions, a

Box 4-4 Stress Treatment Theorem Corollaries

A. Patients want teeth, not implants.
B. If you had (1) ideal bone conditions, (2) optimal patient economic conditions, (3) optimal training, and (4) no time limitation, what would the treatment plan be?
C. Do not compromise a 30-year prosthesis for a 3- to 6-month delay in treatment.
D. Understand the difference between *can* versus *should* (possible versus predictable).
E. When in doubt, overengineer the foundation (e.g., add in implant, increase the design area).
F. When two or more approaches may obtain a similar result, use the least invasive and least complicated to achieve a predictable result.
G. When two or more methods may obtain a similar result, use the least expensive yet predictable method when developing the treatment plan.
H. When two or more approaches may obtain a similar result, use the most predictable rather than the fastest method.
I. Time is a factor in treatment only when all other factors are equal.
J. The patient with economic limitations cannot afford complications.
K. Apply primarily compressive loads when possible at the following levels:
   1. Porcelain
   2. Cement
   3. Abutment
   4. Screw
   5. Implant body
   6. Implant-bone interface
L. If you do not understand biomechanical stress, it will lead to psychological stress.
M. It is often necessary to modify the mouth or modify the mind of the patient.
N. It is better to see the back of the patient's head once than it is to see their face over and over again.

WWW.HIGHDENT.IR

consistent solution is an increase in implant-bone surface area. Additional implants are the solution of choice to decrease stress, along with an increase in implant width or height and the use of more implants to decrease the number of pontics and dissipate stresses more effectively to the bone structure, especially at the crest. The retention of the final prosthesis or superstructure is further improved with additional implant abutments. The amount of bone in contact with the implant is increased as a multiple of the number of implants.

References

1. Rams TE, Roberts TW, Tatum H Jr et al: The subgingival microflora associated with human dental implants, *J Prosthet Dent* 5:529-539, 1984.
2. MacDonald JB: The etiology of periodontal disease. Bacteria as part of a complex etiology, *Dent Clin North Am* 11:699-703, 1960.
3. Waerhaug J: Subgingival plaque and loss of attachment in periodontosis as evaluated on extracted teeth, *J Periodontol* 48:125-130, 1977.
4. Priest GF: Failure rates of restorations for single tooth replacements, *Int J Prosthodont* 9:38-45, 1996.
5. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
6. Creugers NH, Kayser HF, Van't Hof MA: A meta analysis of durability data on conventional fixed bridges, *Community Dent Oral Epidermiol* 22:448-452, 1994.
7. Goodacre CJ, Bernal G, Rungcharassaeng K: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
8. Oh T-J, Yoon J, Misch CE et al: The cause of early implant bone loss: Myth or science? *J Periodontol* 73:322-333, 2002.
9. Jividen G, Misch CE: Reverse torque testing and early loading failures—help or hindrance, *J Oral Implantology* 26:82-90, 2000.
10. Jemt T, Lekholm U, Adell R: Osseointegrated implants in the treatment of partially edentulous patients: a preliminary study on 876 consecutively placed fixtures, *Int J Oral Maxillofac Implants* 4:211-217, 1989.
11. Gunne J, Jemt T, Linden B: Implant treatment in partially edentulous patients: a report on prostheses after 3 years, *Int J Prosthodont* 7:143-148, 1994.
12. Lekholm U, Adell R, Lindhe J et al: Marginal tissue reactions at osseointegrated titanium fixtures. II. A cross sectional retrospective study, *Int J Oral Maxillofac Surg* 15:53-61, 1986.
13. Jaffin R, Berman C: The excessive loss of Brånemark fixtures in type IV bone: a 5 year analysis, *J Periodontol* 62:2-4, 1991.
14. van Steenberghe D, Lekholm U, Bolender C: The applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study on 558 fixtures, *Int J Oral Maxillofac Impl* 5:272-281, 1990.
15. Esposito M, Hirsch J-M, Lekholm U et al: Biological factors contributing to failures of osseointegrated oral implants. II. Etiopathogenesis, *Eur J Oral Sci* 106:721-764, 1998.
16. Lang NP, Wilson TG, Corbet EF: Biological complications with dental implants: their prevention, diagnosis and treatment. *Clin Oral Implants Res* 11(Suppl):146-155, 2000.
17. Tonetti MS, Schmid J: Pathogenesis of implant failures, *Periodontology 2000* 4:127-138, 1994.
18. Heitz-Mayfield LJ, Schmid B, Weigel C et al: Does excessive occlusal load affect osseointegration? An experimental study in the dog, *Clin Oral Impl Res* 15: 259-268, 2004.
19. Johns RB, Jemt T, Heath MR et al: A multicenter study of overdentures supported by Brånemark implants, *Int J Oral Maxillofac Implants* 4:187-194, 1992.
20. Bragger U, Aeschlimann S, Burgin W et al: Biological and technical complications and failures with fixed partial dentures (FPD) on implants and teeth after four to five years of function, *Clin Oral Implants Res* 12:26-34, 2001.
21. Eriksson RA, Albrektsson T: The effect of heat on bone regeneration: an experimental study in the rabbit using the bone growth chamber, *J Oral Maxillofac Surg* 42: 705-711, 1984.
22. Brunski JB, Moccia AF Jr, Pollack SR et al: The influence of functional use of endosseous implants on the tissue implant interface. II. Clinical aspects, *J Dent Res* 58: 1970-1980, 1979.
23. Adell R, Lekholm U, Rockler B et al: A 15 year study of osseointegrated implants in the treatment of the edentulous jaw. *Int J Oral Surg* 10:387-416, 1981.
24. Albrektsson T, Zarb GA, Worthington P et al: The long-term efficacy of currently used dental implants: a review and proposed criteria of success, *Int J Oral Maxillofac Impl* 1:1-25, 1986.
25. Schroeder A, Mawglen G, Sutter F: Hohlzylinderimplantat: Typ-F zur Prothesen-retention bei zahnlosen Kafer, *SSO Schweiz Monatsschr Zahnheilk* 93:720-733, 1983.
26. Smedberg JL, Lothigius E, Bodin I et al: A clinical and radiological two-year follow-up study of maxillary overdentures on osseointegrated implants, *Clin Oral Implants Res* 4:39-46, 1993.
27. Kline R, Hoar J, Beck GH et al: A prospective multicenter clinical investigation of a bone quality-based dental implant system, *Implant Dent* 11:1-8, 2002.
28. Isidor F: Loss of osseointegration caused by occlusal load of oral implants: a clinical and radiographic study in monkeys, *Clin Oral Implants Res* 7:143-152, 1996.
29. Isidor F: Histological evaluation of peri-implant bone at implant subjected to occlusal overload or plaque accumulation, *Clin Oral Implant Res* 8:1-9,1997.
30. Bidez MW, Misch CE: Forces transfer in implant dentistry: basic concepts and principles, *J Oral Implantol* 18:264-274, 1992.
31. Quirynen M, Naert I, van Steenberghe D: Fixture design and overload influence on marginal bone loss and fixture success in the Brånemark implant system, *Clin Oral Implants Res* 3:104-111, 1992.
32. Van Steenberghe D, Tricio J, Van den Eynde et al: Soft and hard tissue reactions towards implant design and surface characteristics and the influence of plaque and/or occlusal loads. In Davidovitch Z, editor: *The biological mechanism of tooth eruption, resorption and replacement by implants,* Boston, 1994, Harvard Society for the Advancement of Orthodontics.
33. Rangert B, Krogh PHJ, Langer B et al: Bending overload and implant fracture: a retrospective clinical analysis, *Int J Oral Maxillofac Implants* 10:326-334, 1995.
34. Snauwaert K, Duyck J, van Steenberghe D et al: Time dependent failure rate and marginal bone loss of implant supported prostheses: a 15-year follow up study, *Clin Oral Invest* 4:13-20, 2000.

WWW.HIGHDENT.IR

35. Kallus T, Bessing C: Loose gold screws frequently occur in full-arch fixed prostheses supported by osseointegrated implants after 5 years, *Int J Oral Maxillofac Implants* 9:169-78, 1991.
36. Boggan S, Strong JT, Misch CE et al: Influence of Hex geometry and prosthetic table width on static and fatigue strength of dental implants, *J Prosthet Dent* 82:436-440, 1999.
37. Linkow LI: Statistical analyses of 173 patients, *J Oral Implants* 4:540-562, 1974.
38. Adell R, Lekholm U, Rockler B et al: Marginal tissue reactions at osseointegrated titanium fixtures (1). A 3-year longitudinal prospective study, *Int J Oral Maxillofac Surg* 15:39-52, 1986.
39. Albrektsson T, Zarb GA, Worthington P et al: The long-term efficacy of currently used dental implants: a review and proposed criteria of success, *Int J Oral Maxillofac Implants* 1:11-25,1986.
40. Misch CE, Suzuki JB, Misch-Dietsh FD et al: A positive correlation between occlusal trauma and peri-implant bone loss: Literature support, *Implant Dent* 14:108-114, 2005.

40a. Glossary of Prosthodontic Terms, ed 7, *J Prosthet Dent* 81:1-141, 1999.

41. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, Dental Learning Systems Co, Inc, *Postgrad Dent* 2:3-17, 1995.
42. van Steenberghe D: A retrospective multicenter evaluation of the survival rate of osseointegrated fixtures supporting fixed partial prostheses in the treatment of partial edentulism, *J Prosthet Dent* 61:217-223, 1989.
43. Rhinelander FW: Circulation of bone, In Bourne GH, editor: *The biochemistry and physiology of bone,* New York, 1972, Academic Press.
44. Roberts WE, Smith RK, Zilberman Y et al: Osseous adaptation to continuous loading of rigid endosseous implants, *Am J Orthodont* 86:95-111, 1984.
45. Wilderman MN, Pennel BM, King K et al: Histogenesis of repair following osseous surgery, *J Periodontol* 41: 551-565, 1970.
46. Haider R, Watzek G, Plenk H: Effects of drill cooling and bone structure on IMZ implant fixation, *Int J Oral Maxillofac Impl* 8:83-91, 1993.
47. Brisman EL: The affect of speed, pressure and time on bone temperature during the drilling of implant sites, *Int J Oral Maxillofac Impl* 11:35-37, 1996.
48. Manz MC: Radiographic assessment of peri-implant vertical bone loss: DIRG Implant Report No 9, *J Oral Maxillofac Surg* 55(Suppl 5):62-71, 1997.
49. Hoar JE, Beck GH, Crawford EA et al: Prospective evaluation of crestal bone remodeling of a bone density based dental system, *Comp Cont Educ Dent* 19:17-24, 1998.
50. Glickman I, Samelos JB: Effect of excessive forces upon the pathway of gingival inflammation in humans, *J Periodontol* 36:141-147, 1965.
51. Lekholm U Ericsson I, Adell R et al: The condition of the soft tissues of tooth and fixture abutments supporting fixed bridges; a microbiological and histological study, *J Oral Clin Periodontol* 13:558-562, 1986.
52. Kent JN, Homsby CA: Pilot studies of a porous implant in dentistry and oral surgery, *J Oral Surg* 30:608, 1972.
53. Becker W, Becker BE, Newman MG et al: Clinical and microbiologic findings that may contribute to dental implant failure, *Int J Oral Maxillofac Impl* 5:31-38, 1990.
54. Koutsonikos A: Implants: success and failure—a literature review, *Ann R Australas Coll Dent Surg* 14: 75-80, 1998.
55. Gargiulo AW, Wentz FM, Orban B: Dimensions and relations of the dentogingival junction in humans, *J Periodontol* 32:261-267, 1961.
56. Vacek JS, Gher ME, Assad DA et al: The dimensions of the human dentogingival junction, *Int J Perio Rest Dent* 14:155-165, 1994.
57. Rateitschak KJ, editor: *Color atlas of dental medicine,* Stuttgart, Germany, 1989, Thieme.
58. Maynard JS, Wilson, RD: Physiologic dimensions of the periodontium significant to the restorative dentist, *J Periodontol* 50:170-174, 1979.
59. Tarnow D, Stahl S, Maner A et al: Human gingival attachment: responses to subgingival crown placement marginal remodeling, *J Clin Periodontol* 13:563-569, 1986.
60. Berglundh T, Lindhe J, Erricsson I et al: The soft tissue barrier at implants and teeth, *Clin Oral Implants Res* 2:81-90, 1991.
61. Lindhe J, Berglundh T, Ericsson I et al: Experimental breakdown of peri-implant and periodontal tissues. A study in the beagle dog, *Clin Oral Implants Res* 3:9-16, 1992.
62. Wallace S, Tarnow D: *The biologic width around implants,* International Congress Oral Implant Meeting, Munich, Germany, October 1995.
63. Wallace SS: Significance of the biologic width with respect to root form implants, *Dent Impl Update* 5:25-29, 1994.
64. James RA, Keller EE: A histopathological report on the nature of the epithelium and underlying connective tissue which surrounds oral implant, *J Biomed Mat Res* 8:373-383, 1974.
65. Gould TRL, Westbury L, Brunette DM: Ultrastructural study of the attachment of human gingival to titanium in vivo, *J Prosthet Dent* 52:418-420, 1984.
66. Hansson HA, Albrektsson T, Brånemark PI: Structural aspects of the interface between tissue and titanium implants, *J Prosthet Dent* 50:108-113, 1983.
67. McKinney RV, Steflik DE, Koth DL: Evidence for a junctional epithelial attachment to ceramic dental implants: a transmission electronmicroscopic study, *J Periodontol* 56:579-591, 1985.
68. Barboza EP, Caúla AL, Carvalho WR: Crestal bone loss around submerged and exposed unloaded dental implants: a radiographic and microbiological descriptive study, *Implant Dent* 11:162-169, 2002.
69. Weber HP, Buser D, Donath K, Fiorellini JP et al: Comparison of healed tissue adjacent to submerged and non-submerged unloaded titanium dental implants: a histometric study in beagle dogs, *Clin Oral Implants Res* 7:11-19, 1996.
70. Cochran DL, Hermann JS, Schenik RS et al: Biologic width around titanium implants: a histometric analysis of the implanto-gingival junction around unloaded and loaded nonsubmerged implants in the canine mandible, *J Periodontol* 68:186-198, 1997.
71. Abrahamsson I, Berglundh T, Wennstrom J et al: The peri-implant hard and soft tissue characteristics at different implant systems: a comparative study in dogs, *Clin Oral Implants Res* 7:212-219, 1996.
72. Hermann JS, Buser D, Schenk RK et al: Crestal bone changes around titanium implants: a histometric evaluation of unloaded non-submerged and submerged

WWW.HIGHDENT.IR

implants in the canine mandible, *J Periodontol* 71:1412-1424, 2000.
73. Jung YC, Han CH, Lee KW: A 1-year radiographic evaluation of marginal bone around dental implants, *Int J Oral Maxillofac Implants* 11:811-818, 1996.
74. Wiskott HW, Belser UC: Lack of integration of smooth titanium surfaces: a working hypothesis based on strains generated in the surrounding bone, *Clin Oral Implants Res* 10:429-444, 1999.
75. Rosenberg ES, Torosian JP, Slots J: Microbial differences in 2 clinically distinct types of failures of osseointegrated implants, *Clin Oral Implants Res* 2:135-144. 1991.
76. Karolyi M: Beobachtungen über Pyorrhea alveolaris, *Osterenorichisch-Ungarische viertel jahresschrift fur Zahnheilkunde* 17:279, 1991.
77. Glickman I: Clinical significance of trauma from occlusion, *J Am Dent Assoc* 70:607-618, 1965.
78. Macapanpan LC, Weinmann JP: The influence of injury to the periodontal membrane on the spread of gingival inflammation, *J Dent Res* 33:263-272, 1954.
79. Posselt U, Emslie RD: Occlusal disharmonies and their effect on periodontal diseases, *Internat Dent J* 9:367-381, 1959.
80. Fleszar TJ, Knowles JW, Monson EC et al: Tooth mobility and periodontal therapy, *J Clin Periodont* 7:495-505, 1980.
81. Bergett F, Ramfjord S, Nissle R et al: A randomized trial of occlusal adjustment in the treatment of periodontitis patients, *J Clin Periodontol* 19:381-387, 1992.
82. Belting CM, Gripta OP: The influence of psychiatric disturbances on the severity of periodontal diseases, *J Periodont* 32:219-226, 1961.
83. Waerhaug J: The infrabony pocket and its relationship to trauma from occlusion and subgingival plaque, *J Periodontol* 50:355-365, 1979.
84. Lordahl A, Scher O, Waerhaug J et al: Tooth mobility and alveolar bone resorption as a function of occlusal stress and oral hygiene, *Acta Odontol Scand* 17:61-77, 1959.
85. Baer P, Kakehashi S, Littleton NW et al: Alveolar bone loss and occlusal wear, *J Am Soc Periodontics* 11-98, 1963.
86. Lindhe J, Nyman S, Ericsson I: Trauma from occlusion. In Lindhe J, editor : *Clinical periodontology and implant dentistry*, ed 4, Oxford, 2003, Blackwell.
87. Cowin SC, Hegedus DA: Bone remodeling I: theory of adaptive elasticity, *J Elasticity* 6:313-326, 1976.
88. Bidez MW, Misch CE: Force transfer in implant dentistry: basic concepts and principles, *Oral Implantol* 18:264-274, 1992.
89. Cowin SC, Moss-Salentign L, Moss ML: Candidates for the mechanosensory system in bone, *J Biomechan Engineer* 113:191-197, 1991.
90. Kummer BKF: Biomechanics of bone: Mechanical properties, functional structure, functional adaptation. In Fung YC, Perrone H, Anliker M, editors: *Biomechanics: foundations and objectives*, Englewood Cliffs, 1972, Prentice-Hall.
91. Frost HM: Bone "mass" and the "mechanostat": a proposal, *Anat Rec* 219:1-9, 1987.
92. Frost HM: Bone's Mechanostat: a 2003 Update, *The Anatomical Record Part A* 275A:1081-1101, 2003.
93. Chiba J, Rubash JE, Kim KJ et al: The characterization of cytokines in the interface tissue obtained from failed cementless total hip arthroplasty with and without femoral osteolysis *Clin Orthop* 300:304-312, 1994.
94. Baumeister T, Avallone EA: *Marks' standard handbook of mechanical engineers,* ed 8, New York, 1978, McGraw-Hill.
95. Bidez M, McLoughlin S, Lemons JE: FEA investigations in plate-form dental implant design. In *Proceedings of the First World Congress of Biomechanics*, San Diego, Calif, 1990.
96. Misch CE: *A three dimensional finite element analysis of two blade implant neck designs* [master's thesis], Pittsburgh, 1989, University of Pittsburgh.
97. Bidez MW, Misch CE: Issues in bone mechanics related to oral implants, *Implant Dent* 1:289-294, 1992.
98. Kilamura E, Slegaroui R, Nomura S et al. Biomechanical aspects of marginal bone resorption around osseointegrated implants: consideration based in a three dimensional finite element analysis, *Clin Oral Implant Res* 15:401-412, 2004.
99. Misch CE, Qu M, Bidez MW: Mechanical properties of trabecular bone in the human mandible: Implications of dental implant treatment planning and surgical placement, *J Oral Maxillofac Surg* 57: 700-706, 1999.
100. Appleton RS, Nummikoski PV, Pigmo MA et al: Peri-implant bone changes in response to progressive osseous loading, *J Dent Res* 76:412, 1997 [special issue].
101. Appleton RS, Nummikoski PV, Pigno MA et al: A radiographic assessment of progressive loading on bone around single osseointegrated implants in the posterior maxilla, *Clin Oral Implants Res* 16:161-167, 2005.
102. Hoshaw S: *Investigation of bone remodeling and remodeling at a loaded bone-implant interface* [thesis], Troy, NY, 1992, Rensselaer Polytechnic Institute.
103. Hoshaw SJ, Brunski JB, Cochran GVB: Mechanical loading of Brånemark fixtures affects interfacial bone modeling and remodeling, *Int J Oral Maxillofac Implants* 9:345-360, 1994.
104. Miyata T, Kobayashi Y, Araki H et al: An experimental study of occlusal trauma to osseointegrated implants: part 2, *Jpn Soc Periodont* 39:234-241, 1997.
105. Miyata T, Kobayashi Y, Araki H, et al: The influence of controlled occlusal overload on peri-implant tissue: a histologic study in monkeys, *Int J Oral Maxillofac Implants* 13:677-683, 1998.
106. Miyata T, Kobayashi Y, Araki H et al: The influence of controlled occlusal overload on peri-implant tissue. Part 3: a histologic study in monkeys, *Int J Oral Maxillofac Implants* 15:425-431, 2000.
107. Duyck J, Ronold HJ, Oosterwyck HV et al: The influences of static and dynamic loading on marginal bone reactions around osseointegrated implants: an animal experimental study, *Clin Oral Implant Res* 12:207-218, 2001.
108. Lindquist JW, Rockler B, Carlsson GE: Bone resorption around fixtures in edentulous patients treated with mandibular fixed tissue integrated prostheses, *J Prosthet Dent* 59-63, 1988.
109. Shackleton JL, Carr L, Slabbert JC: Survival of fixed implant-supported prostheses related to cantilever lengths, *J Prosthet Dent* 71:23-26, 1994.
110. Wyatt CC, Zarb GA: Bone level changes proximal to oral implants supporting fixed partial prostheses, *Clin Oral Implants Res* 13:62-68, 2002.
111. Duyck J, Van Oosterwyck H, Van der Sloten J et al: Magnitude and distribution of occlusal forces on oral implants supporting fixed prostheses: an in vivo study, *Clin Oral Implants Res* 11:465-475, 2000.
112. Naert I, Quirynen M, Van Steenberghe D et al: A study of 589 consecutive implants supporting complete fixed prostheses. Part II: prosthetic aspects, *J Prosthet Dent* 68:949-956, 1992.

WWW.HIGHDENT.IR

113. Uribe R, Penarrocha M, Sanches JM et al: Marginal peri-implantitis due to occlusal overload: a case report, *Med Oral* 9:159-162, 2004.
114. Leung KC, Chew TW, Wat PY et al: Peri-implant bone loss: management of a patient, *Int J Oral Maxillofac Implant* 16:273-277, 2001.
115. Zechner W, Trinki N, Watzek G et al: Radiographic follow-up of peri-implant bone loss around machine-surfaced and rough-surfaced interforaminal implants in the mandible functionally loaded for 3 to 7 years, *Int J Oral Maxillofac Implants* 19:216-222, 2004.
116. Karousis IK, Brägger U, Salvi G et al: Effect of implant design on survival and success rates of titanium oral implants: a 10 year prospective cohort study of the ITI Dental Implant System, *Clin Oral Implant Res* 15: 8-17, 2004.
117. Winemaker MJ, Thorhill TS: Complications of joint replacement in the elderly. In Rosen CJ, Glovacki J, Bilezikian JP, editors: *The aging skeleton*, San Diego, 1999 Academic Press.
118. Maloney WJ, Herzwurm P, Papiosky W et al: Treatment of pelvic osteolysis associated with stable acetabular component inserted without cement as part of a total hip replacement, *J Bone Joint Surg* 79A:1628-1634, 1997.
119. Lewallen DG, Berry DJ: Periprosthetic fracture of the femur after total hip arthroplasty, *J Bone Joint Surg* 79A:1881-1890, 1997.
120. Barrack RL: Modularity of prosthetic implants, *J Am Acad Orthop Surg* 2:16-25, 1994.
121. Hornhill TS, Ozuna RM, Shortkeoff S et al: Biochemical and histologic evaluation of the synovial-like tissue around failed (loose) total joint replacement prostheses in human subject and a canine model, *Biomaterials* 11:69-72, 1990.
122. Morrey BF: Difficult complications after hip joint replacement: dislocation, *Clin Orthop Relat Res* 334: 179-187, 1997.
123. Misch CE, Bidez MW: Occlusion and crestal bone resorption etiology and treatment planning strategies for implants. In Mc Neill C, editor: *Science and practice of occlusion*, ed 1, Chicago,1997, Quintessence.
124. Roberts WE, Turley DK, Brezniak N et al: Bone physiology and metabolism, *J Calif Dent Assoc* 54:32-39, 1987.
125. Roberts WE, Garetto LP, De Castro RA: Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants with threaded or smooth surfaces: Indications for provisional loading and axially directed occlusion, *J Indiana Dent Assoc* 68:19-24, 1989.
126. Piatelli A, Ruggeri A, Franchi M, et al: An histologic and histomorphometric study of bone reactions to unloaded and loaded non-submerged single implants in monkeys: a pilot study, *J Oral Implant* 19:314-319, 1993.
127. Rotter BE, Blackwell R, Dalton G: Testing progressive loading of endosteal implants with the periotest—a pilot study, *Implant Dent* 5:28-32, 1996.
128. Reilly DT, Burstein AH: The elastic and ultimate properties of compact bone tissue, *J Biomech* 8:393-405, 1975.
129. Misch CE: Stress factors: influence on treatment planning. In Misch CE, editor: *Dental implant prosthetics*, St Louis, 2005, Elsevier.
130. Taylor TD, Belser U, Mericke-Stern RI: Prosthodontics considerations, *Clin Oral Implants Res* 11:101-107, 2000.
131. Misch CE: Consideration of bio mechanical stress in treatment with dental implants, *Dent Today* 25:80-85, 2007.
132. Misch CE: The effect of bruxism on treatment planning for dental implants, *Dent Today* 9:76-81, 2002.
133. Misch CE: Clenching and its effect on implant treatment plans, *Oral Health* 11-21, August 2002.
134. Misch CE, Bidez MW: Implant protected occlusion, a biomechanical rationale, *Compend Cont Educ Dent* 15:1330-1342, 1994.
135. Cehreli MC, Iplikcioglu H, Bilir OG: The influence of the location of load transfer on strains around implants supporting four unit cement-retained fixed prostheses: in vitro evaluation of axial versus off-set loading, *J Oral Rehabil* 29:394-400, 2002.
136. Duyck J, Naert I: Failure of oral implants—etiology, symptoms and influencing factors, *Clin Oral Investig* 2:102-114, 1998.
137. Thomsen JS, Ebbesen EN, Mosekilde L: Relationships between static histomorphometry and bone strength measurements in human iliac crest bone biopsies, *Bone* 22:153-163, 1998.
138. Sennerby L, Roos J: Surgical determinants of clinical success of osseointegrated oral implants: a review of the literature, *Int J Prosthodont* 11:408-420, 1998.
139. Bidez MW, Misch CE: Issues in bone mechanics related to oral implants, *Implant Dent* 1:289-294, 1992.
140. Bidez MW, Misch CE: The biomechanics of inter-implant spacing. In *Proceedings of the 4th International Congress of Implants and Biomaterials in Stomatology*, Charleston, SC, 1990.
141. Borchers L, Reichart P: Three dimensional stress distribution around dental implants at different stages of interface development, *J Dent Res* 62:155-159, 1994.
142. Naert I, Koutsikakis G, Duyck J et al: Biologic outcome of implant-supported restoration in the treatment of partial edentulism. Part 1: a longitudinal clinical evaluation, *Clin Oral Implants Res* 13:381-389, 2002.
143. Weinberg LA, Kruger B: An evaluation of torque on implant/prosthesis with staggered buccal and lingual offset, *Int J Oral Maxillofac Implants* 16:253, 1996.
144. Lum LB, Osier JF: Load transfer from endosteal implants to supporting bone: an analysis using statics, *J Oral Implant* 18:343-353, 1992.
145. Lum LB: A biomechanical rationale for the use of short implants, *J Oral Implant* 17:126-131, 1991.
146. Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study, *J Periodontol* 77:1340-1347, 2006.
147. Misch CE: Short dental implants: a literature review and rationale for use, *Dent Today* 26:64-68, 2005.
148. Sertgoz A, Guvener S: Finite element analysis of the effect of cantilever and implant length on stress distribution on implant supported prosthesis, *J Prosthet Dent* 75: 165-169, 1996.
149. Sato Y, Shindoi N, Hosokawa R et al: A biomechanical effect of wide implant placements and offset placements of three implants in the partially edentulous region, *J Oral Rehab* 27:15-21, 2000.
150. Strong JT, Misch CE, Bidez MW et al: Functional surface area: Threadform parameter optimization for implant body design, *Compend Cont Educ Deat* 19:19-25, 1998.

WWW.HIGHDENT.IR

Chapter 5

Prosthetic Options in Implant Dentistry

Carl E. Misch

Implant dentistry is similar to all aspects of medicine in that treatment begins with a diagnosis of the patient's condition. Many treatment options stem from the diagnostic information. Traditional dentistry provides limited treatment options for the edentulous patient. Because the dentist cannot add abutments, the restoration design is directly related to the existing oral condition. On the other hand, implant dentistry can provide a range of additional abutment locations. Bone augmentation may further modify the existing edentulous condition in both the partial and total edentulous arch and therefore also affects the final prosthetic design. As a result, a number of treatment options are available to most partially and completely edentulous patients. Therefore, once the diagnosis is complete, the implant treatment plan of choice at a particular moment is patient and problem based. Not all patients should be treated with the same restoration type or design.

Almost all man-made creations, whether art, building, or prostheses, require the end result to be visualized and precisely planned for optimal results. Blueprints indicate the finest details for buildings. The end result should be clearly identified before the project begins, yet implant dentists often forget this simple but fundamental axiom. Historically in implant dentistry, bone available for implant insertion dictated the number and locations of dental implants. The prosthesis then was often determined after the position and number of implants were selected.

The goals of implant dentistry are to replace a patient's missing teeth to normal contour, comfort, function, esthetics, speech, and health, regardless of the previous atrophy, disease, or injury of the stomatognathic system. It is the final restoration, not the implants, that accomplish these goals. In other words, patients are missing teeth, not implants. To satisfy predictably a patient's needs and desires, the prosthesis should first be designed. In the stress treatment theorem,[1] the final restoration is first planned, similar to the architect designing a building before making the foundation. Only after this is accomplished can the abutments necessary to support the specific predetermined restoration be designed (Figure 5-1).

COMPLETELY EDENTULOUS PROSTHESIS DESIGN

The completely edentulous patient is too often treated as though cost were the primary factor in establishing a treatment plan. However, the doctor and staff should specifically ask about the patient's desires. Some patients have a strong psychologic need to have a fixed prosthesis as similar to natural teeth as possible. On the other hand, some patients do not express serious concerns whether the restoration is fixed or removable as long as specific problems are addressed. To assess the ideal final prosthetic design, the existing anatomy is evaluated *after* it has been determined whether a fixed or removable restoration is desired.

An axiom of implant treatment is to provide the most predictable, most cost-effective treatment that will satisfy the patient's anatomical needs and personal desires. In the completely edentulous patient, a removable implant-supported prosthesis offers several advantages over a fixed-implant restoration (Box 5-1).

Box 5-1 Advantages of Removable Implant-Supported Prostheses in the Completely Edentulous Patient

- Facial esthetics can be enhanced with labial flanges and denture teeth compared with customized metal or porcelain teeth. The labial contours of the removable restoration can replace lost bone width and height and support the labial soft tissues without hygienic compromise.
- The prosthesis can be removed at night to manage nocturnal parafunction.
- Fewer implants may be required.
- Less bone augmentation may be necessary before implant insertion.
- Shorter treatment if no bone augmentation is required.
- The treatment may be less expensive for the patient.
- Long-term treatment of complications is facilitated.
- Daily home care is easier.

WWW.HIGHDENT.IR
انجمن پروتزسازان و دندانپزشکان

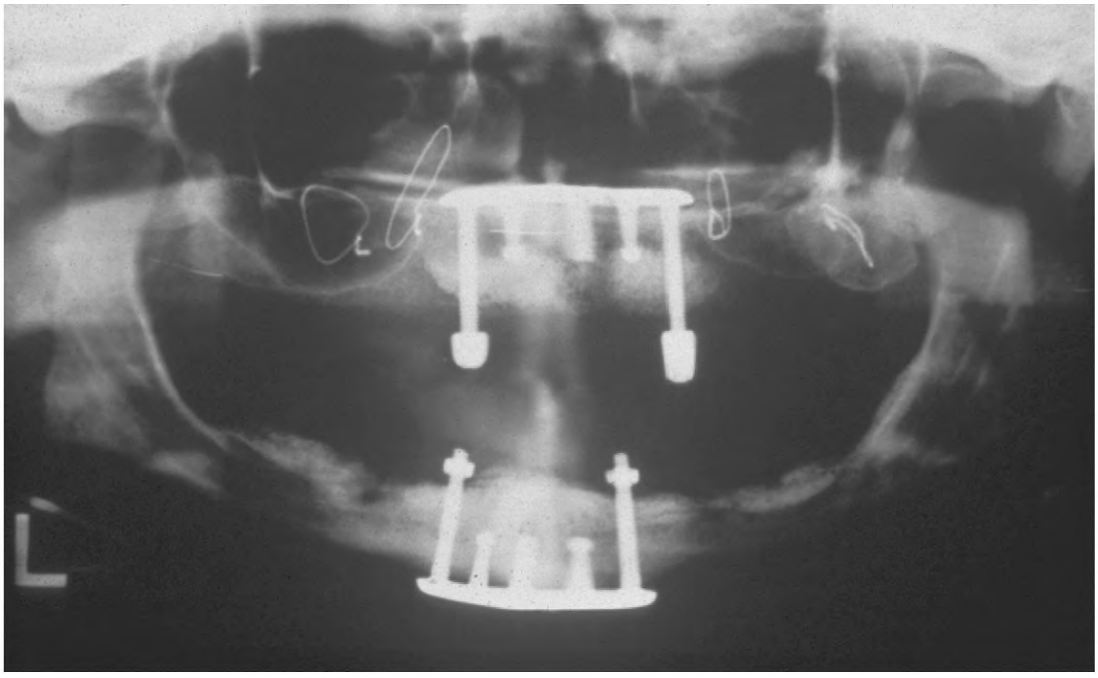


Figure 5-1 The dentist should select the implant type and position after determining a clear indication for a prosthesis. The final restoration, not the implant type and position, should be determined first. The patient wanted a fixed restoration in the maxilla and mandible. The surgeon only used one implant type (a mandibular staple implant). As a consequence, the patient was forced to accept the overdenture in the mandible with soft tissue support in the posterior region (a hydroxyapatite graft was used that increased the soft tissue trauma and risk of paresthesia in the mandible). The mandibular staple also was used in the maxilla, which required a LeFort I (downfracture) procedure. The maxillary abutments are in the middle of the palate. The consequence of the maxillary procedure was sloughing and loss of the maxilla.

However, some completely edentulous patients require a fixed restoration because of desire or because their oral condition makes the fabrication of teeth difficult if a superstructure and removable prosthesis are planned. For example, when the patient has abundant bone and implants have already been placed, the lack of crown height space may not permit a removable prosthesis.[2]

Too often, treatment plans for completely edentulous patients consist of a maxillary denture and a mandibular overdenture with two implants. However, in the long term, this treatment option may prove a disservice to the patient. The maxillary arch will continue to lose bone, and the bone loss may even be accelerated in the premaxilla.[3,4] Once this dimension is lost, the patient will have much more difficulty with retention and stability of the restoration. In addition, the lack of posterior implant support in the mandible will allow posterior bone loss to continue.[5] Paresthesia, facial changes, and reduced posterior occlusion on the maxillary prosthesis are to be expected. The doctor should diagnose the amount of bone loss and its consequences on facial esthetics, function, and the psychological and overall health. Patients should be made aware of future compromises in bone loss and its associated problems with minimal treatment options, which do not address the continued loss of bone in regions where implants are not inserted.

It is even more important to visualize the final restoration at the onset with a fixed-implant restoration. After this first important step, the individual areas of ideal or key abutment support are determined to assess whether it is possible to place the implants to support the intended prosthesis. The patient's force factors and bone density in the region of implant support are evaluated. The additional implants to support the expected forces on the prosthesis designed may then be determined with implant size and design selected to match force and area conditions. Only then is the available bone evaluated to assess whether it is possible to place the implants to support the intended prosthesis. In inadequate natural or implant abutment situations, the existing oral conditions or the needs and desires of the patient must be altered. In other words, either the mouth must be modified by augmentation to place implants in the correct anatomical positions, or the mind of the patient must be modified to accept a different prosthesis type and its limitations. A fixed-implant restoration may be indicated for either the partially or the completely edentulous patient. The psychological advantage of fixed teeth is a major benefit, and edentulous patients often feel the implant teeth are better than their own. The improvement over their removable restoration is significant.

The completely implant-supported overdenture requires the same number of implants as a fixed-implant restoration. Thus the cost of implant surgery may be similar for fixed or removable restorations. Fixed prostheses often last longer than overdentures, because

WWW.HIGHDENT.IR

Box 5-2 Advantages of Fixed Restorations in the Partially Edentulous Patient

1. Psychological (feels more like natural teeth)
2. Less food entrapment
3. Less maintenance (no attachments to change or adjust)
4. Longevity (lasts the life of the implants)
5. Similar overhead cost as completely implant-supported overdentures

Table 5-1 Prosthodontic Classification

TYPE	DEFINITION
FP-1	Fixed prosthesis; replaces only the crown; looks like a natural tooth
FP-2	Fixed prosthesis; replaces the crown and a portion of the root; crown contour appears normal in the occlusal half but is elongated or hypercontoured in the gingival half
FP-3	Fixed prosthesis; replaces missing crowns and gingival color and portion of the edentulous site; prosthesis most often uses denture teeth and acrylic gingiva, but may be porcelain to metal
RP-4	Removable prosthesis; overdenture supported completely by implant
RP-5	Removable prosthesis; overdenture supported by both soft tissue and implant

From Misch CE: Bone classification training keys, *Dent Today* 8:39-44, 1989.

attachments do not require replacement and acrylic denture teeth wear faster than porcelain to metal.[6] The chance of food entrapment under a removable overdenture is often greater than for a fixed restoration, as soft tissue extensions and support are often required in the latter. The laboratory fees for a fixed prosthesis may be similar to a bar, coping attachments, and overdenture. Because the denture or partial denture fees are much less than fixed prostheses, many clinicians charge the patient a much lower fee for removable overdentures on implants. Yet chair time and laboratory fees are often similar for fixed or removable restorations that are completely implant supported. One should consider increasing the patient fees for overdentures to a level more in line with fixed restorations.

PARTIALLY EDENTULOUS PROSTHESIS DESIGN

A common axiom in traditional prosthodontics for partial edentulism is to provide a fixed partial denture whenever applicable.[7,8] The fewer natural teeth missing, the better the indication for a fixed partial denture. This axiom also applies to implant prostheses in the partially edentulous patient. Ideally, the fixed partial denture is completely implant supported rather than joining implants to teeth. This concept leads to the use of more implants in the treatment plan. Although this may be a cost disadvantage, it is outweighed by significant intraoral health benefits. The added implants in the edentulous site result in fewer pontics, more retentive units in the restoration, and less stress to the supporting bone. As a result, complications are minimized and implant and prosthesis longevity are increased (Box 5-2).

PROSTHETIC OPTIONS

In 1989, Misch proposed five prosthetic options for implant dentistry[9,10] (Table 5-1). The first three options are fixed prostheses (FPs). These three options may replace partial (one tooth or several) or total dentitions and may be cemented or screw retained. They are used to communicate the appearance of the final prosthesis to all the implant team members. These options depend on the amount of hard and soft tissue structures replaced and the aspects of the prosthesis in the esthetic zone. Common to all fixed options is the inability of the patient to remove the prosthesis. Two types of final implant restorations are removable prostheses (RPs); they depend on the amount of implant support, not the appearance of the prosthesis.

Fixed Prostheses

FP-1

An FP-1 is a fixed restoration and appears to the patient to replace only the anatomical crowns of the missing natural teeth. To fabricate this restoration type, there must be minimal loss of hard and soft tissues. The volume and position of the residual bone must permit ideal placement of the implant in a location similar to the root of a natural tooth. The final restoration appears very similar in size and contour to most traditional fixed prostheses used to restore or replace natural crowns of teeth (Figure 5-2).

The FP-1 prosthesis is most often desired in the maxillary anterior region, especially in the esthetic zone during smiling or speaking. The final FP-1 restoration appears to the patient to be similar to a crown on a natural tooth. However, the implant abutment can rarely be treated exactly as a natural tooth prepared for a full crown. The cervical diameter of a maxillary central incisor is approximately 6.5 mm with an oval to triangular cross section. However, the implant abutment is usually 4 mm in diameter and round in cross section. In addition, the placement of the implant rarely corresponds exactly to the crown-root position of the original

WWW.HIGHDENT.IR

A B

Figure 5-2 **A,** An implant is positioned in the maxillary right canine position. The hard and soft tissue conditions are ideal for a crown of normal contour and size. **B,** The maxillary right canine implant crown in position. The soft tissue drape is similar to a natural tooth, and the crown contour is similar to the clinical crown contour of a natural tooth. This is the goal of an FP-1 prosthesis.

tooth. The thin labial bone lying over the facial aspect of a maxillary anterior root remodels after tooth loss and the crest width shifts to the palate, decreasing 40% within the first 2 years.[11] The occlusal table is also usually modified in unesthetic regions to conform to the implant size and position and to direct vertical forces to the implant body. For example, posterior mandibular implant-supported prostheses have narrower occlusal tables at the expense of the buccal contour, because the implant is smaller in diameter and placed in the central fossa region of the tooth[12] (Figure 5-3).

Because the width or height of the crestal bone is frequently lacking after the loss of multiple adjacent natural teeth, bone augmentation is often required before implant placement to achieve natural-looking crowns in the cervical region (Figure 5-4). There are no interdental papillae in edentulous ridges; therefore soft tissue augmentation also is often required to improve the interproximal gingival contour. Ignoring this step causes open "black" triangular spaces (where papillae should usually be present) when the patient smiles. FP-1 prostheses are especially difficult to achieve when more than two adjacent teeth are missing. The bone loss and lack of interdental soft tissue complicate the final esthetic result, especially in the cervical region of the crowns.

The restorative material of choice for an FP-1 prosthesis is porcelain to noble-metal alloy. A noble-metal substructure can easily be separated and soldered in case of a nonpassive fit at the metal try-in, and noble metals in contact with implants corrode less than nonprecious alloys. Any history of exudate around a subgingival base-metal margin will dramatically increase the corrosion effect between the implant and the base metal. A single-tooth FP-1 crown may use aluminum oxide cores and porcelain crowns, or ceramic abutments and porcelain crowns. However, the risk of fracture may increase with the latter scenario, as impact forces are greater on implants than natural teeth.

Figure 5-3 This full-arch FP-1 prosthesis has posterior crown contours that are narrower than natural teeth, because the implant is smaller in diameter than the tooth. As a general rule, the maxillary arch has reduced lingual contours and the mandibular posterior has reduced buccal contours.

FP-2

An FP-2 fixed prosthesis appears to restore the anatomical crown and a portion of the root of the natural tooth. The volume and topography of the available bone is more apical compared with the ideal bone position of a natural root (1 to 2 mm below the cement-enamel junction) and dictate a more apical implant placement compared with the FP-1 prosthesis. As a result, the incisal edge is in the correct position, but the gingival third of the crown is overextended, usually apical and lingual to the position of the original tooth. These restorations are similar to teeth exhibiting periodontal bone loss and gingival recession (Figure 5-5).

WWW.HIGHDENT.IR

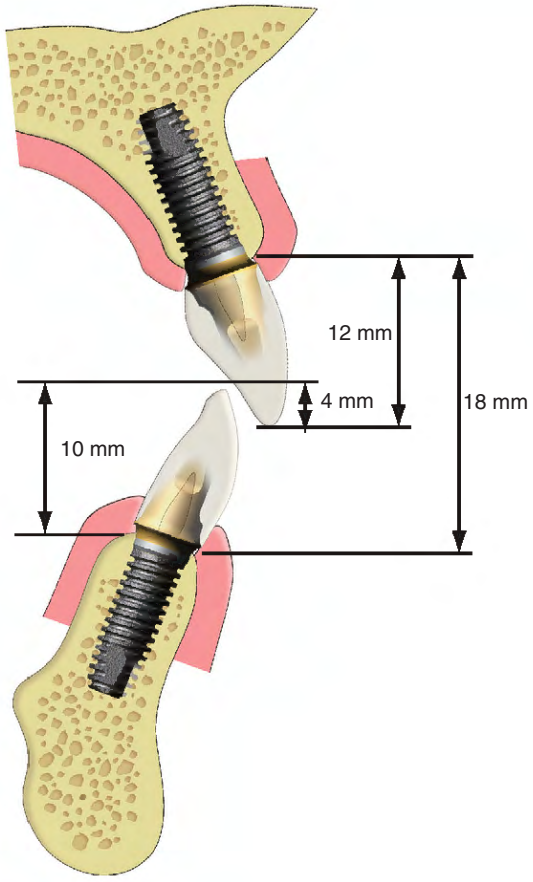


Figure 5-4 The bone and soft tissue must be ideal in volume and position to obtain an FP-1 appearance for the final restoration. When multiple teeth are replaced, bone and tissue augmentation is usually required to obtain an FP-1 prosthesis.

The patient and the clinician should be aware from the onset of treatment that the final prosthetic teeth will appear longer than healthy natural teeth (without bone loss). The esthetic zone of a patient is established during smiling in the maxillary arch and during speech of sibilant sounds for the mandibular arch (Figures 5-6, 5-7). If the high lip line during smiling or the low lip line during speech do not display the cervical regions, the longer teeth are usually of no esthetic consequence, provided that the patient has been informed before treatment (see Figure 5-5, *B*).

As the patient becomes older, the maxillary esthetic zone is altered. Only 10% of younger patients do not show any soft tissue during smiling, whereas 30% of 60 year olds and 50% of 80 year olds do not display gingival regions during smiling (Figure 5-8). The low lip position during speech is not affected as much as the high smile line. Only 10% of older patients show the mandibular soft tissue during speech.[13,14]

A multiple-unit FP-2 restoration does not require as specific an implant position in the mesial or distal position because the cervical contour is not displayed during function. The implant position may be chosen in relation to bone width, angulation, or hygienic considerations rather than purely esthetic demands (as compared with the FP-1 prosthesis). On occasion, the implant may even be placed in an embrasure between two teeth (Figure 5-9). This often occurs for mandibular anterior teeth for full-arch fixed restorations. If this occurs, the most esthetic area usually requires the incisal two thirds of the two crowns to be ideal in width, as though the implant were not present (Figure 5-10). Only the cervical region is compromised. Although the implant is not positioned in an ideal mesiodistal position, it should be placed in the correct facial-lingual position to ensure that contour, hygiene, and direction of forces are not compromised.

The material of choice for an FP-2 prosthesis is precious metal to porcelain. The amount and contour of the metal work is different than for a FP-1 restoration and is more relevant in an FP-2 prosthesis, because the

A B

Figure 5-5 **A,** An FP-2 prosthesis has longer clinical crowns than healthy natural teeth. The soft tissue drape is also reduced around the prosthesis. **B,** The high maxillary lip line during smiling is noted before fabrication of the prosthesis. When the upper lip during smile does not expose any of the interdental papillary regions, an FP-2 prosthesis may be fabricated.

WWW.HIGHDENT.IR

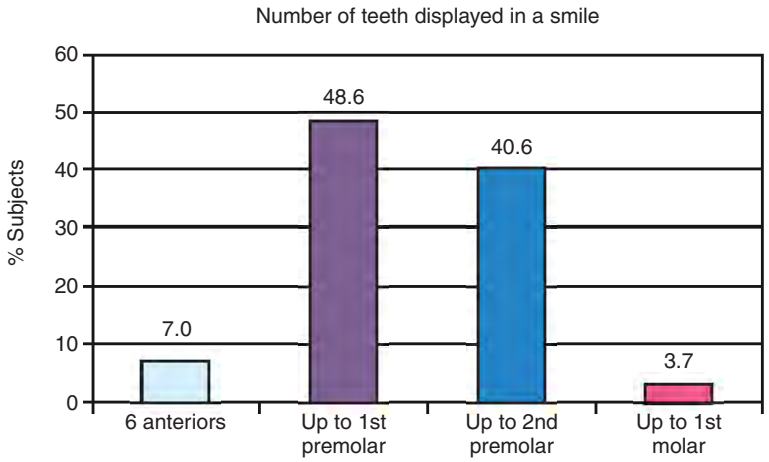


Figure 5-6 The number of teeth observed during a high smile line is variable. Almost 50% of patients display the teeth up to a first premolar. Only 3.7% of patients display the maxillary teeth to the first molar. *(Adapted from Tjan AH, Miller GD, The JG: Some esthetic factors in a smile,* J Prosthet Dent *51:24-28, 1984.)*

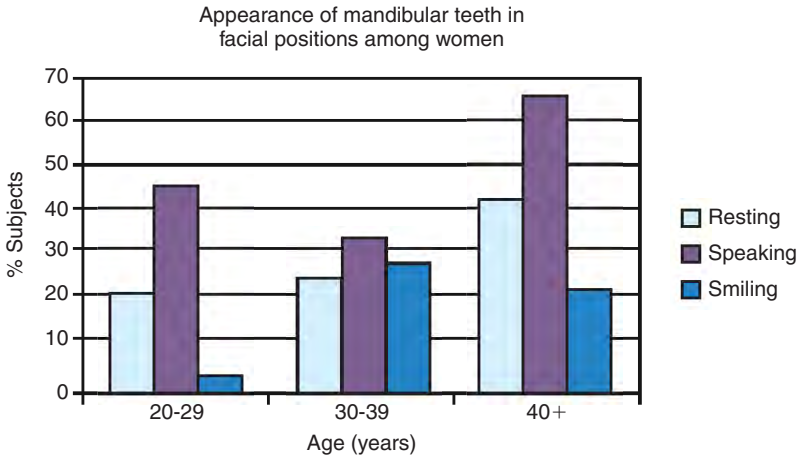


Figure 5-7 The mandibular teeth in the esthetic zone are observed during sibilant sounds, as more teeth show during speech than at rest or smiling. *(Adapted from Cade RE: The role of the mandibular anterior teeth in complete denture esthetics,* J Prosthet Dent *42:368-370, 1979.)*

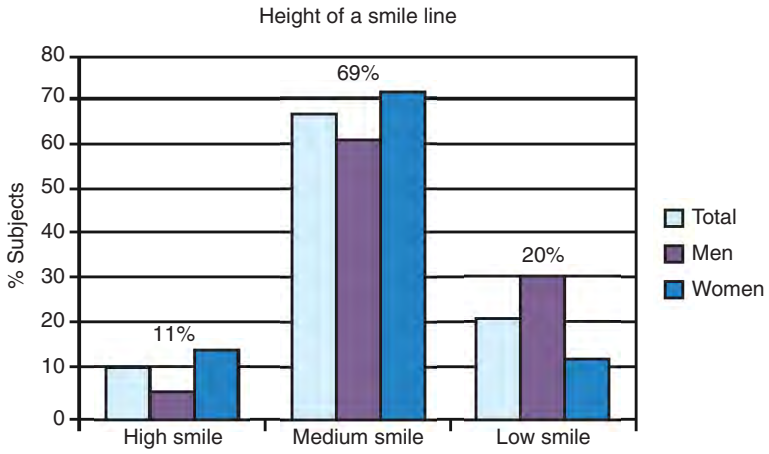


Figure 5-8 A smile that shows interdental papillae but no cervical tissue is ideal and found in 70% of patients. A low smile line shows no soft tissue during smiling and is seen in 20% of patients (more men than women). A high smile line displays interdental papillae and the cervical regions above the teeth and are observed in 11% of patients (more women than men). *(Adapted from Tjan AH, Miller GD, The JG: Some esthetic factors in a smile,* J Prosthet Dent *51:24-28, 1984.)*

amount of additional volume of tooth replacement increases the risk of unsupported porcelain in the final prosthesis, when the metal work is undercontoured.

FP-3

The FP-3 fixed restoration appears to replace the natural teeth crowns and has pink-colored restorative materials to replace a portion of the soft tissue. As with the FP-2 prosthesis, the original available bone height has decreased by natural resorption or osteoplasty at the time of implant placement. To place the incisal edge of the teeth in proper position for esthetics, function, lip support, and speech, the excessive vertical dimension to be restored requires teeth that are unnatural in length. However, unlike the FP-2 prosthesis, the patient may have a normal to high maxillary lip line during smiling

WWW.HIGHDENT.IR

A B

Figure 5-9 **A,** A full-arch maxillary implant prosthesis. Note the maxillary right anterior implant is in an embrasure. **B,** The high smile line of the same patient. The low lip position during smiling permitted the fabrication of an FP-2 prosthesis.

A B

Figure 5-10 **A,** An FP-2 complete mandibular fixed prosthesis from an occlusal view. The anterior teeth appear ideal in width and contour. **B,** The same restoration. Almost every implant is in the interproximal embrasure. The technician fabricated the restoration without regard to the mesiodistal position of the implant.

or a low mandibular lip line during speech. The ideal high smile line displays the interdental papilla of the maxillary anterior teeth but not the soft tissue above the midcervical regions. Approximately 7% of males and 14% of females have a high smile or "gummy" smile and display more than 2 mm of gingival above the free gingival margin of the teeth.[13]

The patient may also have greater esthetic demands even when the teeth are out of the esthetic smile and speech zones. Patients complain that the display of longer teeth appears unnatural even though they must lift or move their lips in unnatural positions to see the covered regions of the teeth. As a result of the restored gingival color of the FP-3, the teeth have a more natural appearance in size and shape and the pink restorative material mimics the interdental papillae and cervical emergence region. The addition of gingival-tone acrylic or porcelain for a more natural fixed prosthesis appearance is often indicated with multiple implant abutments because bone loss is common with these conditions (Figure 5-11).

There are basically two approaches for an FP-3 prosthesis: (1) a hybrid restoration of denture teeth and acrylic and metal substructure[15] or (2) a porcelain-metal restoration (Figures 5-12 to 5-14; Table 5-2). The primary factor that determines the restoration material is the amount of crown height space.[2,16] An excessive crown height space means a traditional porcelain-metal restoration will have a large amount of metal in the substructure, so the porcelain thickness will not be greater than 2-mm thick. Otherwise there is an increase in porcelain fracture. Precious metals are indicated for implant restorations to decrease the risk of corrosion

WWW.HIGHDENT.IR

A B

Figure 5-11 **A,** An intraoral view of this FP-3 restoration shows how it replaces the interdental papillae with pink porcelain. **B,** The high lip line during smiling shows the interdental papillary regions in the anterior maxilla. The fixed prosthesis should replace the gingival regions in the esthetic zone by soft tissue surgery or with the final restoration (FP-3).

Figure 5-12 An FP-3 prosthesis with pink porcelain on a porcelain-to-metal restoration.

Figure 5-13 An FP-3 prosthesis with pink porcelain on a porcelain-to-metal fixed restoration.

and improve the accuracy of the casting, as nonprecious metals shrink more during the casing process. However, the large amount of metal in the substructure acts as a heat sink and complicates the application of porcelain during the fabrication of the prosthesis. In addition, as the metal cools after casting, the thinner regions of metal cool first and create porosities in the structure. This may lead to fracture of the framework after loading. Furthermore when the casting is reinserted into the oven to bake the porcelain, the heat is maintained within the casting at different rates, thus the porcelain cool-down rate is variable, which increases the risk of porcelain fracture. In addition, the amount of precious metal in the casting adds to the weight and cost of the restoration. An FP-3 porcelain-to-metal restoration is more difficult to fabricate for the laboratory technician than an FP-2 prosthesis. The pink porcelain is harder to make appear as soft tissue and usually requires more baking cycles. This increases the risk of porosity or porcelain fracture.

An alternative to the traditional porcelain-metal fixed prosthesis is a hybrid restoration (see Table 5-2). This restoration design uses a smaller metal framework, with denture teeth and acrylic to join these elements together (Figure 5-15). This restoration is less expensive to fabricate and is highly esthetic because of the premade denture teeth and acrylic pink soft tissue replacements. In addition, the intermediary acrylic between the denture teeth and framework may reduce the impact force of dynamic occlusal loads. The hybrid prosthesis is easier to repair in porcelain fracture, as the denture tooth may be replaced with less risk than adding porcelain to a traditional porcelain-metal restoration. However, the fatigue of acrylic is greater than the traditional prosthesis; therefore repair of the restoration is more commonly needed.

The crown height space determination for a hybrid versus the traditional porcelain-metal restoration is 15 mm from the bone to the occlusal plane. When

WWW.HIGHDENT.IR

Table 5-2 Comparison of Porcelain-to-Metal versus Hybrid Prostheses (FP-3)

CONSIDERATION	PORCELAIN -METAL	HYBRID
Occlusal vertical dimension	≤15 mm	≥15 mm
Technique	Same	Same
Retention	Cement or screw	Cement or screw
Precision of fit	Same	Same
Esthetics	Same	Same
Soft tissue	Difficult	Easier
Teeth	Difficult	Easier (resin)
Time/Appointments	Same	Less
Weight	More	Less
Cost	More	Less
Impact forces	More	Less
Volume (bulk)	Same	Same
Long term	Same	Same
Occlusion	Same	Same
Speech	Same	Same
Hygiene	Same	Same
Complications	Same	Same
Aging of materials	Less	More

less than this dimension is available, a porcelain-to-metal restoration is suggested. When a greater crown height space is present, a hybrid restoration is often fabricated.

Implants placed too facial, lingual, or in embrasures are easier to restore when vertical bone has been lost and an FP-2 or FP-3 prosthesis is fabricated, because even extremely high smile lip lines do not expose the

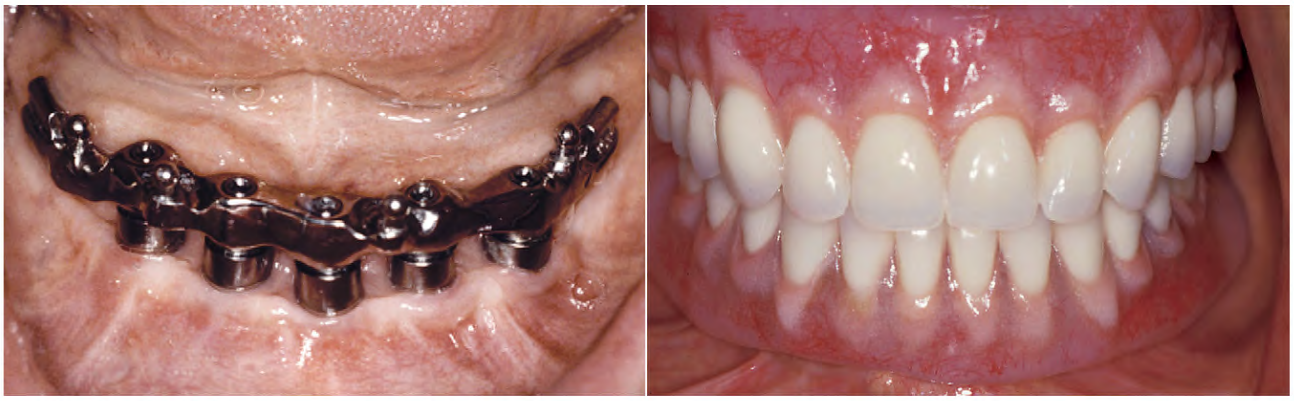
Figure 5-14 An FP-3 prosthesis with pink porcelain on a porcelain-to-metal fixed prosthesis.

A B

C

Figure 5-15 **A,** An FP-3 porcelain-to-metal restoration in the maxilla and a FP-3 hybrid acrylic-metal denture tooth in the mandible. **B,** The hybrid prosthesis in the mandibular arch. **C,** A panoramic radiograph of the same prostheses. Note the difference in the metal substructures.

WWW.HIGHDENT.IR



A B

Figure 5-16 **A,** An RP-4 restoration is a removable prosthesis (usually an overdenture) that is completely implant supported. In this patient, the mandibular restoration has five implants between the mental foramina and a cantilevered bar to the posterior regions. The prosthesis is rigid and therefore requires attention to implant position and an implant number similar to an FP-3 restoration. **B,** A maxillary denture opposing an RP-4 overdenture in the mandible.

implant abutments. The greater crown heights allow the correction of incisal edge positions. However, the FP-2 or FP-3 restoration has greater crown height compared with the FP-1 fixed types of prostheses; therefore a greater moment of force is placed on the implant cervical regions, especially during lateral forces (e.g., mandibular excursions or with cantilevered restorations). As a result, additional implant abutments or shorter cantilever lengths should be considered with these restorations.

An FP-2 or FP-3 prosthesis rarely has the patient's interdental papillae or ideal soft tissue contours around the emergence of the crowns, because these restorations are used when there is more crown height space and the lip does not expose the soft tissue regions of the patient. In the maxillary arch, wide open embrasures between the implants may cause food impaction or speech problems. These complications may be solved by using a removable soft tissue replacement device or making overcontoured cervical restorations. The maxillary FP-2 or the FP-3 prosthesis is often extended or juxtaposed to the maxillary soft tissue so that speech is not impaired. Hygiene is more difficult to control, although access next to each implant abutment is provided.

The mandibular restoration may be left above the tissue, similar to a sanitary pontic. This facilitates oral hygiene in the mandible, especially when the implant permucosal site is level with the floor of the mouth and the depth of the vestibule. However, if the space below the restoration is too great, the lower lip may lack support in the labiomental region.

Removable Prostheses

There are two kinds of removable prostheses, based upon support of the restoration (see Table 5-1). Patients are able to remove the restoration, but not the implant-supported superstructure attached to the abutments. The difference in the two categories of removable restorations is not in appearance (as it is in the fixed categories). Instead, the two removable categories are determined by the amount of implant support.[17] The most common removable implant prostheses are overdentures for completely edentulous patients. Traditional removable partial dentures with clasps on implant abutment crowns have not been reported in the literature with any frequency. No long-term or short-term studies are currently available. On the other hand, complete removable overdentures have often been reported with predictability.[6,18-22] As a result, the removable prosthetic options are primarily overdentures for the completely edentulous patient.

RP-4

RP-4 is a removable prosthesis completely supported by the implants, teeth, or both. The restoration is rigid when inserted: overdenture attachments usually connect the removable prosthesis to a low-profile tissue bar or superstructure that splints the implant abutments. Usually five or six implants in the mandible and six to eight implants in the maxilla are required to fabricate completely implant-supported RP-4 prostheses in patients with favorable dental criteria (Figure 5-16).

The implant placement criteria for an RP-4 prosthesis is different than that for a fixed prosthesis. Denture teeth more acrylic are required for the removable restoration. In addition, a superstructure and overdenture attachments must be added to the implant abutments. This requires a more lingual and apical implant placement in comparison with the implant position for a fixed prosthesis. The implants in an RP-4 prosthesis

WWW.HIGHDENT.IR

Figure 5-17 Intraoral view of three mandibular implants inserted between the foramina. A bar connects the implants and can support an RP-5 mandibular overdenture. Soft tissue support of the restoration is required in the posterior regions because the implant position and number are not conducive to a completely implant-supported prosthesis.

(and an FP-2 or FP-3 restoration) should be placed in the mesiodistal position for the best biomechanical and hygienic situation. On occasion, the position of an attachment on the superstructure or prosthesis may also affect the amount of spacing between the implants. For example, a Hader clip requires the implant spacing to be greater than 6 mm from edge to edge, and as a consequence reduces the number of implants that may be placed between the mental foramina. The RP-4 prosthesis may have the same appearance as an FP-1, FP-2, or FP-3 restoration. A porcelain-to-metal prosthesis with attachments in selected abutment crowns can be fabricated for patients with the cosmetic desire of a fixed prosthesis. The overdenture attachments permit improved oral hygiene or allow the patient to sleep without the excess forces of nocturnal bruxism on the prosthesis.

RP-5

RP-5 is a removable prosthesis combining implant and soft tissue support. The amount of implant support is variable. The completely edentulous mandibular overdenture may have: (1) two anterior implants independent of each other; (2) splinted implants in the canine region to enhance retention; (3) three splinted implants in the premolar and central incisor areas to provide lateral stability; or (4) implants splinted with a cantilevered bar to reduce soft tissue abrasions and to limit the amount of soft tissue coverage needed for prosthesis support. The primary advantage of an RP-5 restoration is the reduced cost. The prosthesis is very similar to traditional overdentures supported by natural teeth (Figure 5-17).

A preimplant treatment denture may be fabricated to ensure the patient's satisfaction. This technique is especially indicated for patients with demanding needs and desires regarding the final esthetic result. The implant dentist can also use the treatment denture as a guide for implant placement. The patient can wear the prosthesis during the healing stage. After the implants are uncovered, the superstructure is fabricated within the guidelines of the existing treatment restoration. Once this is achieved, the preimplant treatment prosthesis may be converted to the RP-4 or RP-5 restoration.

The clinician and the patient should realize that the bone will continue to resorb in the soft tissue–borne regions of the prosthesis. Relines and occlusal adjustments every few years are common maintenance requirements of an RP-5 restoration. Bone resorption with RP-5 restorations may occur two to three times faster than the resorption found with full dentures.[5] This can be a factor when considering this type of treatment in young patients, despite the lesser cost and low failure rate.

SUMMARY

In traditional dentistry, the restoration reflects the existing condition of the patient. Existing natural abutments are first evaluated, and a removable or fixed restoration is fabricated accordingly. Implant dentistry is unique because additional foundation units may be created for a desired prosthodontic result. Therefore both the psychological and anatomical needs and desires of the patient should be determined. The prosthesis that satisfies these goals and eliminates the existing problems may then be designed. The prosthesis may be fixed or removable for the completely edentulous patient, whereas fixed restorations are planned for most partially edentulous patients.

If only one implant approach is used for all patients, the same surgical and prosthetic scenarios and flaws are invariably repeated. For example, if a traditional fixed mandibular staple bone plate is used on all edentulous mandibles, not only are the implant and surgery similar regardless of intraoral or extraoral conditions, but an RP-5 prosthesis will usually result despite the patient's needs and desires.

The benefits of implant dentistry can be realized only when the prosthesis is first discussed and determined. An organized treatment approach based on the prosthesis permits predictable therapy results. Five prosthetic options are available in implant dentistry. Three restorations are fixed and vary in the amount of hard and soft tissue replaced; two are removable and are based on the amount of support for the restoration (Figures 5-18, 5-19). The amount of support required for an implant prosthesis should initially be designed similar to traditional tooth-supported restorations. Once the intended prosthesis is designed, the implants and treatment surrounding this specific result can be established.

WWW.HIGHDENT.IR
انجمن دندانسازان و دندانپزشکان

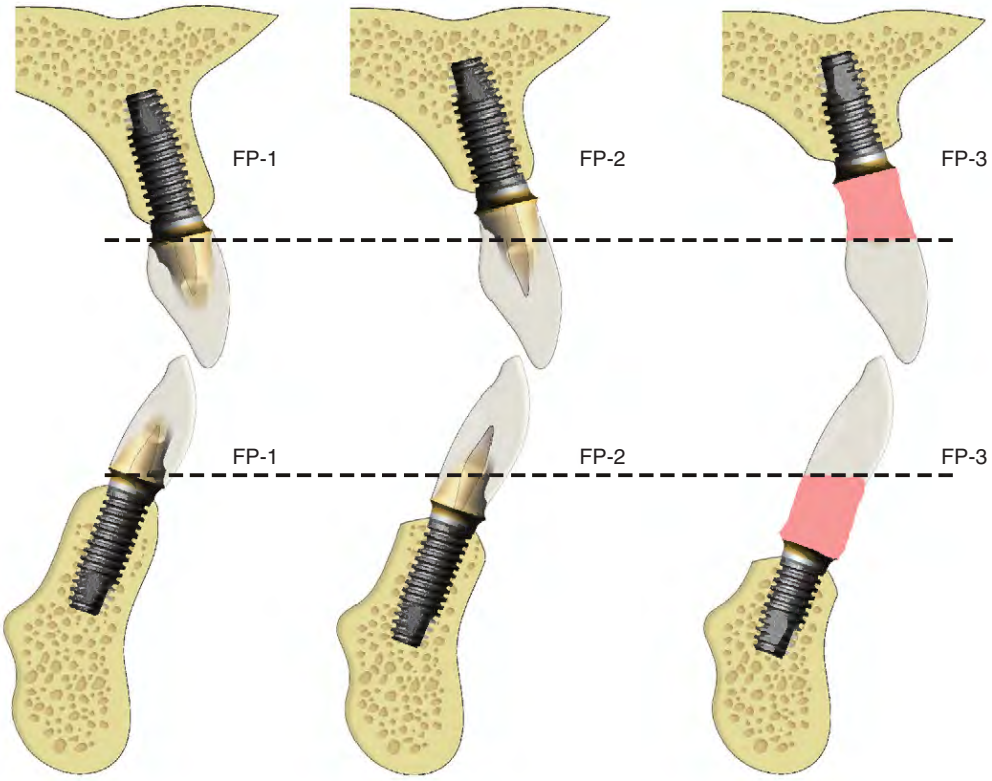


Figure 5-18 Fixed restorations have three categories: FP-1, FP-2, and FP-3. The restoration type is related to the contour of the restoration. (FP-1 is ideal, FP-2 is hypercontoured, and FP-3 replaces the gingiva drape with pink porcelain or acrylic.) The difference between FP-2 and FP-3 most often is related to the high maxillary lip position during smiling or the mandibular lip position during sibilant sounds of speech. FP-2 and FP-3 restorations often require more implant surface area support by increasing implant number or size or by adjusting design considerations.

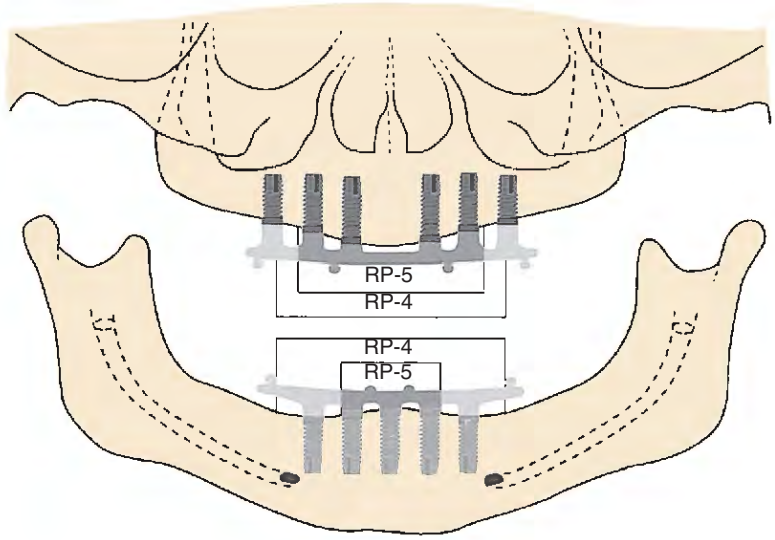


Figure 5-19 Removable restorations have two categories based on implant support. RP-4 prostheses have complete implant support anterior and posterior. In the mandible the superstructure bar often is cantilevered from implants positioned between the foramina. The maxillary RP-4 prosthesis usually has more implants and little to no cantilever. An RP-5 restoration has primarily anterior implant support and posterior soft tissue support in the maxilla or mandible. Often fewer implants are required and bone grafting is less indicated.

WWW.HIGHDENT.IR

References

1. Misch CE: Consideration of biomechanical stress in treatment with dental implants, *Dent Today* 25:80,82,84, 85; quiz 85, 2006.
2. Misch CE, Goodacre CJ, Finley M et al: Consensus Conference Panel Reports: Crown-height space guidelines for implant dentistry—part 1, *Implant Dent* 14:312-318, 2005.
3. Jacobs R, van Steenberghe D, Nys M et al: Maxillary bone resorption in patients with mandibular implant-supported overdentures: a survey, *Int J Prosthodont* 9: 58-64, 1996.
4. Barber HD, Scott RF, Maxon BB et al: Evaluation of anterior maxillary alveolar ridge resorption when opposed by the transmandibular implant, *J Oral Maxillofac Surg* 48: 1283-1287, 1990.
5. Jacobs R, Schotte A, van Steenberghe D et al: Posterior jaw bone resorption in osseointegrated implant overdentures, *Clin Oral Implants Res* 2:63-70, 1992.
6. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prosthodontics, *J Prosthet Dent* 90:121-132, 2003.
7. Dykema RW, Goodacre CJ, Phillips RW: *Johnston's modern practice on fixed prosthodontics,* ed 4, Philadelphia, 1986, WB Saunders.
8. Tylman SD, Malone WFD: *Tylman's theory and practice of fixed prosthodontics,* ed 7, St Louis, 1978, Mosby.
9. Misch CE: Bone classification, training keys, *Dent Today* 8:39-44, 1989.
10. Misch CE: Prosthetic options in implant dentistry, *Int J Oral Implantol* 7:17-21, 1991.
11. Misch CE: Early maxillary bone loss after tooth extraction: a clinical observation. In *Misch Implant Institute Manual,* Dearborn, Mich, 1991, Misch Implant Institute.
12. Misch CE: Posterior single tooth replacement. In Misch CE, editor: *Dental implant prosthetics,* St Louis, 2005, Mosby.
13. Tjan AH, Miller GD, The JG: Some esthetic factors in a smile, *J Prosthet Dent* 51:24-28, 1984.
14. Cade RE: The role of the mandibular anterior teeth in complete denture esthetics, *J Prosthet Dent* 42:368-370, 1979.
15. Brånemark PI, Zarb GA, Albrektsson T: *Tissue integrated prostheses,* Chicago, 1985, Quintessence.
16. Misch CE, Misch-Dietsh F: Preimplant prosthodontics. In Misch CE, editor: *Dental implant prosthetics,* St Louis, 2005, Mosby.
17. Misch CE: Implant overdentures relieve discomfort for the edentulous, *Dentist* 67:37-38, 1989.
18. Naert I, Quirynen M, Theuniers G et al: Prosthetic aspects of osseointegrated fixtures supported by overdentures: a $4^1/_2$-year report, *J Prosthet Dent* 65:671-680, 1991.
19. Spiekermann H, Jansen VK, Richter J: A 10-year follow-up of IMZ and TPS implants in the edentulous mandible using bar-retained overdentures, *Int J Oral Maxillofac Implants* 10:231-243, 1995.
20. Chan MFW, Johnston C, Howell RA et al: Prosthetic management of the atrophic mandible using endosseous implants and overdentures: a 6-year review, *Br Dent J* 179:329-337, 1995.
21. Johns RB, Jemt T, Heath MR et al: A multicenter study of overdentures, supported by Brånemark implants, *Int J Oral Maxillofac Implants* 7:513-522, 1992.
22. Zarb GA, Schmitt A: The edentulous predicament. I. The longitudinal effectiveness of implant-supported overdentures, *J Am Dent Assoc* 127:66-72, 1996.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 6

Treatment Planning: Force Factors Related to Patient Conditions

Carl E. Misch

Biomechanical stress is a significant risk factor in implant dentistry. Its magnitude is directly related to force. As a result, an increase in any dental force factor magnifies the risk of stress-related complications. Different patient conditions place different amounts of force in magnitude, duration, type, and direction. In addition, several factors may multiply or increase the effect of these other conditions. Once the prosthesis option and key implant positions are determined, the potential force levels that will be exerted on the prosthesis should be evaluated and accounted for in order to modify the overall treatment plan. Several elements observed during the dental evaluation predict additional forces on future implant abutments. The initial implant survival, loading survival, marginal crestal bone loss, incidence of abutment or prosthetic screw loosening, and unretained restorations, porcelain fracture, and component fracture are all influenced by the force factors.

Box 6-1 includes primary patient factors affecting the stress environment of the implant and prosthesis.

NORMAL BITE FORCE

The greatest *natural* forces exerted against teeth, and thus against implants, occur during mastication.[1,2] These forces are primarily perpendicular to the occlusal plane in the posterior regions, are of short duration, occur only during brief periods of the day, and range from 5 to 44 lb for natural teeth. The actual force on each tooth during function has been recorded on strain gauges in inlays.[3] A force of 28 psi was needed to chew a raw carrot, and 21 psi was needed to chew meat. The actual time during which chewing forces are applied on the teeth is about 9 minutes each day.[4] The perioral musculature and tongue exert a more constant, yet lighter horizontal force on the teeth or on implants. These forces reach 3 to 5 psi during swallowing.[5] A person swallows 25 times per hour while awake and 10 times per hour while sleeping, for a total of 480 times each day.[4] Therefore natural forces against teeth are primarily in their long axis, less than 30 psi, and for less than 30 minutes for all normal forces of deglutition and mastication (Box 6-2). Forces of mastication placed on implant-supported bridges have been measured in a similar range as natural teeth.

The maximum bite force differs from mastication force, varies widely among individuals, and depends on the state of the dentition and masticatory musculature. There have been many attempts to quantify the normal maximum bite force. In 1681, Borelli suspended weights on a thread over the molars while the mandible was open. The maximum load recorded for which the person was still able to close ranged from 132 to 440 lb. A force

Box 6-1 Patient Force Factors

1. Parafunction
   a. Bruxism
   b. Clenching
   c. Tongue thrust
2. Crown height space
3. Masticatory dynamics
4. Arch position
5. Nature of the opposing arch

Box 6-2 Normal Forces Exerted on Teeth

Bite Forces

Perpendicular to occlusal plane
Short duration
Brief total period (9 min/day)
Force on each tooth: 20 to 30 psi
Maximum bite force: 50 to 500 psi

Perioral Forces

More constant
Lighter
Horizontal
Maximum when swallowing (3 to 5 psi)
Brief total swallow time (20 min/day)

WWW.HIGHDENT.IR

Table 6-1 Mean Maximum Biting Force Recorded on Natural Teeth or Implants

AUTHORS	NATURAL TEETH DENTAL IMPLANTS	MEAN MAXIMUM MASTICATORY FORCE
Carr and Laney, 1987[a]	Conventional denture	59 N
	Implant-supported prostheses	112.9 N
Morneburg and Proschel, 2002[b]	Implant-supported three-unit FPD	220 N
	Single implant: anterior	91 N
	Single implant: posterior	12 N
Fontijn-Tekamp et al., 1998[c]	Implant-supported prostheses	(unilateral)
	Molar region	50-400 N
	Incisal region	25-170 N
Mericske-Stern and Zarb, 1996[d]	Complete denture/implant-supported prostheses	35-330 N
van Eijden, 1991[e]	Canine	469 ± 85 N
	2nd premolar	583 ± 99 N
	2nd molar	723 ± 138 N
Braun et al., 1995[f]	Natural teeth	738 ± 209 N (male > female)
Raadsheer et al., 1999[g]	Male teeth	545.7 N
	Female teeth	383.6 N

Comparison of available studies examining masticatory forces generated under varying loading condition. Study results are reported in Newtons (N) of force unless otherwise indicated. Differences between male and female force generation are noted in applicable studies.
FPD, Fixed partial dentures.
a. Carr AB, Laney WR: Maximum occlusal forces in patients with osseointegrated oral implant prostheses and patients with complete dentures, *Int J Oral Maxillofac Impl* 2:101-108, 1987.
b. Morneburg TR, Proschel PA: Measurement of masticatory forces and implant loads: a methodologic clinical study, *Int J Prosthodont* 15:20-27, 2002.
c. Fontijn-Tekamp FA, Slageter AP, van't Hof MA et al: Bite forces with mandibular implant-retained overdentures, *J Dent Res* 77:1832-1839, 1998.
d. Mericske-Stern R, Assal P, Buergin W: Simultaneous force measurements in three dimensions on oral endosseous implants in vitro and vivo: a methodological study, *Clin Oral Implants Res* 7:378-386, 1996.
e. van Eijden TM: Three dimensional analyses of human bite force magnitude and moment, *Arch Oral Biol* 36:535-539, 1991.
f. Braun S, Bantleon HP, Hnat WP et al: A study of bite force. Part I: relationship to various physical characteristics, *Angle Orthod* 65:367-372, 1995.
g. Raadsheer MC, van Eijden TM, van Ginkel FC et al: Contribution of jaw muscle size and craniofacial morphology to human bite force magnitude, *J Dent Res* 87:31-42, 1999.

of 165 lb was recorded on a gnathodynamometer, the first instrument to record occlusal force, which was developed by Patrick and Dennis in 1892. Black improved this early design and recorded average forces of approximately 170 lb.[6] More recent studies indicate normal maximum vertical biting forces on teeth or implants can range from 45 to 550 psi.[7-22] The forces on the chewing side and the opposite side appear very similar in amplitude[8] (Table 6-1).

It should be emphasized that the maximum bite forces are not expressed by our patients in any routine fashion. However, there are conditions that increase our risks of occlusal overload on the implant prosthesis. Most noteworthy are the parafunctional forces of bruxism and clenching.

PARAFUNCTION

Parafunctional forces on teeth or implants are characterized by repeated or sustained occlusion and have long been recognized as harmful to the stomatognathic system.[23-25] These forces are also most damaging when applied to implant prostheses.[18] The lack of rigid fixation during healing is often a result of parafunction on soft tissue–borne prostheses overlying the submerged implant. The most common cause of both early and late implant failure after successful surgical fixation is the result of parafunction. Such complications occur with greater frequency in the maxilla, because of a decrease in bone density and an increase in the moment of force.[26] The presence of these conditions must be carefully noted during the early phases of treatment planning.

Nadler has classified the causes of parafunction or nonfunctional tooth contact into the following six categories[24]:

1. Local
2. Systemic
3. Psychological
4. Occupational
5. Involuntary
6. Voluntary

Local factors include tooth form or occlusion, as well as soft tissue changes such as ulcerations or pericoronitis. Systemic factors include cerebral palsy, epilepsy, and drug-related dyskinesia. Psychological causes occur with the greatest frequency and include the release of emotional tension or anxiety.[27] Occupational factors concern professionals such as dentists, athletes, and precision workers, as well as the seamstress or musician

WWW.HIGHDENT.IR

who develops altered oral habits. The fifth cause of parafunctional force is involuntary movement that provokes bracing of the jaws, such as during lifting of heavy objects or sudden stops while driving. Voluntary causes include chewing gum or pencils, bracing the telephone between the head and shoulder, and pipe smoking.

The parafunctional groups presented in this chapter are divided into bruxism, clenching, and tongue thrust or size. The dental literature usually does not identify bruxism and clenching as separate entities. Although several aspects of treatment are similar, their diagnosis and treatment are in some ways different. As such, they will be presented as different entities in this discussion. The magnitude of parafunction may be categorized as absent, mild, moderate, or severe. Bruxism and clenching are the most critical factors to evaluate in any implant reconstruction. No long-term success will be obtained with severe parafunction of bruxism or clenching. Therefore the dentist should always try to diagnose the presence of these conditions.

This does not mean that patients with moderate and severe parafunction cannot be treated with implants. A physician treats a patient with uncontrolled diabetes. However, the patient may lose their vision or need their feet amputated, despite treatment, Unsuccessful treatment of the diabetic may not be the fault of the physician. Not recognizing diabetes in the presence of obvious signs and symptoms, of course, is another issue. Because the patient with moderate to severe parafunction represents so many additional risks in implant dentistry, one must be aware of these conditions and the methods to reduce their noxious effects on the entire implant-related system.

Bruxism

Bruxism primarily concerns the horizontal, nonfunctional grinding of teeth. The forces involved are in significant excess of normal physiologic masticatory loads. Bruxism may affect the teeth, muscles, joints, bone, implants, and prostheses. These forces may occur while the patient is awake or asleep and may generate increased force on the system several hours per day. Bruxism is the most common oral habit.[25] Sleep clinic studies have evaluated nocturnal bruxism and found approximately 10% of those observed had obvious movement of the mandible with occlusal contacts.[28,29] More than half of these patients had tooth wear affecting esthetics. Only 8% of these patients were aware of their nocturnal bruxism, and only one quarter of the patients' spouses were aware of the nocturnal habit. Muscle tenderness in the morning was observed less than 10% of the time.[30] A study on bruxing patients with implants showed 80% of sleep bruxism occurred during light sleep stages but did not cause arousal.[31] Therefore patients with bruxism may or may not have obvious tooth wear affecting esthetics; may brux nocturnally, but their bed partners do not know most of the time; rarely have muscle tenderness when they are awake; and are usually unaware of their oral habit. In other words, nocturnal bruxism is sometimes difficult to diagnose.[32]

The maximum biting force of bruxing patients is greater than average. Just as an experienced weight-lifter can lift more weight, the patient constantly exercising the muscles of mastication develops a greater bite force. A man who chews paraffin wax for an hour each day for a month can increase the bite force from 118 to 140 psi within 1 week. Chewing gum, bruxism, and clenching may accomplish the same feat. Eskimos, with a very tenacious diet and who chew their leather to soften it before fabrication of clothing, have maximum bite forces above 300 psi. A 37-year-old patient with a long history of bruxism recorded a maximum bite force of more than 990 psi (four to seven times normal).[33] Fortunately, the bite force does not continue to increase in most bruxing patients. When muscles do not vary their exercise regimen, their size and function adjust to the dynamics of the situation. As a result, the higher bite forces and muscle size usually do not continue in an unending spiral.

Diagnosis

Bruxism does not necessarily represent a contraindication for implants, but it does dramatically influence treatment planning. The first step is to recognize the condition before the treatment is rendered. The symptoms of this disorder, which may be ascertained by a dental history, may include repeated headaches, a history of fractured teeth or restorations, repeated uncemented restorations, and jaw discomfort upon awakening.[23,34] Therefore when the patient is aware of muscle tenderness or the spouse is conscious of the nocturnal condition, the diagnosis is readily obtained. However, many patients do not attribute these problems to excessive forces on the teeth and report a negative history. A lack of these symptoms does not negate bruxism as a possibility.

Fortunately many clinical signs warn of excessive grinding. The signs of bruxism include an increase in size of the temporal and masseter muscles (these muscles and the external pterygoid may be tender), deviation of the lower jaw on opening, limited occlusal opening, increased mobility of teeth, cervical abfraction of teeth, fracture of teeth or restorations, and uncemented crowns or fixed prostheses. However, the best and easiest way to diagnose bruxism is to evaluate the wearing of teeth. Not only is this the easiest method to determine bruxism in an individual patient, it also allows the disorder to be classified as absent, mild, moderate, or severe (Figures 6-1 to 6-3). No anterior wear patterns in the teeth signify an absence of bruxism. Mild bruxism has slight wearing of anterior teeth but is not a cosmetic compromise. Moderate bruxism has obvious anterior

WWW.HIGHDENT.IR

Figure 6-1 This patient has mild bruxism. Note the wear facet on the mandibular canine and the slight notch in the maxillary lateral incisor.

Figure 6-2 Patients often grind their teeth in a specific, repeated movement of the mandible. When the opposing wear facets of the teeth are in contact, one should note the occlusal position of the teeth. The patient shown in Figure 6-1 has a working contact on the mandibular premolar with the maxillary canine in this engram position. The slight cervical abfraction of the mandibular first premolar is a consequence of the parafunction. The patient's posterior teeth should not occlude in this position to decrease the amount of force on the anterior teeth.

incisal wear facets but no posterior occlusal wear pattern. Severe bruxism has absent incisal guidance from excessive wear, and posterior wearing of the teeth is obvious.

Nonfunctional wear facets on the incisal edges may occur on both natural or replacement teeth, especially in the mandible and maxillary canines, and there may be notching of the cingulum in the maxillary anterior teeth. Isolated anterior wear is not much of a concern if all posterior teeth contacts can be eliminated in excursions.

Figure 6-3 This patient has severe bruxism because occlusal wear is anterior and posterior. The incisal guidance should be reestablished before a maxillary arch fixed reconstruction.

Figure 6-4 This patient exhibits an engram pattern of bruxism primarily toward the left canine to central incisors. The right canine and lateral incisor have far fewer wear facets. This "pathway of destruction" is specific.

Tooth wear is most significant when found in the posterior regions and changes the intensity of bruxism from the moderate to the severe category. Posterior wear patterns are more difficult to manage, because this usually is related to a loss of anterior guidance in excursions; once the posterior teeth contact in excursive jaw positions, greater forces are generated.[35] The masseter and temporalis muscles contract when posterior teeth contact. With incisal guidance and an absence of posterior contact, two thirds of these muscles do not contract and, as a consequence, the bite force is dramatically reduced. However, when the posterior teeth maintain contact, the bite forces are similar in excursions, as during posterior biting. Therefore in the patient with severe bruxism, the occlusal plane, the anterior incisal guidance, or both may need modification to eliminate all posterior contacts during mandibular excursions before the implant restoration.

Bruxing patients often repeat mandibular movements, which are different from border movements of the mandible and are in one particular direction. As a result, the occlusal wear is very specific and primarily on one side of the arch, or even on only a few teeth (Figure 6-4).

WWW.HIGHDENT.IR

This engram pattern usually remains after treatment. If the restoring dentist reestablishes incisive guidance on teeth severely affected by an engram bruxing pattern, the incidence of complications on these teeth will be increased. The most common complications on teeth restored in this "pathway of destruction" are porcelain fracture, uncemented prostheses, and root fracture.[34] When implants support the crowns in the pathway of destruction, the implant may fail, fracture, or have crestal bone loss, abutment screw loosening, porcelain fracture, or unretained restorations.[36-39] If the patient continues the severe bruxism pattern, the question is not whether, but when and which complications will occur. The dentist should tell the patient that these habits will cause these problems. Treatment may be rendered to repair these problems, but there will be complications if the bruxism is not reduced.

Bruxism changes normal masticatory forces by the magnitude (higher bite forces), duration (hours rather than minutes), direction (lateral rather than vertical), type (shear rather than compression), and magnification (four to seven times normal).[33,40-42] The method to restore severe bruxism may be problematic, even when the desire is primarily cosmetic. As the anterior teeth wear, they often erupt and the overall occlusal vertical dimension remains unchanged. In addition, the alveolar process may follow the eruption of the tooth. As such, when the anterior teeth are restored for esthetics (or to obtain an incisal guidance), the reduced crown height cannot be increased merely by increasing the height of the crown to an average dimension. Instead, the following guidelines are suggested:

1. Determine the position of the maxillary incisor edge of the anterior teeth. They may be acceptable (if eruption occurred as they wore) or need greater coronal length to correct related incisal wear.
2. Determine the desired occlusal vertical dimension. This is not an exact dimension and may exist at several different positions without consequence. However, like most factors, there is a range that is patient specific and does follow guidelines. The most common methods to determine this dimension relate to facial measurements, closest speaking space, physiologic rest position, speech, and esthetics. This is one of the most important steps. If the vertical dimension is collapsed because of anterior and posterior occlusal wear, much more rehabilitation is required. This condition is observed more often when bruxism is severe, the anterior incisal guidance was lost, and, as a consequence, the severe bruxism wear is increased due to an increase in force factors. The accelerated occlusal wear may cause a loss of occlusal vertical dimension (OVD). The OVD is rarely decreased when incisal guidance is still present, as the posterior teeth maintain the dimension and the anterior teeth have time to erupt because the forces are less and the wear rate is slower.
3. Evaluate and restore the position of the lower anterior teeth where necessary. In the past, several authors have stated that a reconstruction begins with the lower anterior teeth. The mandibular arch cannot be restored until the maxillary anterior teeth and occlusal vertical dimension are established. Many esthetic and speech guidelines are available to help the restoring dentist with the position of the maxillary anterior teeth. For example, when a dentist begins the restoration of a completely edentulous patient, the maxillary anterior wax rim position is often first determined, for similar reasons.

The position of the lower anterior teeth should contact the lingual surfaces of the maxillary anterior teeth at the established occlusal vertical dimension, and the amount of vertical overlap of the maxillary incisal edge and the angle of the incisal contacts in protrusive movements of the mandible determines the angle and height of the anterior guidance. This dimension must be greater than the condylar disc assembly (the angle of the eminentia) so the posterior teeth will separate during mandibular excursions.

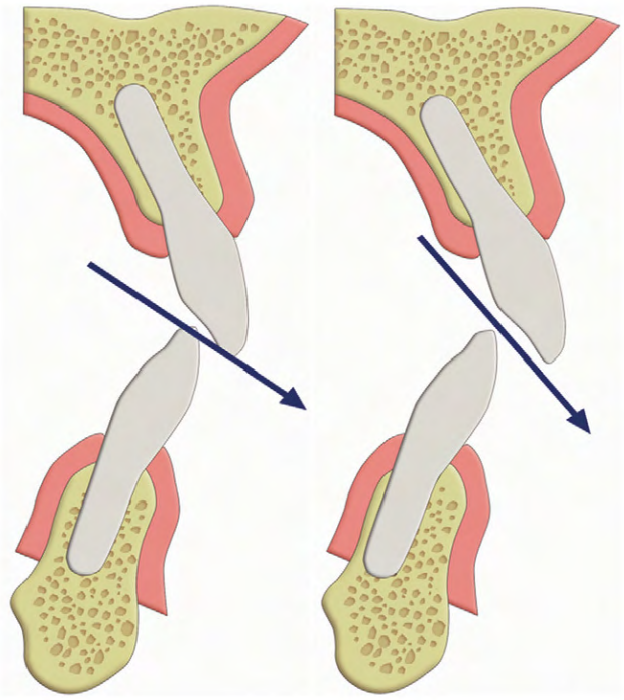
In patients with moderate to severe bruxism, the height of the vertical overjet and the angle of incisal guidance should not be extreme, as the amount of the force on the anterior abutments, cement seals, and porcelain is directly related to these conditions (Figure 6-5). In other words, the greater the incisal overjet, the greater the distance between the posterior teeth in excursions, and the greater the force generated on the anterior teeth during this movement. In patients with severe bruxism, the intensity of the force should be reduced, because the duration of the force is increased.

When anterior tooth wear is accompanied by tooth eruption and maintenance of the occlusal vertical dimension, and alveolar bone in the region has extruded toward the incisal plane, the incisal edges of the teeth should not be elevated. Instead, the alveolar bone and cervical regions should be reduced and crown lengthening should be performed on the teeth before their restoration. This is most often necessary in the mandibular anterior region but may be observed in any region of the mouth after long-term severe bruxism. In addition, endodontic therapy may be required to allow proper anterior tooth preparation.

Crown lengthening and associated procedures are not necessary when the vertical dimension has been reduced in relation to the incisal wear. Instead, the teeth may be prepared in their present state. The restoration restores the occlusal vertical dimension and reestablishes anterior incisal guidance.

4. The posterior plane of occlusion s then determined. This may be accomplished by using first the maxillary arch or the posterior mandibular arch.

WWW.HIGHDENT.IR



A B

Figure 6-5 The incisal guidance for a patient with moderate to severe bruxism **(A)** should be shallow to reduce the force on the anterior teeth during excursive movement **(B)** of the mandible.

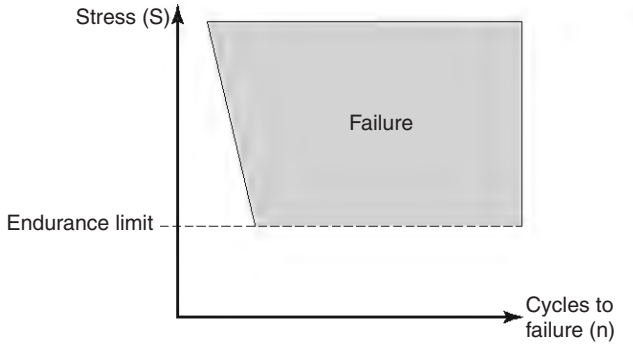


Figure 6-6 On a fatigue curve for a material, stress corresponds to the vertical axis and cycles to failure to the horizontal axis. A point exists at which the stress is so great that the material breaks with only one cycle. When the stress is low enough, the material will not break, regardless of the number of cycles. The stress amount at the highest point of this safe zone is called the en*durance limit*. Patients with parafunction increase the amount of stress to the implant-prosthetic system and increase the number of the cycles for the higher levels. Fatigue failures are therefore common.

However, it is best if the same bilateral posterior quadrants are addressed at the same time, so that the posterior plane may be parallel to the horizontal plane. The maxillary posterior region is most often determined first in the completely edentulous patient.

Fatigue Fractures

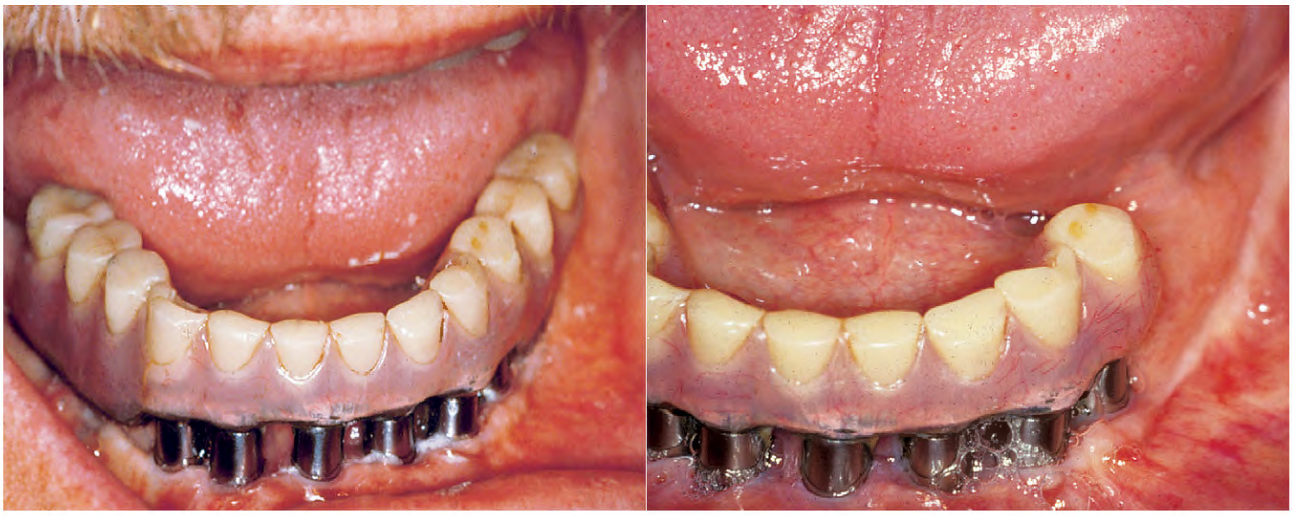
The increase in duration of the force is a considerable problem. Materials follow a fatigue curve, which is affected by the number of cycles and the intensity of the force[43-45] (Figure 6-6). A force can be so great that one cycle causes a fracture (e.g., a karate blow to a piece of wood). However, if a lower force magnitude repeatedly hits an object, the object will still fracture. The wire coathanger that is bent does not break the first time, but repeated bends will fracture the material, not because the last bend was more forceful but because of fatigue. A bruxing patient is at greater risk of fatigue fractures for two reasons. The magnitude of the forces increases over time as the muscles become stronger, and the number of cycles increase on the prosthetic components. Eventually, one of the components (implant, screw, abutment, prosthesis) will break if the parafunction cannot be reduced in intensity or duration (Figure 6-7). No long-term prosthetic result is expected in patients with severe bruxism. Therefore, once the implant dentist has identified the sources of additional force on the implant system, the treatment plan is altered in an attempt to minimize the negative effect on the longevity of the implant, bone, and final restoration. All elements able to reduce stress should be considered.

Occlusal Guards

The cause of bruxism is multifactorial and may include occlusal disharmony.[46] When an implant reconstruction is considered in a bruxing patient, occlusal analysis is warranted. Premature and posterior contacts during mandibular excursions increase stress conditions. An elimination of eccentric contacts may allow recovery of periodontal ligament health and muscle activity within 1 to 4 weeks. Occlusal harmony does not necessarily eliminate bruxism, but this is no reason not to perform an occlusal analysis and eliminate the premature contacts. No study demonstrates an increase in parafunction after occlusal adjustment. Therefore the ability to decrease the risk of occlusal overload on particular teeth and the added benefit of perhaps reducing parafunction is warranted in almost every patient diagnosed with a parafunctional habit of bruxism or clenching.

A night guard can be a useful diagnostic tool to evaluate the influence of occlusal disharmony on nocturnal bruxism. The Michigan night guard exhibits even occlusal contacts around the arch in centric relation occlusion and provides posterior disocclusion with

WWW.HIGHDENT.IR



A B

Figure 6-7 **A,** The patient has a cantilevered hybrid fixed prosthesis in the mandible. Severe bruxism is evident, with anterior and posterior wearing of the teeth. At this point the patient should be informed that the incisal guidance of the restoration should be restored. **B,** The patient from **A** elected not to restore the incisal guidance of the restoration. As a consequence of higher forces with posterior contact during excursions, fatigue failure of the prosthesis occurred.

anterior guidance in all excursions of the mandible.[47] This device may be fabricated with 0.5 to 1 mm colored acrylic resin on the occlusal surface. After 4 weeks of nocturnal wear, if the patient wears this device for an additional month or more, the influence of occlusion on the bruxism may be directly observed. There are no premature contacts while the device is worn; however, if the colored acrylic is still intact, the nocturnal parafunction has been reduced or eliminated.[48] Therefore occlusal reconstruction or modification may proceed. If the colored acrylic on the night guard is ground through, an occlusal adjustment will have little influence on decreasing this parafunctional habit. The night guard is still indicated to relieve stresses during nocturnal parafunction, but the treatment plan should account for the greater forces.

Forces from bruxism are the most difficult to address on a long-term basis. Education and informed consent of the patient are helpful to gain cooperation in eliminating or reducing the noxious effects. If the opposing arch is a soft tissue–supported removable prosthesis, the effects of the nocturnal habit may be minimized if the patient removes the prosthesis at night. The use of a night guard is helpful for a patient with a fixed prosthesis, in order to transfer the weakest link of the system to the removable acrylic device.[49] Centric contacts in centric relation occlusion and anterior-guided disocclusion of the posterior teeth in excursions are strongly suggested on the night guard, which may be designed to fit the maxilla or mandible.

Unlike teeth, implants do not extrude in the absence of occlusal contacts. As a result, in partially edentulous patients, the maxillary night guard can be relieved around the implant crowns so the remaining natural teeth bear the entire load. For example, for a maxillary implant restoration, the night guard is hollow so no occlusal force is transmitted to the implant crown. When the restoration is in the mandible, the occluding surfaces of the maxillary night guard are relieved over the implant crowns so no occlusal force is transmitted to the implants (Figure 6-8).

A mandibular posterior cantilever on a full-arch implant prosthesis may also be taken out of occlusion with a maxillary night guard. When a posterior quadrant of implants supports a fixed prosthesis in the maxilla opposing mandibular teeth, a soft reline material is placed around the implant crowns to act as a stress relief element and decrease the impact force on the restoration (Figure 6-9). When full-arch implant restorations are opposing each other, the night guard provides solely anterior contacts during centric occlusion and mandibular excursions. Thus the parafunctional force is reduced on the anterior teeth/implants and eliminated in the posterior regions.

Treatment Planning

The implant treatment plan is modified primarily in two ways when implants are inserted in the posterior region: (1) additional implants that are wider in diameter are one method used to reduce the overload risk or (2) the

WWW.HIGHDENT.IR

anterior teeth may be modified to recreate the proper incisal guidance and avoid posterior interferences during excursions. The elimination of posterior lateral occlusal contacts during excursive movements is recommended when opposing natural teeth or an implant or tooth-supported fixed prosthesis. This is beneficial in two aspects: (1) because lateral forces dramatically increase stress at the implant-bone interface, the elimination of posterior contacts diminishes the negative effect of angled forces during bruxism; (2) the presence of posterior contacts during excursions, almost all fibers of the masseter, temporalis, and the external pterygoid muscles contract and place higher forces on the anterior teeth/implants. On the contrary, during excursions in the absence of posterior contacts, fewer fibers of the temporalis and masseter muscles are stimulated, and the forces applied on the anterior implant-teeth system are reduced by as much as two thirds.[25]

Figure 6-8 A night guard for a partially edentulous patient restored with implants may be designed to transfer the force to the implant prosthesis. When the implant prosthesis is in the opposing arch to the guard, there are no occludal contacts in centric or during excursion with the guard in place.

Figure 6-9 Full-arch implant prostheses opposing each other may have only anterior occlusal contact in centric and during mandibular excursions with the guard in place.

Clenching

Clenching is a habit that generates a constant force exerted from one occlusal surface to the other without any lateral movement. The habitual clenching position does not necessarily correspond to centric occlusion. The jaw may be positioned in any direction before the static load; therefore a bruxing and clenching combination may exist. The clench position most often is in the same repeated position and rarely changes from one period to another. The direction of load may be vertical or horizontal. The forces involved are in significant excess of normal physiologic loads and are similar to bruxism in amount and duration; however, several clinical conditions differ in clenching.

Diagnosis

Many clinical symptoms and signs warn of excessive grinding. However, the signs for clenching are often less obvious. The forces generated during clenching are directed more vertically to the plane of occlusion, at least in the posterior regions of the mouth. Wearing of the teeth is usually not evident; therefore clenching often escapes notice during the intraoral examination. As a result, the dentist must be more observant to the diagnosis of this disorder.[25,46,49]

Many of the clinical signs of clenching often resemble bruxism. When a patient has a dental history of muscle tenderness upon awakening or tooth sensitivity to cold, parafunction is strongly suspected. In the absence of tooth wear, clenching is the prime suspect. Tooth mobility, muscle tenderness or hypertrophy, deviation during occlusal opening, limited opening, stress lines in enamel, cervical abfraction, and material fatigue (enamel, enamel pits, porcelain and implant components) are all associated clinical signs of clenching. All these conditions may also be found in the bruxing patient. However, enamel wear has such a strong correlation to bruxism that it is the primary and often the only factor needed to evaluate for bruxism. The clenching patient has the "sneaky disease of force." Therefore particular attention is paid to diagnose this disorder from less obvious clinical conditions.

A physical examination for the implant candidate should include palpation of the muscles of mastication. The masseter and temporalis muscles are easily examined at the initial appointment. Hyperactive muscles are not always tender, but tender muscles in the absence of trauma or disease is a sign of excess use or incoordination among muscle groups. The lateral pterygoid muscle is more often overused by the bruxing or clenching patient but is difficult to palpate. The ipsilateral medial pterygoid muscle provides more reliable information in this region. It acts as the antagonist to the lateral pterygoid in hyperfunction and, when tender, provides a good indicator of overuse of the lateral pterygoid.[46]

WWW.HIGHDENT.IR

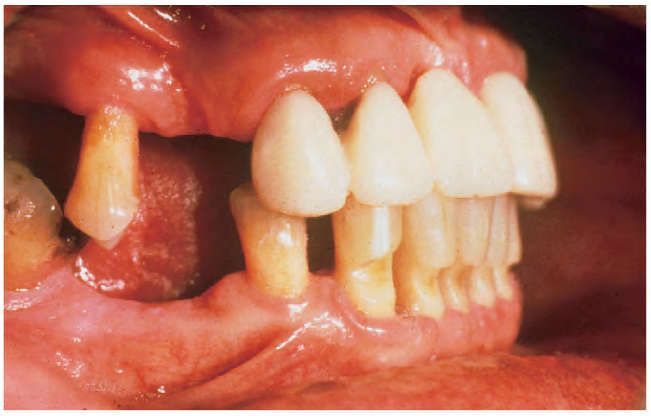


Figure 6-10 Clenching habits are more difficult to diagnose because occlusal wear is often absent. This clenching patient has cervical abfraction of the mandibular anterior teeth. In this patient, the abfraction obviously is not from toothbrush abrasion.

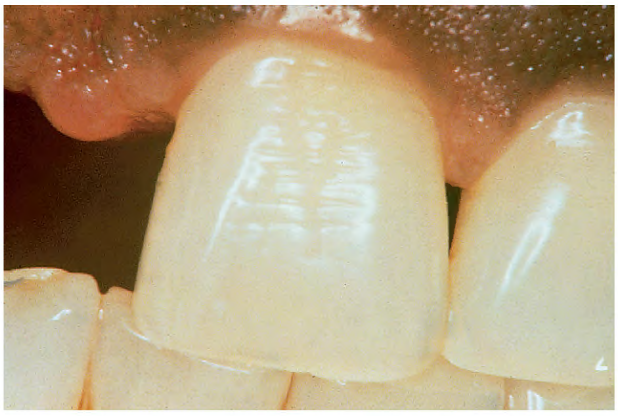


Figure 6-11 This patient has horizontal abfraction lines in the enamel from clenching.

Muscle evaluation for clenching also includes deviation during opening the jaw, limited opening, and tenderness of the temporomandibular joint. Deviation to one side during opening indicates a muscle imbalance on the same side.[46] Limited opening is easily evaluated and may indicate muscular imbalance or degenerative joint disease. The normal opening should be at least 40 mm from the maxillary incisal edge to the mandibular incisal edge in an Angle's Class I patient, taking into consideration an overjet or overlap. If any horizontal overjet or overlap exists, its value in millimeters is subtracted from the 40-mm minimum opening measurement.[50] The range of opening without regard for overlap or overjet has been measured in the range of 38 to 65 mm for men and 36 to 60 mm for women, from incisal edge to edge.[51]

Increased mobility of teeth may be an indication of a force beyond physiologic limits, bone loss, or their combination. This requires further investigation in regard to parafunction and is very important if an implant may be placed in the region of the mobile teeth. The rigid implant may receive more than its share of occlusal force when surrounded by mobile teeth. Fremitus, a vibration type of mobility of a tooth, is often present in the clenching patient. To evaluate this condition, the dentist's finger barely contacts the facial surface of one tooth at a time and feels for vibrations while the patient taps the teeth together. Fremitus is symptomatic of local excess occlusal loads.

Cervical erosion is primarily a sign of parafunctional clenching or bruxism (Figure 6-10). In the past, Black analyzed the eight most popular theories for gingival ditching of the teeth, finding all inconclusive. This observation has frequently been called "toothbrush abrasion." McCoy has reported this condition on every other tooth, only one tooth, and even on the teeth of some animals. Parafunction was the common link among patients presenting with this condition.[52] The notched appearance of the cervical portion of the tooth directly correlates with the concentration of forces shown in three-dimensional finite analysis[53] and photoelasticity studies.[54] Abfraction of teeth was also observed in cats, rats, and marmosets and was described in the literature as early as 1930.[55] A study of a noninstitutionalized older human population revealed that cervical abrasion was present in 56% of the participants.[56]

Other signs of enamel or occlusal material fatigue encountered in bruxing or clenching patients include occlusal invaginations or pits, stress lines in enamel, stress lines in alloy restorations or acrylic (lines of Luder), and material fracture (Figures 6-11 and 6-12). Fremitus can be noticed clinically on many cervically eroded, nonmobile teeth. Not all gingival erosions are caused by parafunction. However, when present, the occlusion should be carefully evaluated along with other signs of excess force. If excessive forces appear to be the cause, the condition is referred to as *cervical abfraction*.[57]

A common clinical finding of clenching is a scalloped border of the tongue (Figure 6-13). The tongue is often braced against the lingual surfaces of the maxillary teeth during clenching, exerting lateral pressures and resulting in the scalloped border. This braced tongue position may also be accompanied by an intraoral vacuum, which permits a clench to extend for a considerable time, often during sleep.

Fatigue Fractures

The increase in force magnitude and duration is a significant problem, whether by bruxism or clenching. The fatigue curve previously presented for bruxism also applies to clenching. In addition, the clenching patient may suffer from a phenomenon called *creep*, which also results in fracture of components. Creep occurs in a material when an increasing deformation is expressed as a function of time, when subjected to a constant load (Figure 6-14). Although the cycles of load may

WWW.HIGHDENT.IR

Figure 6-12 **A,** This patient has cervical abfraction on the mandibular hybrid fixed implant-supported prosthesis from clenching. **B,** The patient in **A** refused to wear an occlusal guard at night. The abfraction continued and affected the denture teeth and cervical areas of the hybrid fixed-implant restoration in the maxilla. The mandibular implants then fractured from fatigue and creep.

Figure 6-13 A scalloped border of the tongue most often is found in a clenching patient. To maintain the force between the teeth, a vacuum is created in the mouth, and the impression of the lingual contours of the upper teeth is seen on the tongue.

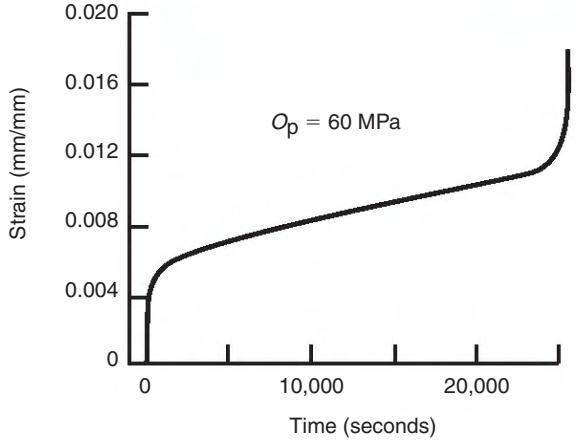


Figure 6-14 A creep curve for materials is created, placing strain on the vertical axis and time on the horizontal axis when a constant load is applied. This is a creep curve for bone at a load of 60 MPa. The bone changes shape (i.e., strain) at the initial stress condition and then at an increasing amount over time until the material breaks.

not be present to affect the deformation of a material, the constant force is still able to cause fracture. In other words, something will break if the continued force is not abated or at least reduced in intensity or duration (Figure 6-15). This condition may also occur in bone, which may result with implant mobility and failure. All elements to reduce the excessive force of clenching and its consequence should be considered.

Clenching affects the treatment plan in a fashion similar to bruxism. However, the vertical forces are less detrimental than horizontal forces, and alteration of the anterior occlusal scheme is not as critical as with the bruxing patient. Night guards are also less effective. However, a hard acrylic shell and softer, resilient liner night guard, which is slightly relieved over the implants, is often beneficial to a clenching patient. Unlike teeth,

WWW.HIGHDENT.IR

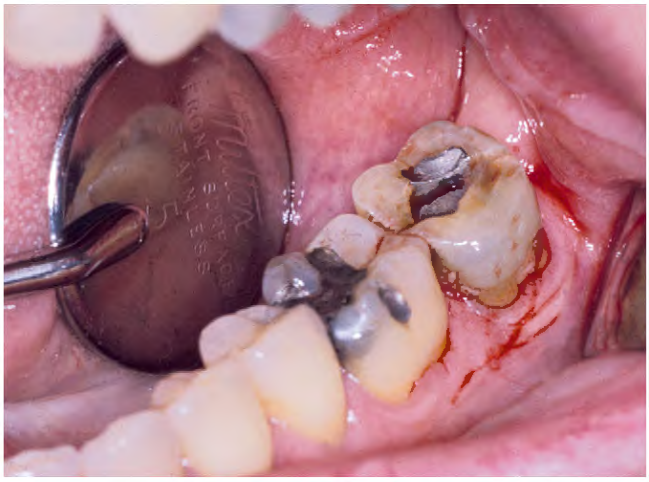


Figure 6-15 The patient exhibits clenching, diagnosed from the enlarged size of the masseter and temporalis muscles. The mandibular second molar has fractured from mesial to distal. Note the slight abfraction on the distal buccal root of the first mandibular molar.

Figure 6-16 A night guard with a rigid acrylic shell and a soft resilient liner can decrease stress during clenching episodes.

implants do not extrude. As a result, the night guard can be relieved around an intermediate implant, and the teeth bear the entire load. In a full-arch or quadrant implant restoration, the night guard provides a biomechanical advantage to reduce the impact of the force during clenching[58] (Figure 6-16).

A common cause of implant failure during healing is parafunction in a patient wearing a soft tissue–supported prosthesis over a submerged implant. The tissue overlying the implant is compressed during the parafunction. The premature loading may cause micromovement of the implant body in the bone and may compromise osteointegration. When an overlying soft tissue–borne restoration exerts pressure as a result

Figure 6-17 When a removable partial denture is worn over healing implants, a 6-mm hole over each implant site reduces the risk of parafunctional loads transferred through the overlying tissue.

of parafunction, pressure necrosis causes soft tissue dehiscence over the implant. This condition is not corrected by surgically covering the implant with soft tissue, but the soft tissue support region of the prosthesis over the implant should be generously relieved during the healing period whenever parafunction is noted. A removable partial denture over a healing implant is especially of concern. The acrylic between the soft tissue–borne region and metal substructure is usually less than 1 mm thick. Removing the thin acrylic region over the implant is often insufficient. Instead, a 6-mm–diameter hole through the metal substructure should be prepared (Figure 6-17).

Prosthetic Considerations

The time intervals between prosthodontic restoration appointments may be increased to provide additional time to produce load-bearing bone around the implants through progressive bone-loading techniques. Anterior implants submitted to lateral parafunction forces require further treatment considerations. Additional implants are indicated, preferably of greater diameter. The excursions are canine guided if natural, healthy canines are present. Mutually protected occlusion, with additional anterior implants or teeth distributing forces, is developed if the implants are in the canine position or if this tooth is restored as a pontic. The prosthesis may be designed to improve the distribution of stress throughout the implant system with centric vertical contacts aligned with the long axis of the implant whenever possible. Narrow posterior occlusal tables to prevent inadvertent lateral forces and to decrease the occlusal forces are beneficial.[59] Enamoplasty of the cusp tips of the opposing natural teeth is indicated to help improve the direction of vertical forces, within the guidelines of the intended occlusion.

Clenching increases the risk of mechanical failure, such as porcelain fracture, uncemented restoration,

WWW.HIGHDENT.IR

Figure 6-18 This patient fractured the porcelain on her fixed mandibular implant–supported restoration. The cervical regions were the primary sites of fracture because the patient had a clenching habit.

Figure 6-19 Six different types of tongue-thrust habits have been classified. This patient has an anterior tongue thrust and, as a result, does not have anterior guidance.

Figure 6-20 This patient has a unilateral posterior tongue thrust. When the patient swallows, the tongue is forced between the maxillary canine and first premolar, the mandibular lateral incisor and canine, and the posterior edentulous regions in both arches. Posterior one-stage implants would receive an immediate horizontal load. The patient will feel that the implant prosthesis is constricting the tongue.

abutment screw fracture, implant body fracture, and crestal bone loss (Figure 6-18).

TONGUE THRUST AND SIZE

Parafunctional tongue thrust is the unnatural force of the tongue against the teeth during swallowing.[60] A force of approximately 41 to 709 g/cm^2 on the anterior and lateral areas of the palate has been recorded during swallowing.[61] In orthodontic movement, a few grams of constant force are sufficient to displace teeth. Six different types of tongue thrust have been identified; anterior, intermediate, posterior, and either unilateral or bilateral may be found, and in most any combination (Figures 6-19 and 6-20). A common question is which came first, the aberrant tongue position or the misalignment of teeth? Regardless, this condition can contribute to implant healing and prosthetic complications. Although the force of tongue thrust is of lesser intensity than in other parafunctional forces, it is horizontal in nature and can increase stress at the permucosal site of the implant. This is most critical for one-stage surgical approaches in which the implants are in an elevated position at initial placement and the implant interface is in an early healing phase. The tongue thrust may also contribute to incision line opening, which may compromise both the hard and soft tissues.

A tongue-thrust habit may lead to tooth movement or mobility, which is of consequence when implants are present in the same quadrant. If the natural teeth in the region of the tongue thrust were lost as a result of an aberrant tongue position or movement, the implants are at increased risk during initial healing and early prosthetic loading. If the remaining teeth exhibit increased mobility, the implant prosthesis may be subject to increased occlusal loads. To evaluate anterior tongue thrust, the doctor holds the lower lip down, squirts water into the mouth with the water syringe, and asks the patient to swallow. A normal patient forms a vacuum in the mouth, positions the tongue on the anterior aspect of the palate, and is able to swallow without difficulty. A patient with an anterior tongue thrust is not able to create the vacuum needed to swallow when the lower lip is retracted, because the seal and vacuum for the patient are achieved between the tongue and the lower lip. As a consequence, the patient is unable to swallow while the lower lip is withdrawn.

A posterior tongue thrust is evaluated by retracting one cheek at a time away from the posterior teeth/edentulous region with a mirror, injecting water into the mouth with a water syringe, and asking the patient to swallow. Visual evidence of the tongue during

WWW.HIGHDENT.IR

Figure 6-21 This patient has a maxillary complete denture and no posterior mandibular teeth. The patient has developed a posterior tongue position to support the denture and prevent it from dropping posteriorly when the patient occludes. This tongue will adapt easily to a mandibular posterior implant prosthesis.

Figure 6-22 In a patient with missing teeth and no prosthetic replacement, such as a complete or partial denture, the tongue often increases in size. Although the tongue does not transfer an active lateral force during swallowing, prosthetic complications of tongue biting are at a greater risk.

deglutition may also be accompanied by pressure against the instrument and confirms a lateral force. The posterior tongue thrust may occur in patients wearing a maxillary denture opposing a Kennedy Class 1 mandibular arch, without a mandibular prosthesis replacing the posterior teeth. Under these conditions, the maxillary denture often loses valve seal and drops posteriorly, as only anterior teeth contact. To limit this problem, the patient extends the lateral aspect of the tongue into the edentulous region to prevent the maxillary denture from dislodgement (Figure 6-21).

A potential prosthetic complication for a patient with a lateral tongue thrust is the complaint of inadequate room for the tongue once the mandibular implants are restored. A prosthetic mistake is to reduce the width of the lingual contour of the mandibular teeth. The lingual cusp of the restored mandibular posterior teeth should follow the curve of Wilson and include proper horizontal overjet to protect the tongue during function. A reduction in the width of the posterior teeth often increases the occurrence of tongue biting and may not dissipate with time. Rather than being a short-term inconvenience, the prosthesis may need to be refabricated. The restoring dentist should identify the tongue position before treatment and inform the patient about the early learning curve for the tongue once the teeth are delivered on the implants.

Even in the absence of tongue thrust, the tongue often accommodates to the available space, and its size may increase with the loss of teeth. As a result, a patient not wearing a mandibular denture often has a larger than normal tongue. The placement of implants and prosthetic teeth in such a patient results in an increase in lateral force, which may be continuous. This patient complains of inadequate room for the tongue and may bite it during function. However, this condition is usually short-lived, and the patient eventually adapts to the new intraoral condition (Figure 6-22).

CROWN HEIGHT SPACE

The interarch distance is defined as the vertical distance between the maxillary and mandibular dentate or dentate arches under specific conditions (e.g., the mandible is at rest or in occlusion).[62] A dimension of only one arch does not have a defined term in prosthetics; therefore Misch proposed the term *crown height space* (CHS).[63] The CHS for implant dentistry is measured from the crest of the bone to the plane of occlusion in the posterior region and the incisal edge of the arch in question in the anterior region (Figure 6-23; Box 6-3). In the anterior regions of the mouth, the presence of a vertical overbite means the CHS is larger in the maxilla than the space from the crest of the ridge to the opposing teeths incisal edge. In general, when the anterior teeth are in contact in centric occlusion, there is a vertical overbite. The anterior mandibular CHS is therefore usually measured from the crest of the ridge to the mandibular incisal edge. However, the anterior maxillary CHS is measured from the maxillary crestal bone to the maxillary incisal edge, not the occlusal contact position.

The ideal CHS needed for a fixed implant prosthesis should range between 8 and 12 mm. This measurement accounts for the biological width, abutment height for cement retention or prosthesis screw fixation, occlusal material strength, esthetics, and hygiene considerations around the abutment crowns. Removable prostheses often require a CHS greater than 12 mm for denture teeth and acrylic resin base strength, attachments, bars, and oral hygiene considerations.[64,65]

WWW.HIGHDENT.IR

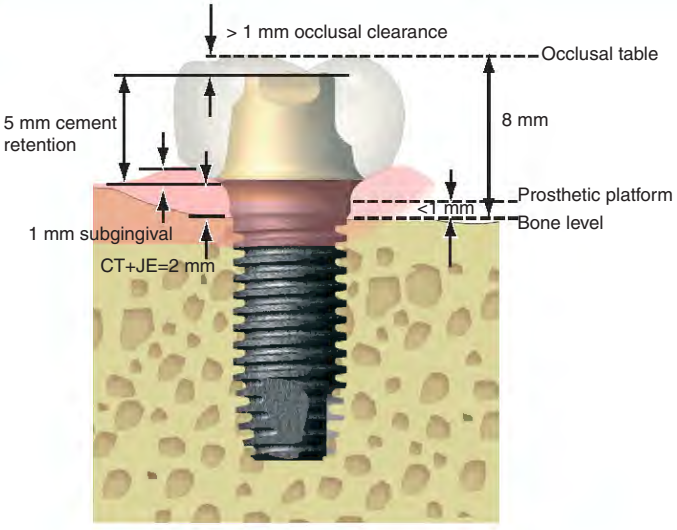


Figure 6-23 The crown height space (CHS) is measured from the occlusal plane to the crest of the bone.

Biomechanic Consequences of Excessive Crown Height Space

Mechanical complication rates for implant prostheses are often the highest of all complications reported in the literature.[39,66] Mechanical complications are often caused by excessive stress applied to the implant-prosthetic system (see Box 6-3). Implant failure may occur from overload and result in prosthesis failure and bone loss around the failed implants. Implant body fracture may result from fatigue loading of the implant at a higher force, but occurs at less incidence than most complications. The higher the force, the fewer the number of cycles before fracture, so the incidence increases. Crestal bone loss may also be related to excessive forces and often occurs before implant body fracture. Porcelain and occlusal material fracture rates may increase as the force to the restoration is increased. The risk of fracture to the opposing prosthesis increases with an average of 12% in implant overdentures opposing a denture.[66] With resin veneer implant fixed partial dentures, 22% of the veneers fractured. Clips or attachment fractures in overdentures may average 17%. Fracture of the framework or substructure may also occur as a result of an increase in biomechanical forces.

Force magnifiers are situations or devices that increase the amount of force applied and include a screw, pulley, incline plane, and a lever.[43] The biomechanics of CHS are related to lever mechanics. The properties of a lever have been appreciated since the time of Archimedes, 2000 years ago. The issues of cantilevers and implants were demonstrated in the edentulous mandible, where the length of the posterior cantilever directly related to complications or failure of the prosthesis.[39] Rather than a posterior cantilever, the CHS is a vertical cantilever when any lateral or cantilevered load is applied and, therefore, is also a force magnifier.[44,45] As a result, because CHS excess increases the amount of force, any of the mechanical-related complications related to implant prostheses may also increase (Figure 6-24).

When the direction of a force is in the long axis of the implant, the stresses to the bone are not magnified in relation to the CHS. However, when the forces to the implant are on a cantilever, or a lateral force is

Box 6-3 Biomechanical Principles Related to Crown Height Space

1. The CHS is measured from the occlusal plane to the crest of the bone.
2. Mechanical complications are the primary cause of complications after prosthesis delivery.
3. Mechanical complications are often caused by excessive stresses.
4. Excessive stress can cause implant failure, crestal bone loss, implant fracture, screw loosening, occlusal material fracture, prosthesis fracture, or attachment wear and fracture.
5. The crown height is a vertical cantilever.
6. The biomechanics are more unfavorable as the CHS increases.
7. An increase in CHS increases the forces on cantilevered or angled loads.
8. Crestal bone loss around the implant increases the CHS and therefore increases the moment forces to the implant and prosthesis components.
9. CHS does not have a specific ideal dimension. With fixed restorations, the acceptable range for CHS is between 8 and 12 mm.
10. Removable implant restorations often require a CHS of 12 mm or more, especially when a bar connects the individual implants.
11. Stresses applied to implants are concentrated in the crestal region, so increasing implant length is less effective to reduce the effects of crown height than a natural tooth root.
12. Methods to decrease stress should be considered when the CHS is increased (i.e., increase implant number, size, and surface area of design; splint implants together; shorten cantilevers; consider removable restorations; add soft tissue support in overdentures).
13. An increase in prosthetic complications occurs with either limited or excessive CHS.

CHS, Crown height space.

WWW.HIGHDENT.IR

Influences on Moment		Imposed Moments (N/mm) at Implant Crown-to-Crest Interface					
OCCLUSAL HEIGHT (mm)	CANTILEVER LENGTH (mm)	LINGUAL	FACIAL	APICAL	OCCLUSAL	FACIAL TRANSVERSE	LINGUAL TRANSVERSE
10	10	100	0	50	200	0	100
10	20	100	0	50	400	0	200
10	30	100	0	50	600	0	300
20	10	200	0	100	200	0	100
20	20	200	0	100	400	0	200
20	30	200	0	100	600	0	300

FP-1 FP-3

Figure 6-24 The crown height space (CHS) is a vertical cantilever. The FP-3 prosthesis on the right will deliver greater stresses to the implant compared with the implant on the left. Therefore a wider-diameter implant is of benefit to support the implant restoration on the right.

applied to the crown, the forces are magnified in direct relationship to the crown height. Bidez and Misch evaluated the effect of a cantilever on an implant and its relation to crown height.[44,45] When a cantilever is placed on an implant, there are six different potential rotation points (i.e., moments) on the implant body (Figure 6-25; Table 6-2). When the crown height is increased from 10 to 20 mm, two of six of these moments are increased 200%. A cantilevered force may be in any direction: facial, lingual, mesial, or distal. Forces cantilevered to the facial and lingual direction are often called *offset loads*. The bone width decrease is primarily from the facial aspect of the edentulous ridge. As a result, implants are often placed more lingual than the center of the natural tooth root. This condition often results in a restoration cantilevered to the facial. When the available bone height is also decreased, the CHS is increased. Therefore the potential length of the implant reduced in excessive CHS conditions, and the implant position results in offset loads.

An angled load to a crown will also magnify the force applied to the implant. A 12-degree force to the implant

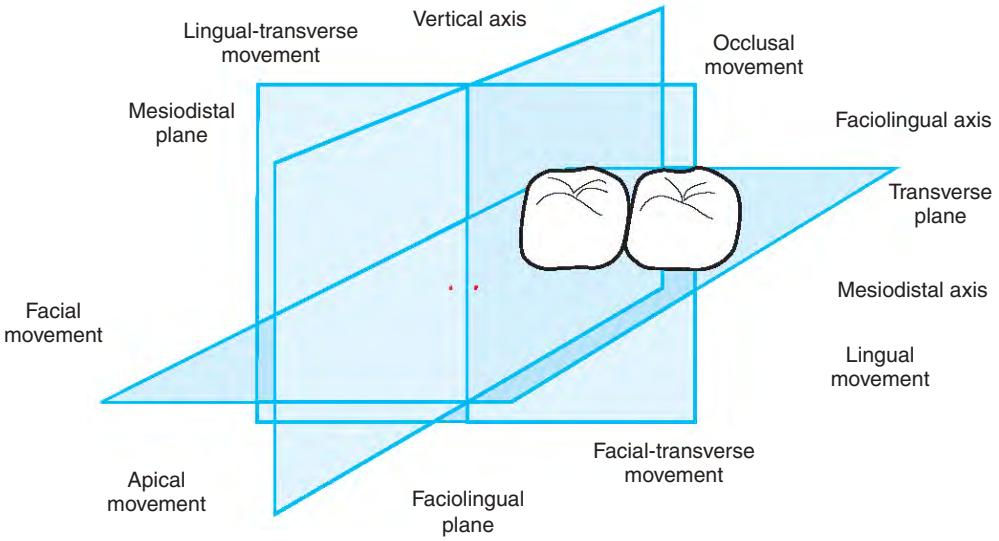


Figure 6-25 Moment loads tend to induce rotations in three planes. Clockwise and counterclockwise rotations in these three planes result in six moments: lingual-transverse, facial-transverse, occlusal, apical, facial, and lingual.

WWW.HIGHDENT.IR

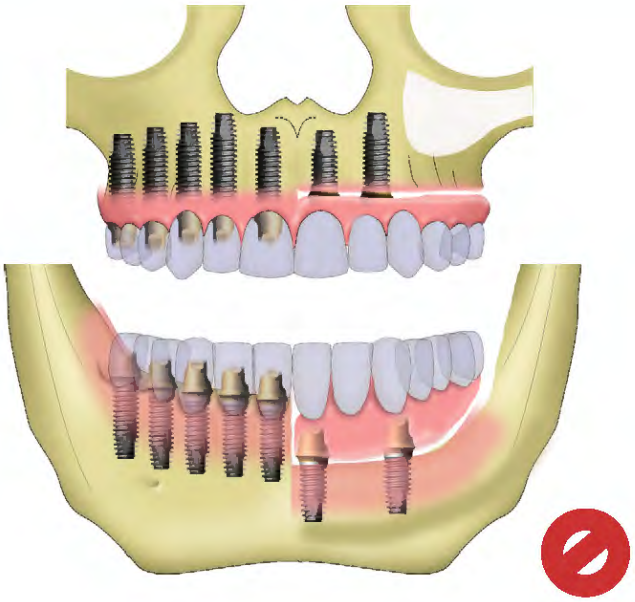


Figure 6-26 In the past, treatment plans included more implants in abundant bone and fewer implants in less available bone. However, crown height increases as bone height decreases, and this approach is creating unfavorable mechanics.

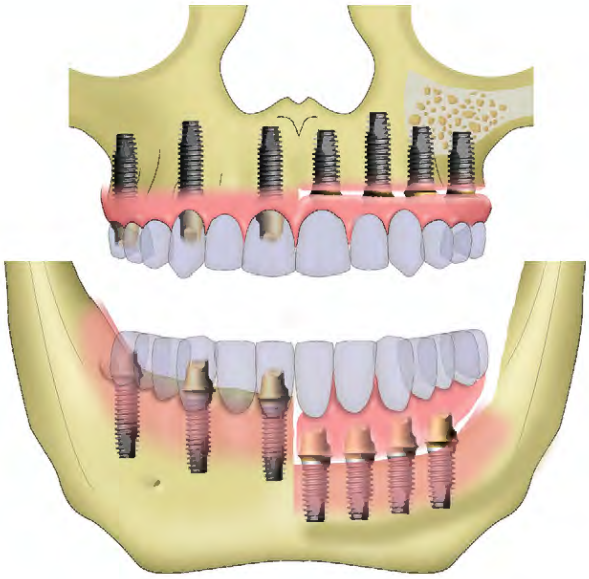


Figure 6-27 Crown height is a force magnifier to any lateral load or horizontal cantilever. Therefore when available bone height decreases, more implants should be inserted and cantilever length reduced.

will be increased by 20%. This increase in force is further magnified by the crown height. For example, a 12-degree angle with a force of 100 N will result in a force of 315 N-mm on a crown height of 15 mm.[44] Maxillary anterior teeth are usually at an angle of 12 degrees or more to the occlusal planes. Even implants placed in an ideal position are usually loaded at an angle. Maxillary anterior crowns are often longer than any other teeth in the arch, so the effects of crown height cause greater risk.

The angled force to the implant also may occur during protrusive or lateral excursions, as the incisal guide angle may be 20 degrees or more. Anterior implant crowns will therefore be loaded at a considerable angle during excursions, compared with the long axis position of the implant. As a result, an increase in the force to maxillary anterior implants should be compensated for in the treatment plan.

Most forces applied to the osteointegrated implant body are concentrated in the crestal 7 to 9 mm of bone, regardless of implant design and bone density.[67] Therefore implant body height is not an effective method to counter the effects of crown height. In other words, crown-root ratio is a prosthetic concept that may guide the restoring dentist when evaluating a natural tooth abutment. The longer the natural tooth root, the shorter the crown height, which acts as a lever to rotate the tooth around an axis located two thirds down the root. However, the crown height–implant ratio is not a direct comparison. Crown height is a vertical cantilever that magnifies any lateral or cantilever force in either a tooth- or an implant-supported restoration. However, this condition is not improved by increasing implant length to dissipate stresses. The implant does not rotate away from the force in relation to implant length. Instead, it captures the force at the crest of the ridge. The greater the CHS, the greater number of implants usually required for the prosthesis, especially in the presence of other force factors. This is a complete paradigm shift to the concepts advocated originally, with many implants in greater available bone and small crown heights and fewer implants with greater crown heights in atrophied bone (Figures 6-26 and 6-27).

The CHS increases when crestal bone loss occurs around the implants. An increased CHS may increase the forces to the crestal bone around the implants and increase the risk of crestal bone loss. This in turn may further increase both the CHS and the moment forces to the entire support system, resulting in screw loosening, crestal bone loss, implant fracture, and implant failure.

The vertical distance from the occlusal plane to the opposing landmark for implant insertion is typically a constant in an individual. Therefore as the bone resorbs, the crown height becomes larger, but the available bone height decreases (Figure 6-28). An indirect relationship is found between the crown and implant height. Moderate bone loss before implant placement may result in a crown height–bone height ratio greater than 1, with greater lateral forces applied to the crestal bone than in abundant bone (in which the crown height is less). A linear relationship exists between the applied load and internal stresses.[68,69] Therefore the greater the load applied, the greater the tensile and compressive stresses transmitted at the

WWW.HIGHDENT.IR

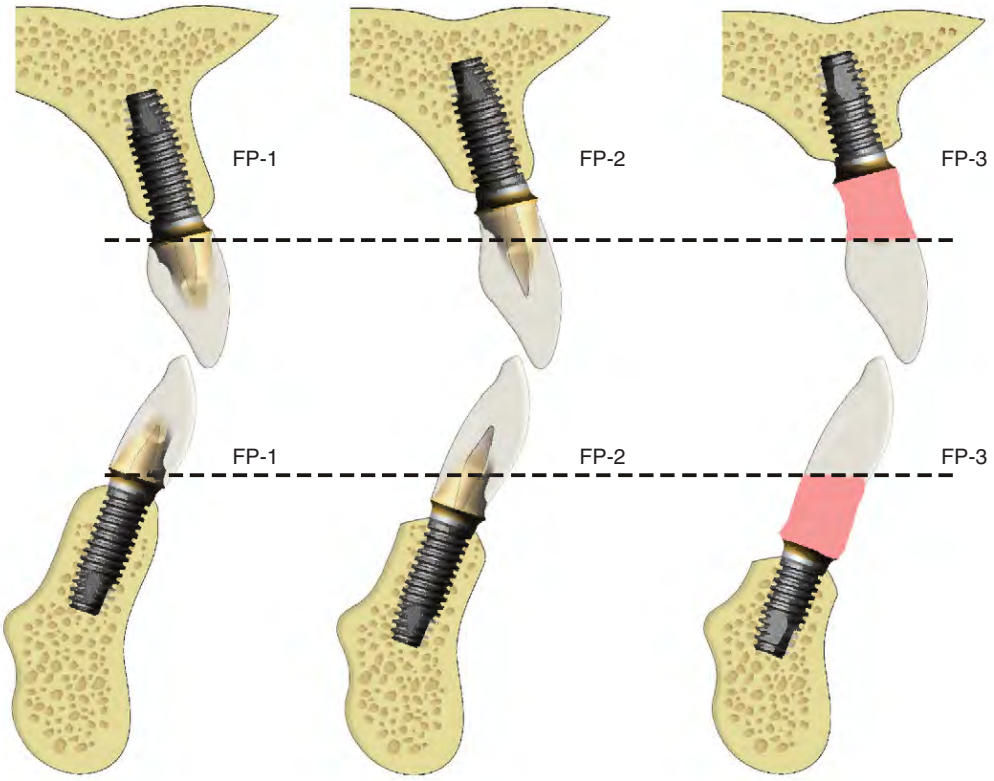


Figure 6-28 As the crown height increases, the available bone height decreases. This is especially noteworthy in the maxillary arch, as the initial available bone height is less than in the mandible. As a consequence, shorter implants in the maxilla are a common occurrence.

Box 6-4 Excessive Crown Height Space

1. Excessive CHS increases mechanical complications in fixed prostheses.
2. The need for gingival replacement procedures should be evaluated before implant placement for fixed restorations.
3. Metal and porcelain shrinkage is more of a problem in traditional fixed prosthetic cases.
4. Hybrid FP-3 fixed prostheses with denture teeth, metal substructure, and acrylic resin are indicated.
5. Overdentures in completely edentulous patients with RP-4 and RP-5.
6. The implant support for RP-4 should be as great as that for a fixed prosthesis.
7. With mobile overdentures, there should be adequate soft tissue support when designing RP-5.
8. In overdentures, there may be two different components of the CHS: the distance from the crest of the bone to the height of the attachment and the distance from the attachment to the occlusal plane.

CHS, Crown height space.

bone interface and to the prosthetic components. And yet, many implant treatment plans are designed with more implants in abundant bone situations and fewer implants in atrophied bone volume. The opposite scenario should exist. The lesser the bone volume, the greater the crown height, and the greater the number of implants indicated.

Excessive CHS

Crown height space greater than 15 mm is excessive; it is primarily the result of the vertical loss of alveolar bone from long-term edentulism. Other causes may include genetics, trauma, and implant failure (Box 6-4). Treatment of excessive CHS before implant placement includes orthodontic and surgical methods. Orthodontics in partially edentulous patients is the method of choice, as other surgical or prosthetic methods are usually more costly and have greater risks of complications. Several surgical techniques may also be considered, including block onlay bone grafts, particulate bone grafts with titanium mesh or barrier membranes, interpositional bone grafts, and distraction osteogenesis. A staged approach to reconstruction of the jaws is often preferred to simultaneous implant placement, especially when large-volume gains are required. Significant vertical bone augmentation may even require multiple surgical procedures.

Distraction osteogenesis has several advantages over onlay bone grafting techniques for vertical bone growth. Vertical bone gains are not limited by factors such as graft size or expansion of the existing soft tissue volume. There is no donor site morbidity, and the surgery may

WWW.HIGHDENT.IR

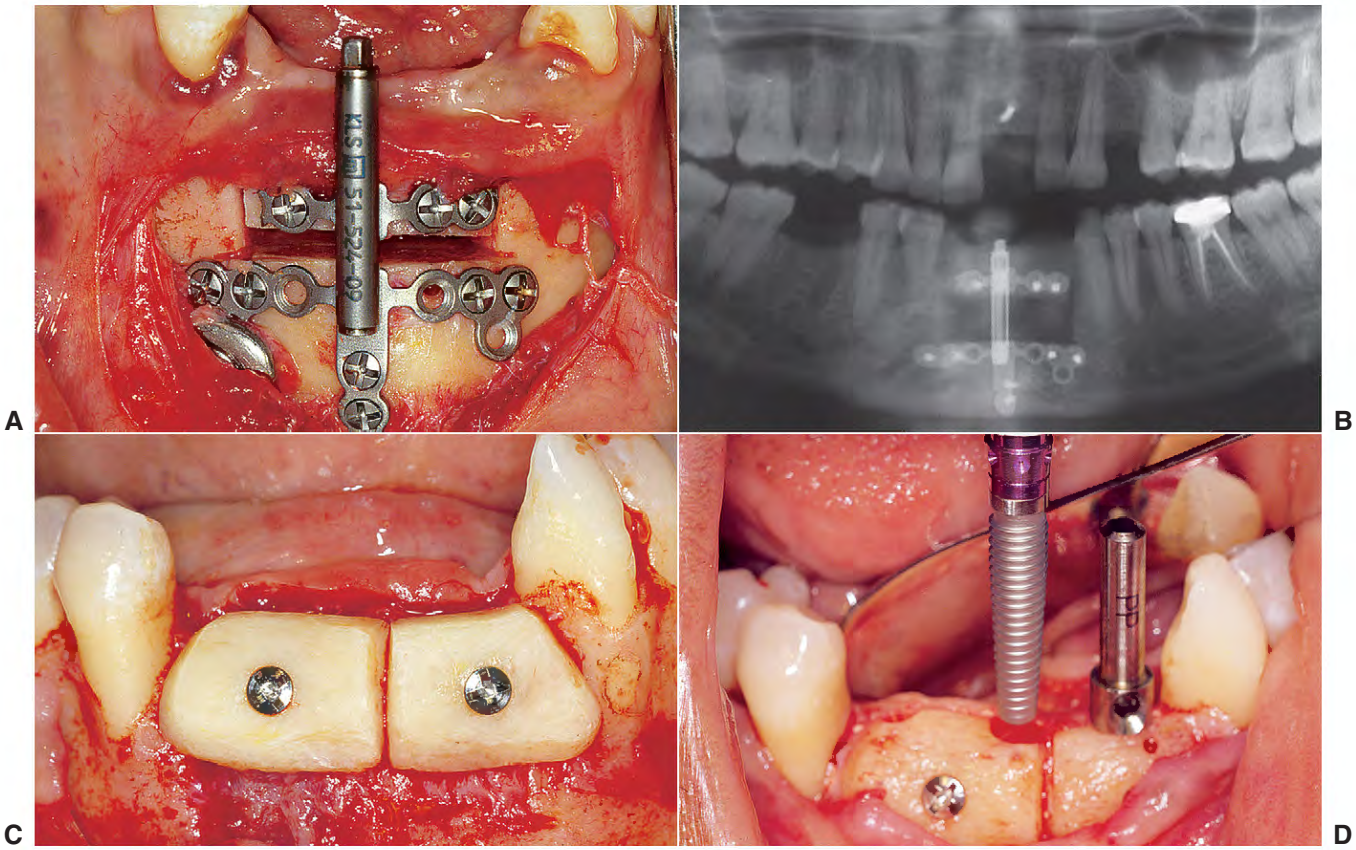


Figure 6-29 **A,** An anterior atrophic mandible with a distraction osteogenesis device to gain vertical bone height between the canines. **B,** A panoramic radiograph of the distracted anterior mandible. **C,** An onlay bone graft from the ramus is used to gain horizontal bone volume. **D,** Anterior endosteal implants are placed after distraction and onlay bone grafts have matured. (*Courtesy Craig Misch, Sarasota, Fla.*)

be performed in an office setting. However, distraction osteogenesis requires patient compliance, and bone volume gains are unidirectional. In addition, clinical studies found that secondary bone augmentation procedures are often required for dental implant placement.[70] Misch presented a unique approach combining vertical distraction and horizontal onlay bone grafting to reconstruct the deficiency three-dimensionally. Osseous distraction is performed first to vertically increase the ridge and expand the soft tissue volume. Secondarily an onlay bone graft is used to complete the repair of the defect (Figure 6-29).[65]

In case of excessive CHS, bone augmentation may be preferred to prosthetic replacement. Surgical augmentation of the residual ridge height will reduce the CHS and improve implant biomechanics. Augmentation will often permit the placement of wider body implants with the associated benefit of increased surface area. Although prosthetics is the most commonly used option to address excess CHS, it should be the last choice. Using gingival colored prosthetic materials (pink porcelain or acrylic resin) on fixed restorations or changing the prosthetic design to a removable restoration should often be considered when restoring excessive CHS.

In the maxilla, a vertical loss of bone results in a more palatal ridge position. As a consequence, implants are often inserted more palatal than the natural tooth position. Removable restorations have several advantages under these clinical circumstances. The removable prosthesis does not require embrasures for hygiene. The removable restoration may be removed during sleep to decrease the effects of an increase in CHS on nocturnal parafunction. The removable restoration may improve the lip and facial support, which is deficient because of the advanced bone loss. The overdenture may have sufficient bulk of acrylic resin to decrease the risk of prosthesis fracture. The increase in CHS permits denture tooth placement without infringement of the substructure.

Soft tissue support in addition to implant-supported removable implant restorations with an excessive CHS are recommended when it is not possible to overengineer the implant support system. A rigid overdenture has identical requirements to a fixed prosthesis, because it is rigid during function. Misch describes the "hidden cantilever" beyond the cantilevered bar with a rigid implant overdenture.[71] When the overdenture has no movement during function, the cantilever does not stop at the end of the cantilevered substructure but ends at the last occlusal contact position on the prosthesis, often the distal of a second molar.

The position and type of overdenture attachments may render an overdenture rigid during function, even

WWW.HIGHDENT.IR

Figure 6-30 When the crown height space is greater than 12 mm, pink porcelain (or acrylic) is often used to replace the soft tissue drape in the prosthesis.

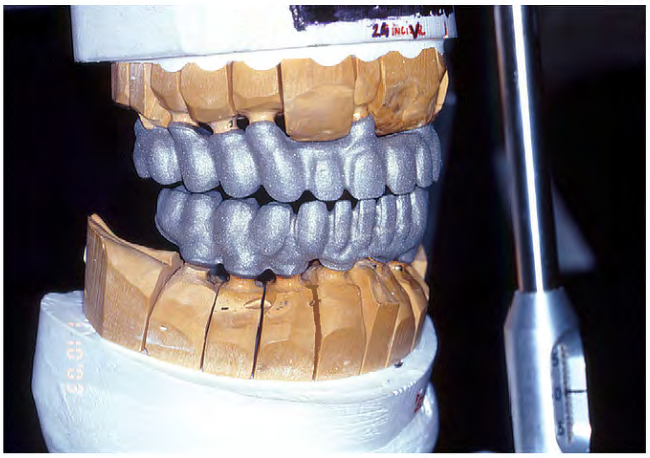


Figure 6-31 Porcelain thickness for fixed prostheses should not be greater than 2 mm. When the crown height space is greater than 15 mm, the amount of metal in the substructure may be extensive.

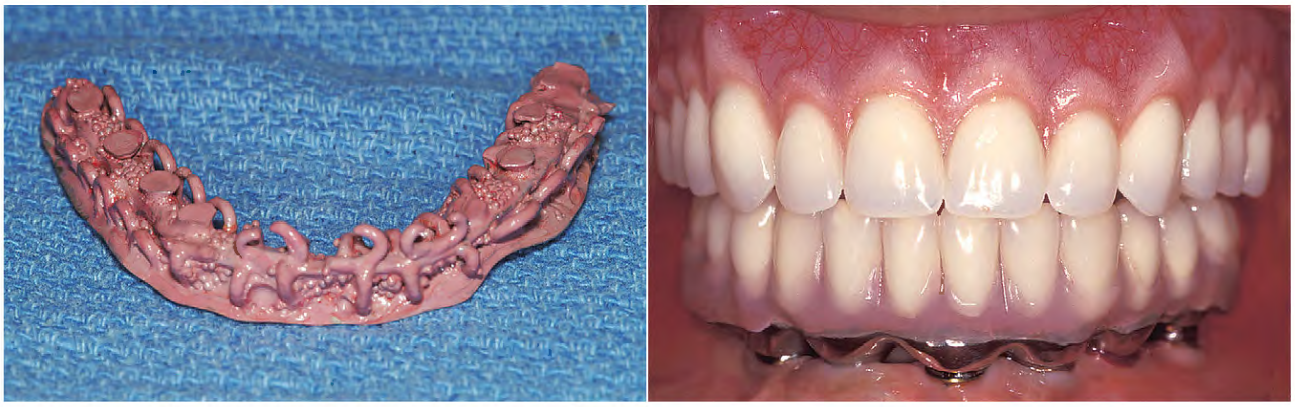
in the absence of distal cantilevers on the bar. For example, when three anterior implants are splinted together and a Hader clip is used to retain the prosthesis, if the Hader clips are placed at angles to the midline, the attachments have limited movement and result in a rigid overdenture during function. Misch suggests the prosthesis movement, not the individual attachment movement, should be evaluated.[71] Excessive CHS with overdentures are situations that benefit from a prosthesis designed to have more than one direction of movement.

The ideal CHS for a fixed prosthesis is between 8 and 12 mm, accounting for an ideal 3 mm of soft tissue, 2 mm of occlusal material thickness, and a 5-mm or greater abutment height. A CHS greater than 12 mm may be of concern in fixed restorations. The replacement teeth are elongated and often require the addition of gingival tone materials in esthetic regions (Figure 6-30). The greater impact force on implants compared with teeth, combined with the increased crown height, creates increased moment forces on implants and risks of component and material fracture. These problems are especially noted when associated with less favorable biomechanics on cantilevered sections of fixed restorations.[39,66]

A CHS greater than 15 mm means a large amount of metal must be used in the substructure of a traditional fixed restoration to keep porcelain to its ideal 2-mm thickness (Figure 6-31). Fine-tuning techniques for traditional fixed restorations allowed Dabrowsky to manufacture and monitor multiple full-mouth, cement-retained prostheses with a large CHS, delivered in various centers across the United States.[72] Controlling surface porosities of metal substructures after casting as their different parts cool at different rates becomes increasingly difficult. Furthermore, when the casting is reinserted into the oven to bake the porcelain, the heat is maintained within the casting at different rates, so the porcelain cools in different regions at different rates.[73] If not controlled properly, both these factors increase the risk of porcelain fracture after loading.[74] For excessive CHS, considerable weight of the prosthesis (approaching 3 oz of alloy) may affect maxillary trial placement appointments, because the restoration does not remain in place without the use of adhesive. Noble metals must be used to control alloy's heat expansion or corrosion; therefore the costs of such implant restorations have dramatically increased. Proposed methods to produce hollow frames to alleviate these problems, including the use of special custom trays to achieve a passive fit will double or triple the labor costs.[75]

An alternative method of fabricating fixed prostheses in situations of CHS of 15 mm or greater is the fixed complete denture or hybrid prosthesis, with a smaller metal framework, denture teeth, and acrylic resin to join these elements together (Figure 6-32). The reduced metal framework compared with a porcelain-to-metal fixed prosthesis exhibits fewer dimensional changes and may more accurately fit the abutments, which is important for a screw-retained restoration. It is less expensive to fabricate than a porcelain-to-metal fixed prosthesis, is highly esthetic (premade denture teeth), easily replaces teeth and soft tissue in appearance, and is easier to repair if fracture occurs. Because resin acts as an intermediary between the teeth and metal substructure, the impact force during dynamic occlusal loading may also be reduced. Therefore this type of fixed prosthesis is often indicated for implant restorations with a large CHS. On occasion, undercontoured interproximal areas are designed by the laboratory in such restorations to assist oral hygiene and have been referred to as "high water" restorations. This is an excellent method in the mandible; however, it results in food entrapment, affects air flow patterns, and may contribute to speech problems in the anterior maxilla.

WWW.HIGHDENT.IR



A B

Figure 6-32 **A,** A metal framework for a hybrid prosthesis composed of metal, acrylic, and denture teeth presents several advantages for fixed prostheses with a CHS greater than 15 mm. **B,** Denture teeth are then added to the metal substructure.

Because an increase in the biomechanical forces are in direct relationship to the increase in CHS, the treatment plan of the implant restoration should consider stress reducing options whenever the CHS is increased. Methods to decrease stress include[63-65]:

1. Shorten cantilever length
2. Minimize offset loads to the buccal or lingual
3. Increase the number of implants
4. Increase the diameters of implants
5. Design implants to maximize the surface area of implants
6. Fabricate removable restorations that are less retentive and incorporate soft tissue support
7. Remove the removable restoration during sleeping hours to reduce the noxious effects of nocturnal parafunction
8. Splint implants together, whether they support a fixed or removable prosthesis

Because CHS is a considerable force magnifier, the greater the crown height, the shorter the prosthetic cantilever that should extend from the implant support system. In CHS greater than 15 mm, no cantilever should be considered, unless all other force factors are minimal. The occlusal contact intensity should be reduced on any offset load from the implant support system. Occlusal contacts in centric relation occlusion may even be eliminated on the most posterior aspect of a cantilever. In this way, a parafunction load may be reduced, as the most cantilevered portion of the prosthesis is only loaded during functional activity (such as chewing).

MASTICATORY DYNAMICS

Masticatory muscle dynamics are responsible for the amount of force exerted on the implant system. Several criteria are included under this heading: patient size, gender, age, and skeletal position.[9,16,11,21,76-79] The size of the patient can influence the amount of bite force. Large, athletic men can generate greater forces; patients of weak physical condition often develop less force than athletic patients (Figure 6-33). In general, the forces recorded in women are 20 lb less than those in men. In a clinical report by van Steenberghe et al., partially edentulous men have a 13% implant failure rate compared with women with a 77% failure rate.[80] In a report by Wyatt and Zarb, first-year radiograph bone loss was positively correlated with males, younger patients, and implants supporting a distal extension prosthesis.[81] Older patients record lower bite forces than young adults. In addition, the younger patient lives longer and requires the additional implant support for the prosthesis for a longer time. (An 80-year-old patient will need implant support for far fewer years than a 20-year-old, all other factors being equal.)

Figure 6-33 Masticatory dynamics are affected by the size of the patient (larger persons generally have greater bite forces).

The skeletal arch position may influence the amount of maximum bite force. The brachiocephalic, with a

WWW.HIGHDENT.IR

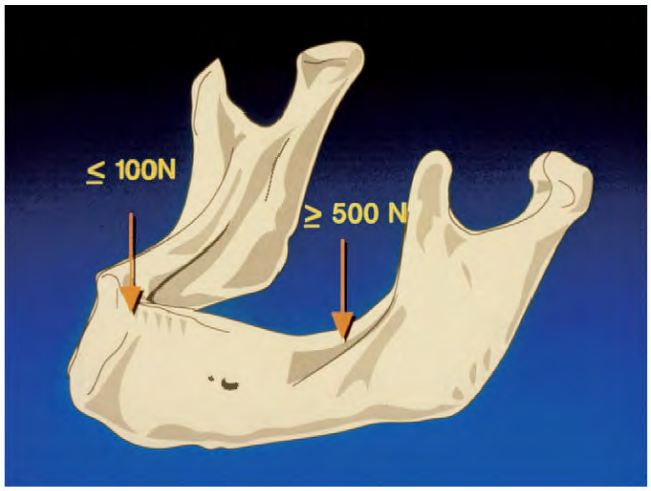


Figure 6-34 The maximum bite forces are greater in the posterior regions of the jaws compared with the anterior regions.

stout head shape, may generate three times the bite force compared with a regular head shape. This is especially noteworthy when accompanied by moderate to severe bruxism or clenching. The maximum bite force decreases as muscle atrophy progresses throughout years of edentulism. A maximum occlusal force of 5 psi may be the result of 15 years without teeth.[77] This force may increase 300% in the 3 years after implant placement.[21,22,77-79] Therefore sex, muscle mass, exercise, diet, state of the dentition, physical status, and age all influence muscle strength, masticatory dynamics, and maximum bite force.

The skeletal Class III patient is primarily a vertical chewer and generates vertical forces with little excursive movement. However, some patients appear "pseudo-Class III" as a result of anterior bone resorption or loss of posterior support and collapse of the vertical dimension with an anterior rotation of the mandible. These patients do exhibit lateral excursive movements when the incisal edge position is restored to its initial position.

As a general rule, the implant treatment plan should reduce other force magnifiers when masticatory musculature dynamics increase. For example, a cantilever length should be reduced in cases of elevated masticatory dynamics. A crown height may be reduced by bone augmentation. The prosthesis may be made removable so nocturnal bruxism is reduced (if they do not wear their prosthesis). The implant number, size, and design may also be increased to increase the surface area of load.

ARCH POSITION

The maximum biting force is greater in the molar region and decreases as measurements progress anteriorly. Maximum bite forces in the anterior incisor region correspond to approximately 35 to 50 psi; those in the canine region range from 47 to 100 psi; whereas those in the molar area vary from 127 to 250 psi[8] (Figure 6-34). Mansour et al. evaluated occlusal forces and moments mathematically using a Class III lever arm, the condyles being the fulcrum and the masseter and temporalis muscles supplying the force.[82] Similar figures were obtained by mathematical calculation and by direct measurement. In addition, the forces at the second molar were 10% higher than at the first molar, indicative of a range from 140 to 275 psi.

In a study by Chung et al. with 339 implants in 69 patients in function for an average of 8.1 years (range of 3 to 24 years), the posterior implants (even with keratinized mucosa) showed a 3.5-fold greater average bone loss per year than anterior implants.[83]

The anterior biting force is decreased in the absence of posterior tooth contact and greater in the presence of posterior occlusion or eccentric contacts.[35,84] Besides the mechanical properties of a Class III lever function, there also is a biological component to increased bite force in the posterior regions. When the posterior teeth are in contact, the large masticatory muscles contract. When the posterior teeth are not in contact, two thirds of the temporalis and masseter muscles do not contract their fibers. As a consequence, the bite force is reduced.

In the anterior regions with less force, the anterior natural tooth roots are smaller in diameter and root surface area compared with posterior teeth. Yet in implant dentistry, we often alter the implant length primarily and place longer implants in the anterior region and shorter implants in the posterior regions (or cantilever off the anterior implants, which results in posterior bite forces magnified by the cantilever length). This approach should be corrected to conform to biomechanics similar to that observed with natural teeth. In other words, implants in the posterior regions should often be of greater diameter, especially in the presence of additional force factors. The greater increase in natural tooth surface area occurs in the molar region, with a 200% increase compared with the premolars. Hence, the larger implant diameter is especially considered in the molar region.

The edentulous bone density varies in function of arch position. The natural teeth are surrounded by a thin cortical plate of bone and periodontal complex, which is similar for all teeth and arch positions. However, after the teeth are lost, the bone density in the edentulous site is different for each region of the mouth. The posterior regions, in general, form less bone density after tooth loss than the anterior regions. The mandibular anterior implant sites benefit from denser bone than the maxillary anterior implant sites. The denser the bone, the greater its resistance to stress applied at the implant-bone interface. In other words, the edentulous bone density is inversely related to the amount of force generally applied in that arch position. As a result, the posterior

WWW.HIGHDENT.IR

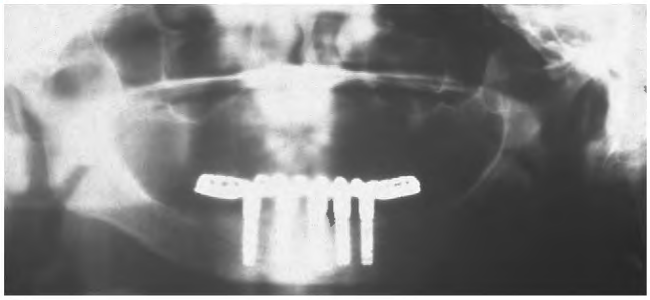


Figure 6-35 As a general rule, the greater the number of opposing teeth, the greater the bite force. When the opposing arch is a denture (as depicted in this panoramic radiograph in the maxilla), the bite forces are less.

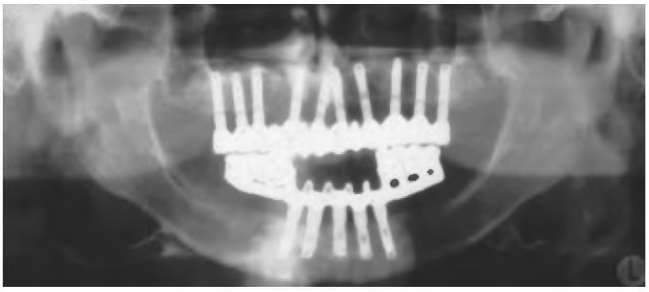


Figure 6-37 When the opposing arch has a fixed implant prosthesis, the bite forces are greatest. The decrease in proprioception results in higher forces during function and parafunction. In this patient, the cantilever should be reduced significantly, especially because the crown height is also large.

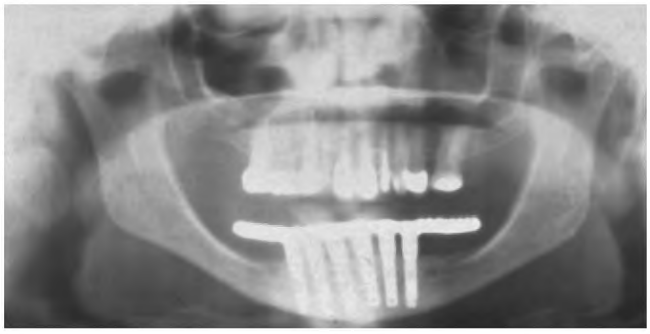


Figure 6-36 The opposing arch consists of natural teeth in this panoramic radiograph. The cantilever length should be reduced or the number of implants increased, compared with an edentulous opposing arch.

Figure 6-38 The opposing arches in this patient are complete implant-supported prostheses. As a general rule, no posterior cantilevers are designed in the maxilla and limited cantilevers are designed in the mandible.

maxilla is the most at-risk arch position, followed by the posterior mandible, then the anterior maxilla. The most ideal region is the mandibular anterior.

OPPOSING ARCH

Natural teeth transmit greater impact forces through occlusal contacts than soft tissue–borne complete dentures. In addition, the maximum occlusal force of patients with complete dentures is limited and may range from 5 to 26 psi.[77] The force is usually greater in recent denture wearers and decreases with time. Muscle atrophy, thinning of the oral tissues with age or disease, and bone atrophy often occur in the edentulous patient as a function of time.[85] Some denture wearers may clench on their prosthesis constantly, which may maintain muscle mass. However, this condition usually accelerates bone loss. Implant overdentures improve the masticatory performance and permit a more consistent return to centric relation occlusion during function. The maximum force generated in an implant prosthesis is related to the amount of tooth or implant supporting the opposing arch[12,77,78] (Figures 6-35 and 6-36).

A complete implant fixed prosthesis does not benefit from proprioception as do natural teeth, and patients bite with a force four times greater than with natural teeth. Thus, the highest forces are created with implant prostheses (Figure 6-37). In addition, premature contacts in occlusal patterns or during parafunction on the implant prostheses do not alter the pathway of closure, as occlusal awareness is decreased with implant prostheses when compared with natural teeth.[17,18] Therefore continued stress increases can be expected to occur with the implant restoration.

Partial denture patients may record forces intermediate between that of natural teeth and complete dentures, depending on the location and condition of the remaining teeth, muscles, and joints. In the partially edentulous patient with implant-supported fixed prostheses, force ranges are more similar to those of natural dentition, but lack of proprioception may magnify the load amount during parafunctional activity.

As a consequence of the opposing arch affecting the intensity of forces applied to an implant prosthesis, the treatment plan may be modified to reduce the risk of overload. Rarely should the opposing arch be maintained in a traditional denture to decrease the stress to the implant arch. Instead, the implant arch should be designed to compensate for the higher stresses expected from an implant-supported opposing arch (Figure 6-38).

WWW.HIGHDENT.IR

SUMMARY

Patient force factors are highly variable from one person to another. An implant foundation should be designed to support the load and resist the stresses while the restoration is in service. An ideal treatment plan may be established relative to the number and position of missing teeth. The treatment plan is then modified dependent on the force factors of the individual patient.

It is far better to overengineer the amount of support necessary for a prosthesis. If just one too few or too small an implant is used, implant bone loss, fracture, and failure may occur. As a general rule, the best way to reduce the risk of biomechanical overload is to add an implant.

The five most important force factors related to patient conditions were presented in this chapter. Of these, parafunction is the predominant element to account for in the treatment plan. On a scale of 1 to 10, severe bruxism is a 10; an excessive CHS can double a force, and therefore is a 7 on the importance scale. Severe masticatory dynamics can also double a force component and result in a 7 on this scale. Position of the abutment in the arch determines the magnitude of force and is a 1 or 2 when in the mandibular anterior region, a 3 or 4 in the maxillary anterior, a 5 in the posterior mandible, and a 6 or 7 in the posterior maxilla (as bone density is most ideal in the anterior mandible and least biomechanically favorable in the posterior maxilla). Direction of load under ideal implant placement conditions is a factor of 3 or 4 in the maxillary anterior regions. The other arch positions may have a more ideal direction of load, unless cantilever loads are positioned on the implant restoration. The opposing arch under typical treatment conditions is the least important force component modifier. A complete implant restoration may be a factor of 3, natural teeth a 2, and an opposing soft tissue–supported denture a 1.

References

1. Picton DC, Johns RB, Wills DJ et al: The relationship between the mechanisms of tooth and implant support, *Oral Sci Rev* 5:3-22, 1971.
2. Picton DC: The effect of external forces on the peridontium. In Melcher AH, Bowen WH, editors: *Biology of the periodontium,* London, 1969, Academic Press.
3. Scott I, Ash MM Jr: A six-channel intra-oral transmitter for measuring occlusal forces, *J Prosthet Dent* 16:56, 1966.
4. Graf H: Bruxism, *Dent Clin North Am* 13:659-665, 1969.
5. Proffit WR: The facial musculature in its relation to the dental occlusion. In Carlson DS, McNamara JA, editors: *Muscle adaptation in the craniofacial region.* Proceedings of symposium, Craniofacial Growth Series, Monograph 8, Ann Arbor, Mich, 1978, University of Michigan.
6. Black GV: An investigation of the physical characters of the human teeth in relation to their diseases, and to practical dental operations, together with the physical characters of filling materials, *Dent Cosmos* 37:469, 1895.
7. Craig RG: *Restorative dental materials,* ed 6, St Louis, 1980, Mosby.
8. Carlsson GE: Bite force and masticatory efficiency. In Kawamura Y, editor: *Physiology of mastication,* Basel, Switzerland, 1974, Karger.
9. Ingervall B, Helkimo E: Masticatory muscle force and facial morphology in man, *Arch Oral Biol* 23:203-206, 1978.
10. Helkimo E, Carlsson GE, Helkimo M: Bite force and state of dentition, *Acta Odontol Scand* 35:297-303, 1977.
11. Lassila V, Holmlund J, Koivumaa KK: Bite forces and its correlations in different denture types, *Acta Odontol Scand* 43:127-132, 1985.
12. Haraldson T, Carlsson GE: Bite force and oral function in patients with osseointegrated implants, *Scand J Dent Res* 85:200-208, 1977.
13. van Eijden TM: Three dimensional analyses of human bite force magnitude and moment, *Arch Oral Biol* 36: 535-539, 1991.
14. Lindquist LW, Carlsson GE: Long-term effects on chewing with mandibular fixed prostheses on osseointegrated implants, *Acta Odontol Scand* 43:39-45, 1985.
15. Lundgren D, Laurell L, Falk J et al: Distribution of occlusal forces in a dentition unilaterally restored with a bridge construction supported on osseointegrated titanium implants. In van Steenberghe D, editor: *Tissue integration in oral and maxillo-facial reconstruction,* Brussels, 1985, Excerpta Medica.
16. Braun S, Bantleon HP, Hnat WP et al: A study of bite force. Part I: relationship to various physical characteristics, *Angle Orthod* 65:367-372, 1995.
17. Haraldson T, Zarb GA: A 10-year follow-up study of the masticatory system after treatment with osseointegrated implant bridges, *Scand J Dent Res* 96:243-252, 1988.
18. Falk J, Laurell L, Lundgren D: Occlusal interferences and cantilever joint stress in implant supported prostheses occluding with complete dentures, *Int J Oral Maxillofac Impl* 5:70-77, 1990.
19. Richter E-J: In vivo vertical forces on implants, *Int J Oral Maxillofac Impl* 10:99-108, 1995.
20. Falk H: On occlusal forces in dentitions with implants supported fixed cantilever prostheses. *Swed Dent J* 69: 1-40, 1990.
21. Raadsheer MC, van Eijden TM, van Ginkel FC et al: Contribution of jaw muscle size and craniofacial morphology to human bite force magnitude, *J Dent Res* 87:31-42, 1999.
22. Morneburg TR, Proschel PA: Measurement of masticatory forces and implant loads: a methodologic clinical study, *Int J Prosthodont* 15:20-27, 2002.
23. Ramfjord SP, Ash MM: *Occlusion,* ed 4, Philadelphia, 1995, WB Saunders.
24. Nadler SC: Bruxism, a clinical and electromyographic study, *J Am Dent Assoc* 62:21, 1961.
25. Alderman MM: Disorders of the temporomandibular joint and related structures. In Burket LW, editor: *Oral medicine,* ed 6, Philadelphia, 1971, JB Lippincott.
26. Jaffin R, Berman C: The excessive loss of Brånemark fixtures in type IV bone: a 5-year analysis, *J Periodontol* 62:2-4, 1991.

WWW.HIGHDENT.IR

27. Fischer WF, O'Toole ET: Personality characteristics of chronic bruxers, *Behav Med* 19:82-86, 1993.
28. Lavigne GJ, Montplaisir JY: Restless legs syndrome and sleep bruxism: prevalence and association among Canadians, *Sleep* 17:739-743, 1994.
29. Glass EG, McGlynn FD, Glaros AG et al: Prevalence of TM disorder symptoms in a major metropolitan area, *Cranio* 11:217-220, 1993.
30. Ohayon MM, Li KK, Guilleminault C: Risk factors for sleep bruxism in the general population *Chest* 119: 453-461, 2002.
31. Tosun T, Krabuda C, Cuhadaroglu C et al: Evaluation of sleep bruxism by polysomnographic analysis in patients with dental implants, *Int J Oral Maxillofac Imp* 18: 286-292, 2003.
32. Thorpy MD, Broughton RJ, Cohn MA et al, editors: *The international classification of sleep disorders, revised: diagnostic and coding manual,* Westchester, Ill, 2001, American Academy of Sleep Medicine.
33. Gibbs CH, Mahan PE, Mauderli A et al: Limits of human bite force, *J Prosthet Dent* 56:226-229, 1986.
34. Glaros AG, Rao SM: Effects of bruxism: a review of the literature, *J Prosthet Dent* 38:149-157, 1977.
35. Williamson EH, Lundquist DO: Anterior guidance: its effect on electromyographic activity of temporal and masseter muscles, *J Prosthet Dent* 49:816-823, 1983.
36. Del Valle V, Faulkner G, Walfaardt J: Craniofacial osseointegrated implant-induced strain distribution: a numerical study, *Int J Oral Maxillofac Impl* 12: 200-210, 1997.
37. Ishigaki S, Nakano T, Yamada S et al: Biomechanical stress in bone surrounding an implant under simulated chewing, *Clin Oral Implant Res*14:97-102, 2002.
38. Oh T, Yoon J, Misch CE et al: The cause of early implant bone loss: myth or science? *J Periodontol* 73: 322-333, 2002.
39. Bragger U, Aeschlimann S, Burgin W et al: Biological and technical complications and failures with fixed partial dentures (FPD) on implants and teeth after four to five years of function, *Clin Oral Impl Res* 12:26-43, 2001.
40. Misch CE, Palattella A: Bruxism and its effect on treatment plans, *Int Mag Oral Implant* 2:6-18, 2002.
41. Mericske-Stern R, Assal P, Buergin W: Simultaneous force measurements in three dimensions on oral endosseous implants in vitro and vivo: a methodological study, *Clin Oral Implants Res* 7:378-386, 1996.
42. Choy E, Kydd WL: Bite force duration: a diagnostic procedure for mandibular dysfunction, *J Prosthet Dent* 60:365-368, 1988.
43. Bidez MW, Misch CE: Force transfer in implant dentistry: basic concepts and principles, *Oral Implantol* 18:264-274, 1992.
44. Bidez MW, Misch CE: Issues in bone mechanics related to oral implants, *Implant Dent* 1:289-294, 1992.
45. Misch CE, Bidez MW: Biomechanics in implant dentistry. In Misch CE, editor: *Contemporary implant dentistry*, St Louis, 1993, Mosby.
46. Dawson PE: *Differential diagnosis and treatment of occlusal problems,* ed 2, St Louis, 1989, Mosby.
47. Rateitschak KJ, editor: *Color atlas of dental medicine,* Stuttgart, 1989, Thieme.
48. Sheikholescham A, Riise C: Influence of experimental interfering occlusal contacts on the activity of the anterior temporal and masseter muscles during submaximal and maximal bite in the intercuspal position, *J Oral Rehabil* 10:207-214, 1983.
49. Misch CE: Clenching and its effects on implant treatment plans, *Oral Health* 92:11-24, 2002.
50. Tanaka TT: Recognition of the pain formula for head, neck, and TMJ disorders: the general physical examination, *Calif Dent Assoc J* 12:43-49, 1984.
51. Mezitis M, Rallis G, Zachariatides N: The normal range of mouth opening, *J Oral Maxillofac Surg* 47:1028-1029, 1989.
52. McCoy G: The etiology of gingival erosion, *J Oral Implantol* 10:361-362, 1982.
53. Selna LG, Shillingburg HT Jr, Kerr PA: Finite element analysis of dental structure asymmetric and plane stress idealizations, *J Biomed Mater Res* 9:235-237, 1975.
54. Hood JAA: Experimental studies on tooth deformation: stress distribution in Class V restorations, *N Z Dent J* 68:116-131, 1968.
55. DuPont GA, DeBowers LJ: Comparison of periodontics and root replacement in cat teeth with resorptive lesions, *J Vet Dent* 19:71-75; erratum in *J Vet Dent* 19:230, 2002.
56. Hand ASJ, Hunt A, Reinhardt JW: The prevalence and treatment implications of cervical abrasion in the elderly, *Gerodontics* 2:167-170, 1986.
57. Grippo JO: Abfractions: a new classification of hard tissue lesions of teeth, *J Esthet Dent* 3:14-19, 1991.
58. Perel M: Parafunctional habits, nightguards, and root form implants, *Implant Dent* 3:261-263, 1994.
59. Misch CE, Bidez MW: Implant protected occlusion, a biomechanical rationale, *Compend Cont Educ Dent* 15:1330-1342, 1994.
60. Kydd WL, Toda JM: Tongue pressures exerted on the hard palate during swallowing, *J Am Dent Assoc* 65:319, 1962.
61. Winders RV: Forces exerted on the dentition by the peri-oral and lingual musculature during swallowing, *Angle Orthod* 28:226, 1958.
62. The glossary of prosthodontic terms, *J Prosthet Dent* 81: 39-110, 1999.
63. Misch CE, Misch-Dietsh F: Pre-implant prosthodontics. In Misch CE, editor: *Dental implant prosthetics,* St Louis, 2005, Mosby.
64. Misch CE, Goodacre CJ, Finley JM et al: Consensus conference panel report: crown-height space guidelines for implant dentistry—part 1, *Implant Dent* 14:312-318, 2005.
65. Misch CE, Goodacre CJ, Finley JM et al: Consensus conference panel report: crown-height space guidelines for implant dentistry—part 2, *Implant Dent* 15:113-121, 2006.
66. Goodacre CJ, Bernal G, Rungcharassareng K et al: Clinical complications with implants and implant prostheses. *Prosthet Dent* 90:121-132, 2003.
67. Misch CE, Bidez MW: Occlusion and crestal bone resorption: etiology and treatment planning strategies for implants. In McNeill C, editor: *Science and practice of occlusion*, Chicago, 1997, Quintessence.
68. Kakudo Y, Amano N: Dynamic changes in jaw bones of rabbit and dogs during occlusion, mastication, and swallowing, *J Osaka Univ Dent Soc* 6:126-136, 1972.

WWW.HIGHDENT.IR

69. Kakudo Y, Ishida A: Mechanism of dynamic responses of the canine and human skull due to occlusal, masticatory, and orthodontic forces, *J Osaka Univ Dent Soc* 6:137-144, 1972.
70. Jensen OT, Cockrell R, Kuhlke L et al. Anterior maxillary alveolar distraction osteogenesis: a prospective 5-year clinical study, *Int J Oral Maxillofac Implants,* 17:507-516, 2002.
71. Misch CE: Screw-retained versus cement-retained implant-supported prostheses, *Pract Perio Esthet* 7:15-18, 1995.
72. Dabrowsky T: Personal communication, 2005.
73. Bidger DV, Nicholls JI: Distortion of ceramometal fixed partial dentures during the firing cycle, *J Prosthet Dent* 45:507-514, 1981.
74. Bertolotti RL, Moffa JP: Creep rate of porcelain-bonding alloys as a function of temperature, *J Dent Res* 59: 2062-2065, 1980.
75. Bryant RA, Nicholls JI: Measurement of distortion in fixed partial dentures resulting from degassing, *J Prosthet Dent* 42:515-520, 1979.
76. Howell AH, Bruderold F: Vertical forces used during chewing of food, *J Dent Res* 29:133, 1950.
77. Carr AB, Laney WR: Maximum occlusal forces in patients with osseointegrated oral implant prostheses and patients with complete dentures, *Int J Oral Maxillofac Impl* 2: 101-108, 1987.
78. Carlsson GE, Haraldson T: Functional response in tissue-integrated prostheses osseointegration. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Clinical dentistry,* Chicago, 1985, Quintessence.
79. Fontijn-Tekamp FA, Slageter AP, van't Hof MA et al: Bite forces with mandibular implant-retained overdentures, *J Dent Res* 77:1832-1839, 1998.
80. van Steenberghe D, Lekholm U, Bolender C et al: Applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study on 558 fixtures, *Int J Oral Maxillofac Implants* 5:272-281, 1990.
81. Wyatt CC, Zarb Z: Bone level changes proximal to oral implants supporting fixed partial prostheses, *Clin Oral Impl Res* 13:162-168, 2002.
82. Mansour RM, Reynik RJ, Larson PC: In vivo occlusal forces and moments: forces measured in terminal hinge position and associated moments, *J Dent Res* 56:114-120, 1975.
83. Chung DM, Oh TJ, Shotwell B et al: *Significance of keratinized mucosa in maintenance of dental implants with different surface conditions* [master's thesis], Ann Arbor, Mich, 2005, University of Michigan.
84. Belser UC: The influence of altered working side occlusal guidance on masticatory muscles and related jaw movement, *J Prosthet Dent* 53:406-413, 1985.
85. Michael CG, Javid NS, Colaizzi FA et al: Biting strength and chewing forces in complete denture wearers, *J Prosthet Dent* 3:549-553, 1990.

WWW.HIGHDENT.IR

Chapter 7

Bone Density: A Key Determinant for Treatment Planning

Carl E. Misch

Available bone is particularly important in implant dentistry and describes the external architecture or volume of the edentulous area considered for implants. Historically, the available bone was not modified in the implant candidate. Instead, the existing bone volume was the primary factor used to develop a treatment plan. Short implants and fewer implants were used in less available bone and long implants in greater numbers were inserted in larger bone volumes. Today, the treatment plan first considers the final prosthesis options. The patient force factors are then noted. The next consideration is the bone density in the sites of the implant abutments.

The internal structure of bone is described in terms of quality or density, which reflects a number of biomechanical properties, such as strength and modulus of elasticity. The external and internal architecture of bone controls virtually every facet of the practice of implant dentistry. The density of available bone in an edentulous site is a determining factor in treatment planning, implant design, surgical approach, healing time, and initial progressive bone loading during prosthetic reconstruction.[1,2] This chapter presents the aspects of bone density related to overall planning of an implant prosthesis.

INFLUENCE OF BONE DENSITY ON IMPLANT SUCCESS RATES

The quality of bone is often dependent upon the arch position.[3-7] The most dense bone is usually observed in the anterior mandible, followed by the anterior maxilla and posterior mandible, and the least dense bone is typically found in the posterior maxilla. Following a standard surgical and prosthetic protocol, Adell et al.[8] reported an approximately 10% greater success rate in the anterior mandible as compared with the anterior maxilla. Schnitman et al. also noted lower success rates in the posterior mandible as compared with the anterior mandible when the same protocol was followed.[9] The highest clinical failure rates have been reported in the posterior maxilla, where the force magnitude is greater and the bone density is poorer.[5-7,9-13] A range of implant survival has been found relative to arch location.

In addition to arch location, several independent groups have reported different failure rates related to the quality of the bone.[3-20a] Engquist et al. observed that 78% of all reported implant failures were in soft bone types.[16] Friberg et al. observed that 66% of their group's implant failures occurred in the resorbed maxilla with soft bone.[3] Jaffin and Berman, in a 5-year study, reported a 44% implant failure when poor-density bone was observed in the maxilla.[15] The article documented a 35% implant loss in any region of the mouth when bone density was poor. Fifty-five percent of all implant failures within their study sample occurred in the soft bone type. Johns et al. reported 3% failure of implants in moderate bone densities, but a 28% implant failure in the poorest bone type.[17] Smedberg et al. reported a 36% failure rate in the poorest bone density.[18] The reduced implant survival most often is more related to bone density than arch location. In a 15-year follow-up study, Snauwaert et al. reported early annual and late failures were more frequently found in the maxilla.[12] Hermann et al.[13] found implant failures were strongly correlated to patient factors, including bone quality, especially when coupled with poor bone volume (65% of these patients experienced failure). These reported failures are not primarily related to surgery healing, but instead occur after prosthetic loading. Therefore, over the years, many independent clinical groups, following a standardized surgical protocol, documented the indisputable influence of bone density on clinical success (Figure 7-1).*

However, a protocol established by the author, which adapts the treatment plan, implant selection, surgical approach, healing regimen, and initial prosthetic loading, has resulted in similar implant success rates in all bone densities and all arch positions.[21-24] This chapter proposes a scientific rationale for the modification of a

*References 3, 5-7, 9, 11-13, 16-20a.

WWW.HIGHDENT.IR

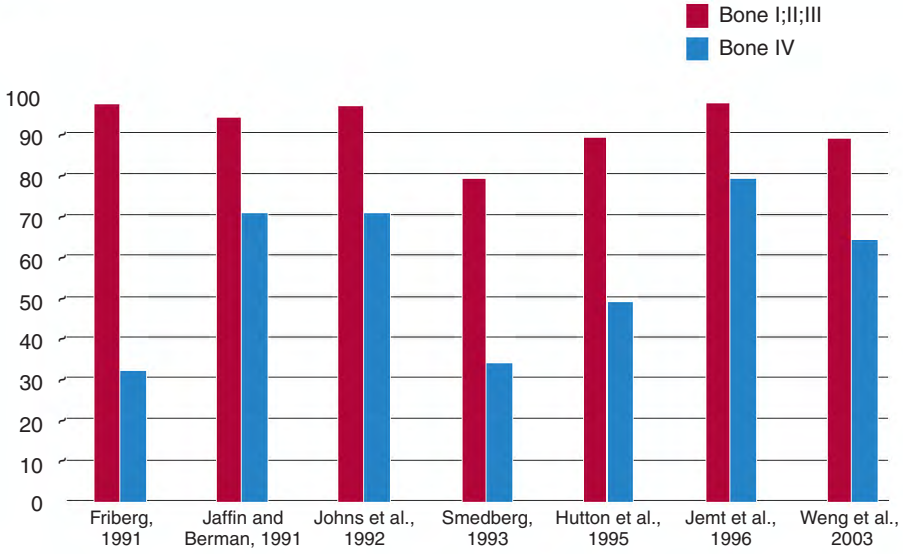


Figure 7-1 Over the years, several clinical reports observed higher success rates in better bone quality and lower survival rates in poor bone quality (Bone IV).

Figure 7-2 The maxilla is a force distribution unit, and the mandible is a force absorption unit.

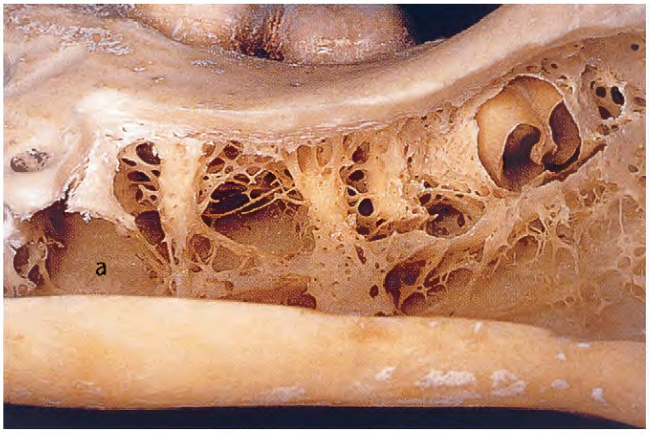


Figure 7-3 The trabecular bone in a dentate mandible is more coarse compared with the maxilla. The mandible, as an independent structure, is a force-absorbing element.

treatment plan in function of implant density to achieve comparable success rates in all bone types.

ETIOLOGY OF VARIABLE BONE DENSITY

Bone is an organ that is able to change in relation to a number of factors, including hormones, vitamins, and mechanical influences. However, biomechanical parameters, such as duration of edentulous state, are predominant.[25-29] Awareness of this adaptability has been reported for more than a century. In 1887, Meier qualitatively described the architecture of trabecular bone in the femur.[30] In 1888, Kulmann noticed the similarity between the pattern of trabecular bone in the femur and tension trajectories in construction beams.[31] Wolff, in 1892, further elaborated on these concepts and published, "Every change in the form and function of bone or of its function alone is followed by certain definite changes in the internal architecture, and equally definite alteration in its external conformation, in accordance with mathematical laws."[32] The modified function of bone and the definite changes in the internal and external formation of the vertebral skeleton as influenced by mechanical load were reported by Murry.[33] Therefore the external architecture of bone changes in relation to function, and the internal bony structure is also modified.

MacMillan[34] and Parfitt[35] have reported on the structural characteristics and variation of trabeculae in the alveolar regions of the jaws. For example, the maxilla and mandible have different biomechanical functions (Figure 7-2). The mandible, as an independent structure, is designed as a force-absorption unit. Therefore, when teeth are present, the outer cortical bone is denser and thicker and the trabecular bone is more coarse and dense

WWW.HIGHDENT.IR

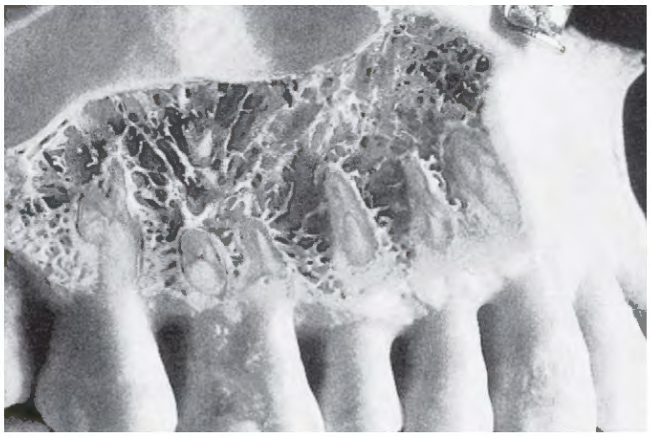


Figure 7-4 The dentate maxilla has a finer trabecular pattern compared with the mandible. The maxilla is a force distribution unit and is designed to protect the orbit and brain.

Figure 7-5 The trabecular bone of each jaw has structural variations. The trabecular bone is most dense next to the teeth, where it forms the cribriform plate. Between the teeth, the bone is usually most dense near the crest and least dense at the apex.

(Figure 7-3). On the other hand, the maxilla is a force-distribution unit. Any strain to the maxilla is transferred by the zygomatic arch and palate away from the brain and orbit. As a consequence, the maxilla has a thin cortical plate and fine trabecular bone supporting the teeth (Figure 7-4). They also noted that the bone is most dense around the teeth (cribriform plate) and more dense around the teeth at the crest, compared with the regions around the apices (Figure 7-5). Alveolar bone resorption associated with orthodontic therapy also illustrates the biomechanical sensitivity of the alveolar processes.[36] Generalized trabecular bone loss in the jaws occurs in regions around a tooth from a decrease in mechanical strain.[37] Orban demonstrated a decrease in the trabecular bone pattern around a maxillary molar with no opposing occlusion, compared with a tooth with occlusal contacts on the contralateral side[38] (Figure 7-6). Bone density in the jaws also decreases after tooth loss. This loss is primarily related to the length of time the region has been edentulous and not loaded appropriately, the initial density of the bone, flexure and torsion in the mandible, and parafunction before

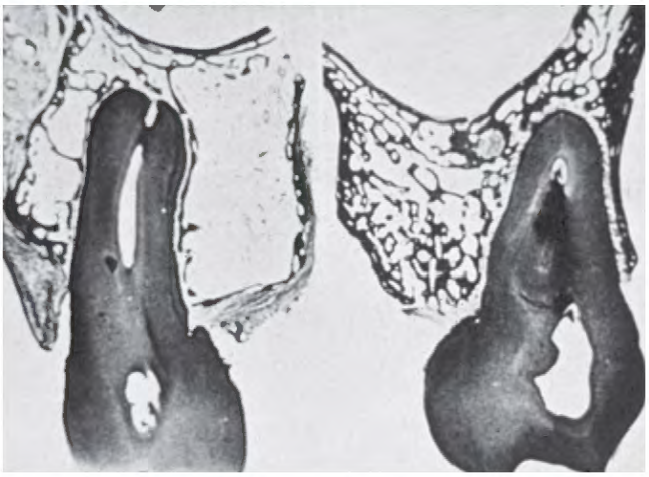


Figure 7-6 On the left, the opposing mandibular tooth was removed. A lack of occlusal contact resulted in loss of trabecular bone around the maxillary tooth. The tooth on the right is from the same monkey, with the opposing mandibular tooth in place. The trabecular bone is much more dense around the tooth. The disuse atrophy observed on the left is from inadequate microstrain conditions to maintain the bone. (*From Orban B*: Oral histology and embryology, *ed 3, St. Louis, 1953, Mosby*.)

and after tooth loss. In general, the density change after tooth loss is greatest in the posterior maxilla and least in the anterior mandible.

Cortical and trabecular bone throughout the body are constantly modified by either modeling or remodeling.[39] Modeling has independent sites of formation and resorption and results in the change of the shape or size of bone. Remodeling is a process of resorption and formation at the same site that replaces previously existing bone and primarily affects the internal turnover of bone, including that region where teeth are lost or the bone next to an endosteal implant.[40,41] These adaptive phenomena have been associated with the alteration of the mechanical stress and strain environment within the host bone.[42,43] Stress is determined by the magnitude of force divided by the functional area over which it is applied. Strain is defined as the change in length of a material divided by the original length. The greater the magnitude of stress applied to the bone, the greater the strain observed in the bone.[44] Bone modeling and remodeling are primarily controlled, in part or whole, by the mechanical environment of strain. Overall, the density of alveolar bone evolves as a result of mechanical deformation from microstrain. Frost proposed a model of four histologic patterns for compact bone as it relates to mechanical adaptation to strain.[45] The pathologic overload zone, mild overload zone, adapted window, and acute disuse window were described for bone in relation to the amount of the microstrain experienced (Figure 7-7). These four categories also may be used to describe the trabecular bone response in the jaws.

The bone in the acute disuse window loses mineral density, and disuse atrophy occurs because modeling for new bone is inhibited and remodeling is stimulated,

WWW.HIGHDENT.IR

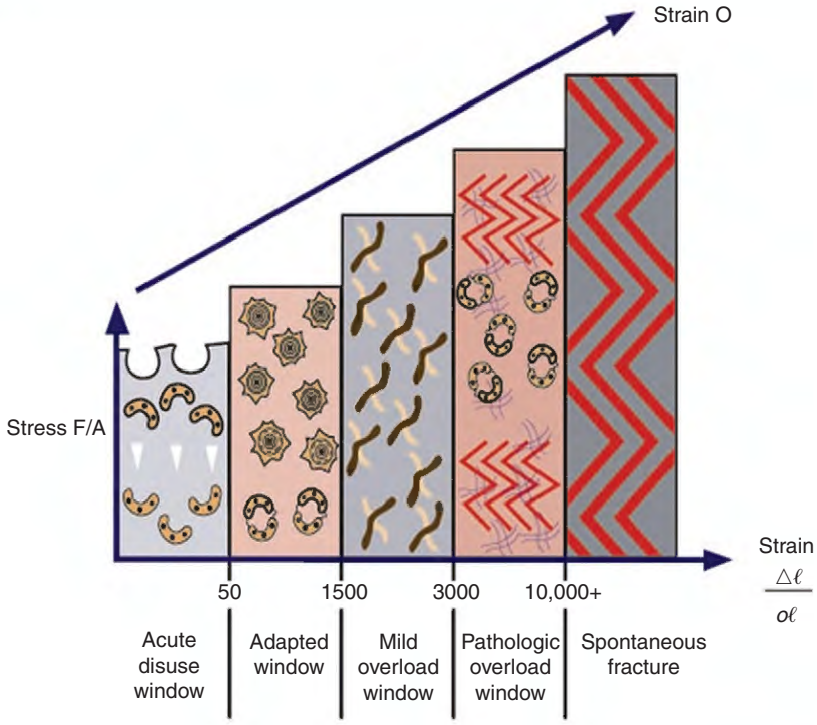


Figure 7-7 Four zones for bone related to mechanical adaption to strain before spontaneous fracture. The acute disuse window is the lowest microstrain amount. The adapted window is an ideal physiologic loading zone. The mild overload zone causes microfracture and triggers an increase in bone remodeling, which produces more woven bone. The pathologic overload zone causes increase in fatigue fractures, remodeling, and bone resorption.

with a gradual net loss of bone. The microstrain of bone for trivial loading is reported to be 0 to 50 microstrain. This phenomenon may occur throughout the skeletal system, as evidenced by a 15% decrease in the cortical plate and extensive trabecular bone loss consequent to immobilized limbs for 3 months.[46] A cortical bone density decrease of 40% and a trabecular bone density decrease of 12% also have been reported with disuse of bone.[47,48] Interestingly, bone loss similar to disuse atrophy has been associated with microgravity environments in outer space, because the microstrain in bone resulting from the earth's gravity is not present in the "weightless" environment of space.[49] In fact, an astronaut aboard the Russian Mir space station for 111 days lost nearly 12% of his bone minerals.[50,51]

The adapted window (50 to 1500 microstrain) represents an equilibrium of modeling and remodeling, and bone conditions are maintained at this level. Bone in this strain environment remains in a steady state, and this may be considered the homeostatic window of health. The histologic description of this bone is primarily lamellar or load-bearing bone. Approximately 18% of trabecular bone and 2% to 5% of cortical bone is remodeled each year[25] in the physiologic loading zone, which corresponds to the adapted window. This is the range of strain ideally desired around an endosteal implant, once a stress equilibrium has been established (Figure 7-8). Bone turnover is required in the adapted window, as Mori and Burr provide evidence of remodeling in regions of bone microfracture from fatigue damage within the physiologic range.[52]

The mild overload zone (1500 to 3000 microstrain) causes a greater rate of fatigue microfracture and increase

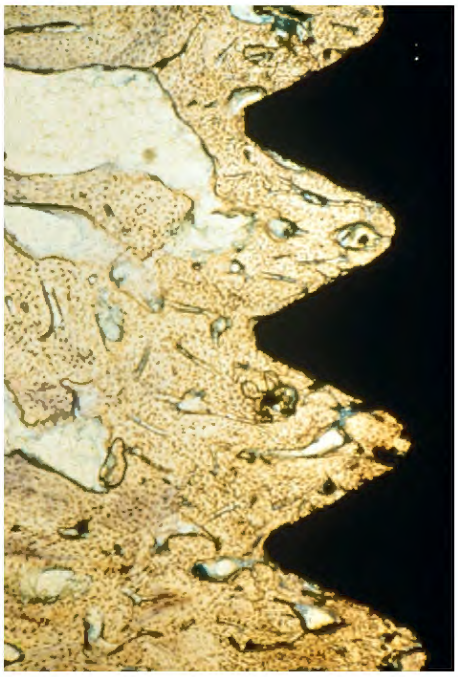


Figure 7-8 An ideal bone-implant interface has organized, lamellar bone next to the implant. The adapted window zone of microstrain balances remodeling and allows the bone to maintain this condition.

in the cellular turnover rate of bone. As a result, the bone strength and density may eventually decrease. The histologic description of bone in this range is usually woven or repair bone. This may be the state for bone when an endosteal implant is overloaded and the bone interface attempts to change the strain environment. During the repair process, the woven bone is weaker

WWW.HIGHDENT.IR

than the more mature, mineralized lamellar bone.[40] Therefore, while bone is loaded in the mild overload zone, care must be taken because the "safety range" for bone strength is reduced during the repair.[41]

Pathologic overload zones are reached when microstrains are greater than 3000 units.[45] Cortical bone fractures occur at 10,000 to 20,000 microstrain (1% to 2% deformation). Therefore pathologic overload may begin at microstrain levels of only 20% to 40% of the ultimate strength or physical fracture of cortical bone. The bone may resorb and form fibrous tissue, or when present, repair woven bone in this zone, as a sustained turnover rate is necessary. The marginal bone loss evidenced during implant overloading may be a result of the bone in the pathologic overload zone. Implant failure from overload may also be a result of bone in the pathologic overload zone.

BONE CLASSIFICATION SCHEMES RELATED TO IMPLANT DENTISTRY

An appreciation of bone density and its relation to oral implantology has existed for more than 25 years. Linkow, in 1970, classified bone density into three categories[53]:

- Class I bone structure: This ideal bone type consists of evenly spaced trabeculae with small cancellated spaces.
- Class II bone structure: The bone has slightly larger cancellated spaces with less uniformity of the osseous pattern.
- Class III bone structure: Large marrow-filled spaces exist between bone trabeculae.

Linkow stated that Class III bone results in a loose-fitting implant; Class II bone was satisfactory for implants; and Class I bone was the most ideal foundation for implant prostheses.

In 1985, Lekholm and Zarb listed four bone qualities found in the anterior regions of the jawbone[54] (Figure 7-9). Quality 1 was composed of homogeneous compact bone. Quality 2 had a thick layer of compact bone surrounding a core of dense trabecular bone. Quality 3 had a thin layer of cortical bone surrounding dense trabecular bone of favorable strength. Quality 4 had a thin layer of cortical bone surrounding a core of low-density trabecular bone. Irrespective of the different bone qualities, all bone was treated with the same implant design and standard surgical and prosthetic protocol.[8] Following this protocol, Schnitman and others observed a 10% difference in implant survival between Quality 2 and Quality 3 bone, and 22% lower survival in the poorest bone density.[3,9,16] Johns et al. reported 3% failure in Type III bone, but 28% in Type IV bone.[17] Smedberg et al. reported a 36% failure rate in Type IV bone.[18] Higuchi and others also experienced a greater failure in the soft bone of the maxilla.* It is obvious that a standardized surgical, prosthetic, and implant design protocol does not yield similar results in all bone densities. In addition, these reports are for implant survival, not the quality of health of surviving implants. The amount of crestal bone loss also has been related to bone density,[56-60] and further supports a different protocol for soft bone.

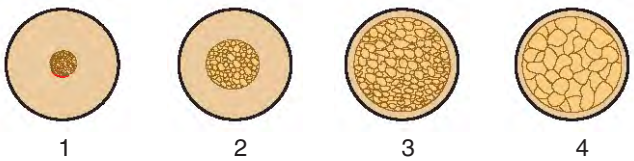


Figure 7-9 Four bone qualities for the anterior region of the jaws. Quality 1 is composed of homogenous compact bone. Quality 2 has a thick layer of cortical bone surrounding dense trabecular bone. Quality 3 has a thin layer of cortical bone surrounded by dense trabecular bone of favorable strength. Quality 4 has a thin layer of cortical bone surrounding a core of low-density trabecular bone. *(From Lekholm U, Zarb GA: Patient selection and preparation. In Brånemark P-I, Zarb GA, Albrektsson T, editors*: Tissue integrated protheses: osseintegration in clinical denistry, *Chicago, 1985, Quintessence.)*

In 1988, Misch proposed four bone density groups independent of the regions of the jaws, based on macroscopic cortical and trabecular bone characteristics.[1,2] The regions of the jaws with similar densities were often consistent. Suggested treatment plans, implant design, surgical protocol, healing, and progressive loading time spans have been described for each bone density type.[23,60,61] Following this regimen, similar implant survival rates have been observed for all bone densities.[21-23]

MISCH BONE DENSITY CLASSIFICATION

Dense or porous cortical bone is found on the outer surfaces of bone and includes the crest of an edentulous ridge. Coarse and fine trabecular bone types are found within the outer shell of cortical bone and occasionally on the crestal surface of an edentulous residual ridge. These four macroscopic structures of bone may be arranged from the least dense to the most dense, as first described by Roberts and Frost[25,45] (Figure 7-10).

In combination, these four increasing macroscopic densities constitute four bone categories described by Misch (D1, D2, D3, and D4) located in the edentulous areas of the maxilla and mandible (Figure 7-11). The regional locations of the different densities of cortical bone are more consistent than the highly variable trabecular bone.

D1 bone is primarily dense cortical bone. D2 bone has dense-to-porous cortical bone on the crest and, within

* References 5-7, 12, 13, 19, 20, 55.

WWW.HIGHDENT.IR

Figure 7-10 The macroscopic structure of bone may be described, from the least dense to the most dense, as (1) fine trabecular, (2) coarse trabecular, (3) porous cortical, and (4) dense cortical.

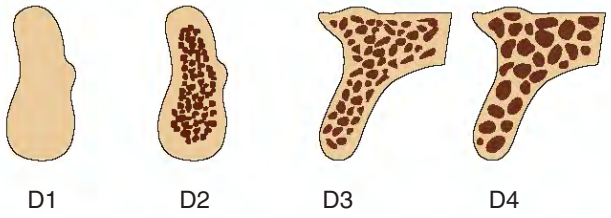


Figure 7-11 Four bone densities found in the edentulous regions of the maxilla and mandible. D1 bone is primarily dense cortical bone; D2 bone has dense to thick porous cortical bone on the crest and coarse trabecular bone underneath; D3 bone has a thinner porous cortical crest and fine trabecular bone within; and D4 bone has almost no crestal cortical bone. The fine trabecular bone composes almost all of the total volume of bone.

Table 7-1 Misch Bone Density Classification Scheme

BONE DENSITY	DESCRIPTION	TACTILE ANALOG	TYPICAL ANATOMICAL LOCATION
D1	Dense cortical	Oak or maple wood	Anterior mandible
D2	Porous cortical and coarse trabecular	White pine or spruce wood	Anterior mandible Posterior mandible Anterior maxilla
D3	Porous cortical (thin) and fine trabecular	Balsa wood	Anterior maxilla Posterior maxilla Posterior mandible
D4	Fine trabecular	Styrofoam	Posterior maxilla

Table 7-2 Usual Anatomic Location of Bone Density Types (% Occurrence)

BONE	ANTERIOR MAXILLA	POSTERIOR MAXILLA	ANTERIOR MANDIBLE	POSTERIOR MANDIBLE
D1	0	0	6	3
D2	25	10	66	50
D3	65	50	25	46
D4	10	40	3	1

the bone, has coarse trabecular bone. D3 bone types have a thinner porous cortical crest and fine trabecular bone in the region next to the implant. D4 bone has almost no crestal cortical bone. The fine trabecular bone composes almost all of the total volume of bone next to the implant (Table 7-1, Figure 7-12). A very soft bone, with incomplete mineralization and large intertrabecular spaces, may be addressed as D5 bone. This bone type is most often immature bone in a developing sinus graft. The bone density may be determined by tactile sense during surgery, the general location, or radiographic evaluation.

BONE DENSITY LOCATION

A review of the literature and a survey of completely and partially edentulous patients postsurgery indicated that the location of different bone densities often may be superimposed on the different regions of the mouth[3-7,11-13,15-20,62-66] (see Tables 7-1 and 7-2). D1 bone is almost never observed in the maxilla and is rarely observed in most mandibles. In the mandible, D1 bone is observed approximately 6% of the time in the Division A anterior mandible and 3% of the time in the posterior mandible, primarily when the implant

WWW.HIGHDENT.IR

Figure 7-12 The four macroscopic bone qualities are, from left to right, D1, D2, D3, and D4. The bone density variance is dependent upon anatomical location and the local strain history of the bone after tooth loss.

Figure 7-13 A cross section of a D2 mandible in the region of the mental foramen. A thick cortical plate exists on the crest and a coarse trabecular bone pattern exists within.

is engaging the lingual cortical plate of bone. In a C–h bone volume (moderate atrophy) in the anterior mandible, the prevalence of D1 bone approaches 25% in males. The C–h mandible often exhibits an increase in torsion, flexure, or both in the anterior segment between the foramina during function. This increased strain may cause the bone to increase in density. D1 bone also may be encountered in the anterior Division A mandible of a Kennedy Class IV partially edentulous patient with a history of parafunction and recent extractions. It may also be observed in the anterior or posterior mandible when the angulation of the implant may require the engagement of the lingual cortical plate.

The bone density D2 is the most common bone density observed in the mandible (Figure 7-13). The anterior mandible consists of D2 bone approximately two thirds of the time. Almost half of patients have D2 bone in the posterior mandible. The maxilla presents D2 bone less often than the mandible. Approximately one fourth of patients have D2 bone, and this is more likely in the partially edentulous patient's anterior and premolar region, rather than the completely edentulous posterior molar areas. Single-tooth or two-tooth, partially edentulous spans in either arch almost always have D2 bone.

Bone density D3 is very common in the maxilla (Figure 7-14). More than half the patients have D3 bone in the upper arch. The anterior edentulous maxilla has D3 bone about 75% of the time, whereas almost half the patients have posterior maxillae with D3 bone (more often in the premolar region). Almost half of the posterior mandibles also present with D3 bone, whereas approximately 25% of the anterior edentulous mandibles have D3 bone.

The softest bone, D4, is most often found in the posterior maxilla (approximately 40%), especially in the molar regions or after a sinus graft augmentation (where almost two thirds of the patients have D4 bone) (Figure 7-15). The anterior maxilla has D4 bone less

WWW.HIGHDENT.IR

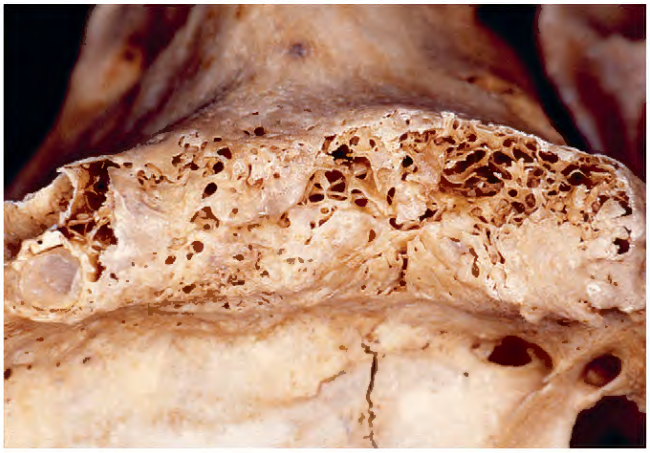


Figure 7-14 A posterior maxilla demonstrating D3 bone with a thin porous cortical plate on the crest with fine trabecular bone underneath.

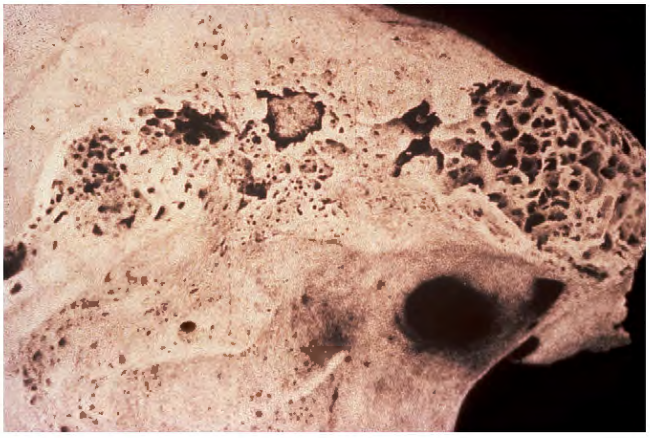


Figure 7-15 In a D4 posterior maxilla, the posterior crestal region has little to no cortical bone on the crest and is composed primarily of fine trabecular bone.

than 10% of the time—more often after an onlay iliac crest bone graft. The mandible presents with D4 bone in less than 3% of the patients. When observed, it is usually Division A bone in a long-term, completely edentulous patient after an osteoplasty to remove the crestal bone.

Generalizations for treatment planning can be made prudently, based on location. The bone density by location method is the first way the dentist can estimate the bone density in the implant sites to develop an initial treatment plan. It is safer to err on the side of less dense bone during treatment planning, so the prosthesis will be designed with slightly more, rather than less, support. Therefore the initial treatment plan before computed tomographic (CT) radiographic scans or surgery suggests the anterior maxilla is treated as D3 bone, the posterior maxilla as D4 bone, the anterior mandible as D2 bone, and the posterior mandible as D3 bone. A more accurate determination of bone density is made with computerized tomograms before surgery or tactilely during implant surgery.

RADIOGRAPHIC BONE DENSITY

Periapical or panoramic radiographs are not very beneficial to determine bone density, because the lateral cortical plates often obscure the trabecular bone density. In addition, the more subtle changes of D2 to D3 cannot be quantified by these radiographs. Therefore the initial treatment plan, which often begins with these radiographs, follows the bone density by location method. Bone density may be more precisely determined by tomographic radiographs, especially computerized tomograms.[67-70] Computed tomography produces axial images of the patient's anatomy, perpendicular to the long axis of the body. Each CT axial image has 260,000 pixels, and each pixel has a CT number (Hounsfield unit) related to the density of the tissues within the pixel. In general, the higher the CT number, the denser the tissue.

Box 7-1 CT Determination of Bone Density

D1:	>1250 Hounsfield units
D2:	850 to 1250 Hounsfield units
D3:	350 to 850 Hounsfield units
D4:	150 to 350 Hounsfield units
D5:	<150 Hounsfield units

CT, Computed tomography.

Modern CT scanners can resolve objects less than 0.5 mm apart. In addition, software is available to electronically position the implant on the CT scan and evaluate to Hounsfield numbers in contact with the implant. In a retrospective study of CT scan images from implant patients, Kirkos and Misch established a correlation between CT Hounsfield units and density at the time of surgery.[69] The Misch bone density classification may be evaluated on the CT images by correlation to a range of Hounsfield units (Box 7-1).[69] The very soft bone observed after some immaterialized bone grafts may be 50 to 200 units.[69] Even negative numbers, suggestive of fat tissue, have been observed with the cortical plates of some jaws, including the anterior mandible. Norton and Gamble also found an overall correlation between subjective bone density scores of Lekholm and Zarb and the CT values.[71] Several studies correlating torque forces at implant insertion with preoperative bone density values from CTs have reported similar conclusions.[72-74] Preoperative CT scan data of areas that lead to successful and unsuccessful implant placement have been reported. In the mandible, failed sites exhibited higher Hounsfield numbers than usual. This was correlated with failure in dense bone, possibly due to the lack of vascularization or overheating during

WWW.HIGHDENT.IR

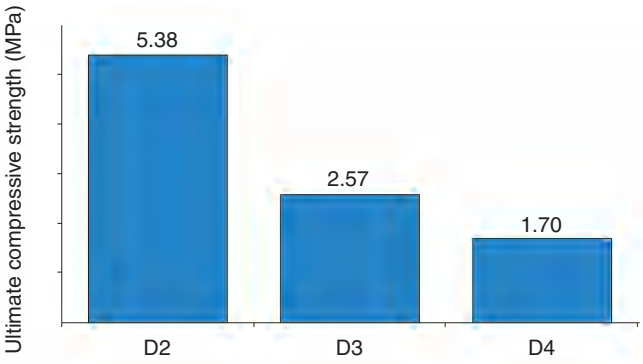


Figure 7-16 The ultimate compressive strength of D2 trabecular bone is greater than D3 trabecular bone. D4 trabecular bone is the weakest.

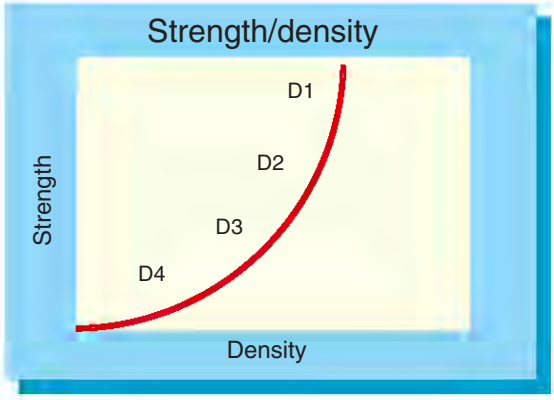


Figure 7-17 The strength of bone is related directly to the density of bone.

surgery. By contrast, in the maxilla, the bone density was low for the failed sites.[68]

The bone density may be different near the crest, compared with the apical region where the implant placement is planned.[75] The most critical region of bone density is the crestal 7 to 10 mm of bone, as this is where most stresses are applied to an osteointegrated bone-implant interface. Therefore when the bone density varies from the most crestal to apical region around the implant, the crestal 7 to 10 mm determines the treatment plan protocol.

BONE DENSITY–TACTILE SENSE

There is a great difference in the tactile sensation during osteotomy preparation in different bone densities, as the density is directly related to its strength.[1,2,74,76] To communicate more broadly to the profession relative to the tactile sense of different bone densities, Misch proposed the different densities of his classification be compared with materials of varying densities.[1,2] Site preparation and implant placement in D1 bone is similar to the resistance on a drill preparing an osteotomy in oak or maple wood. D2 bone is similar to the tactile sensation of drilling into white pine or spruce. D3 bone is similar to drilling into a compressed balsa wood. D4 bone is similar to drilling into a compressed Styrofoam or a light balsa wood. This clinical observation may be correlated to different histomorphometric bone density determinations.[62] When an implant drill can operate at 2000 to 2500 rpm, it may be difficult to feel the difference between D3 and D4 bone. In D4 bone, the drill may be inserted to the full desired depth without the drill rotating. In other words, a bone compression rather than extraction process may be used with the drill. D3 bone is very easy to prepare, but requires the drill to rotate while it is pressed into position. When this tactile method is the primary site, the surgeon should know how to modify the treatment plan if this bone density is different than first estimated when developing the treatment plan.

SCIENTIFIC RATIONALE OF A BONE DENSITY–BASED TREATMENT PLAN

Bone Strength and Density

Bone density is directly related to the strength of bone before microfracture.[77,78] Misch et al. reported on the mechanical properties of trabecular bone in the mandible, using the Misch density classification.[76] A tenfold difference in bone strength may be observed from D1 to D4 bone. D2 bone exhibited a 47% to 68% greater ultimate compressive strength, compared with D3 bone (Figure 7-16). In other words, on a scale of 1 to 10, D1 bone is a 9 to 10 relative to strength. D2 bone is a 7 to 8 on this scale. D3 bone is 50% weaker than D2 bone and is a 3 or 4 on the strength scale. D4 bone is a 1 to 2 and up to 10 times weaker than D1 bone (Figure 7-17). Bidez and Misch performed three-dimensional, finite stress analyses on bone volumes of Division A, B, and C–w patients.[79] Each model reproduced the cortical and trabecular bone material properties of the four densities described. Clinical failure was mathematically predicted in D4 bone and some D3 densities under occlusal loads (Figure 7-18). The bone densities that originally relied on clinical impression are now fully correlated to quantitative objective values obtained from CT scans and bone strength measurements. These values can help prevent failure in specific situations of weak densities.

Elastic Modulus and Density

The elastic modulus describes the amount of strain (changes in length divided by the original length) as a result of a particular amount of stress. It is directly related to the apparent density of bone.[78] The elastic modulus of a material is a value that relates to the stiffness of the material. The elastic modulus of bone is more flexible than titanium. When higher stresses

WWW.HIGHDENT.IR

are applied to an implant prosthesis, the titanium has lower strain (change in shape) compared with the bone. The difference between the two materials may create microstrain conditions of pathologic overload and cause implant failure (Figure 7-19, *A*). When the stresses applied to the implant are low, the microstrain difference between titanium and bone is minimized and remains in the adapted window zone, maintaining load-bearing lamellar bone at the interface (Figure 7-19, *B*).

Misch et al. found the elastic modulus in the human jaw to be different for each bone density[76] (Figure 7-20). As a result, when a stress is applied to an implant prosthesis in D1 bone, the titanium–D1 bone interface exhibits very small microstrain difference. In comparison, when the same amount of stress is applied to an implant in D4 bone, the microstrain difference between titanium and D4 bone is greater and may be in the pathologic overload zone (Figure 7-21). As a result, D4 bone is more likely to cause implant mobility and failure.[79] Several studies using finite element analysis (FEA) models with various implant designs and bone quality have evaluated the stress/strain distribution in the bone around the implants. Conclusions agree with the prior study to show the importance of bone

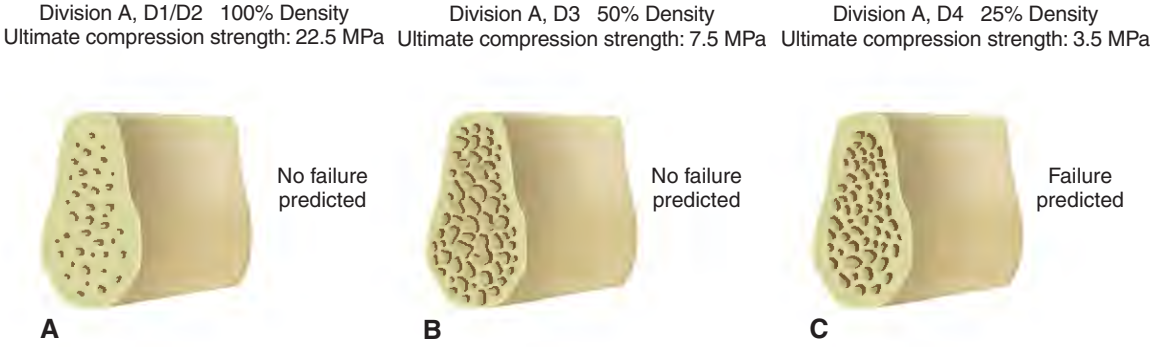


Figure 7-18 **A,** A finite element analysis study of D1 bone with a Division A, B, or C bone volume predicted no implant failure. **B,** In a finite element analysis study of D3 bone of one third the strength, no failure was predicted in Division A bone. **C,** In a finite element analysis study, D4 bone was inadequate in strength for implant success, even in Division A bone volume.

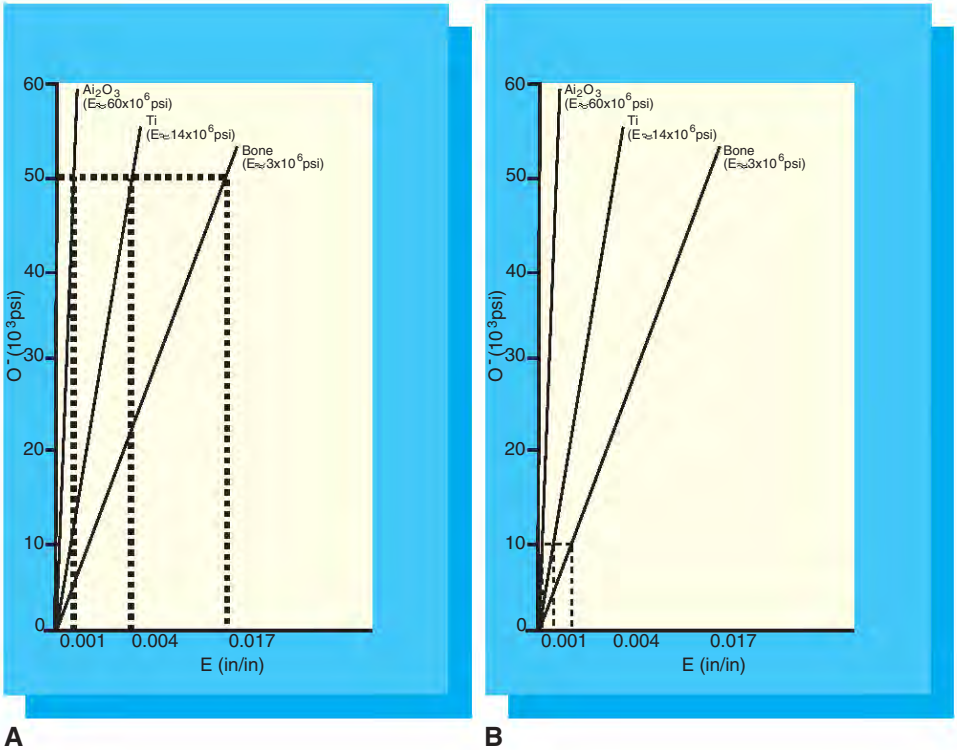


Figure 7-19 **A,** When the microstrain is high (50×10^3 psi in this example), the change in shape difference of titanium and bone is large and may result in a pathologic overload zone. As a result, fibrous tissue at the interface and implant mobility is expected. **B,** When the microstrain is low (10×10^3 psi in this example), the change in shape difference between titanium and bone is small and may result in the ideal adapted window zone. As a result, organized, lamellar bone may remain at the implant interface.

WWW.HIGHDENT.IR

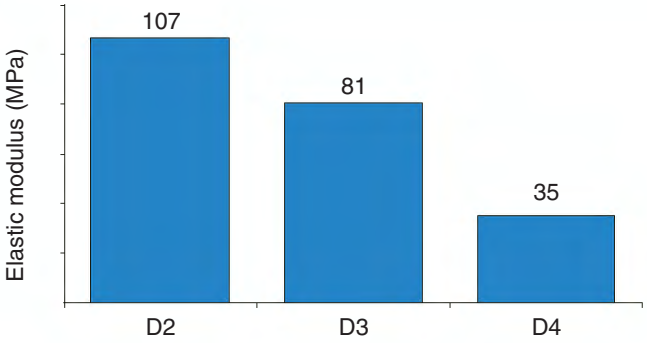


Figure 7-20 The elastic modulus for D2 trabecular bone is greater than D3 trabecular bone, and D4 trabecular bone has the lowest elastic modulus.

quality in the treatment planning phase for long-term prognosis.[80-85]

Bone Density and Bone-Implant Contact Percentage

The initial bone density not only provides mechanical immobilization of the implant during healing, but after healing also permits distribution and transmission of stresses from the prosthesis to the implant-bone interface. The mechanical distribution of stress occurs primarily where bone is in contact with the implant. Open marrow spaces or zones of unorganized fibrous tissue do not permit controlled force dissipation or microstrain conditions to the local bone cells. Because stress equals force divided by the area over which the force is applied, the less the area of bone contacting the implant body, the greater the overall stress, other factors being equal. Therefore the bone-implant contact percent may influence the amount of stress/strain at the interface.

Misch noted in 1990 that the bone density influences the amount of bone in contact with the implant surface, not only at first stage surgery, but also at the second stage uncovery and early prosthetic loading.[2] The bone-implant contact (BIC) percentage is significantly greater in cortical bone than in trabecular bone. The very dense D1 bone of a C–h resorbed anterior mandible or of the lingual cortical plate of a Division A anterior or posterior mandible provides the highest percentage of bone in contact with an endosteal implant and may approximate more than 85% BIC (Figure 7-22). D2 bone, after initial healing, usually has 65% to 75% BIC (Figure 7-23). D3 bone typically has 40% to 50% BIC after initial healing (Figure 7-24). The sparse trabeculae of the bone often found in the posterior maxilla (D4) offer fewer areas of contact with the body of the implant. With a machined-surface implant this may

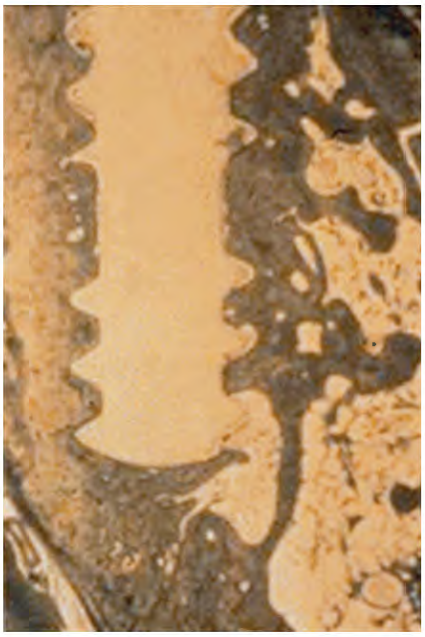


Figure 7-22 D1 bone density has the greatest amount of bone-implant contact. Because stress equals force divided by area, the increase in the area of contact results in a decreased amount of stress.

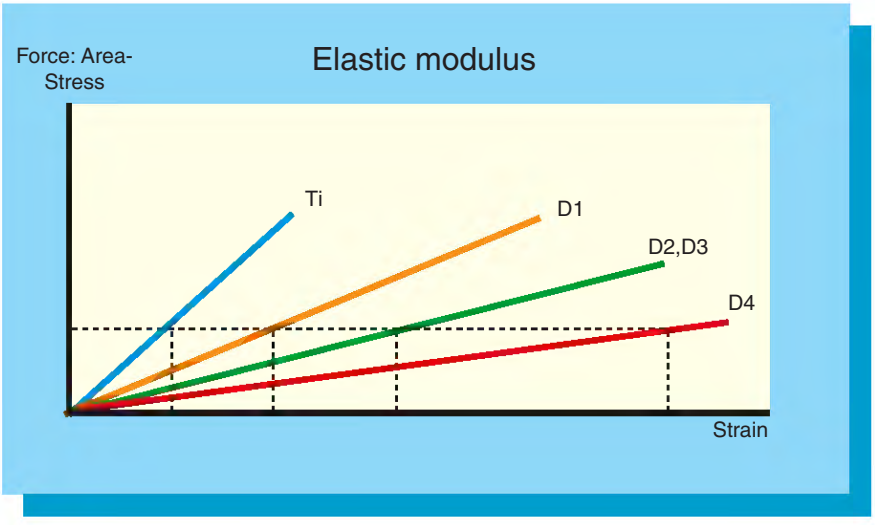


Figure 7-21 The microstrain difference between titanium and D4 bone is great and may be in the pathologic overload zone, whereas at the same stress level, the microstrain difference between titanium and D2 bone may be within the ideal adapted window zone.

WWW.HIGHDENT.IR

Figure 7-23 In D2 bone density, one finds primarily coarse trabecular bone next to the implant. The bone-implant contact is greater than D3 bone but less than D1 bone.

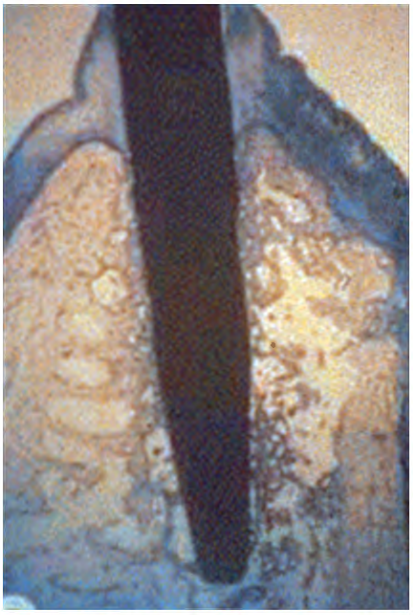


Figure 7-24 The fine trabecular bone of D3 initially heals next to the implant with 40% to 50% bone implant contact.

approximate less than 30% BIC and is most related to the implant design and surface condition (Figure 7-25). Consequently, greater implant surface area is required to obtain a similar amount of bone-implant contact in soft bone, compared with a denser bone quality found around an anterior mandibular implant.

Bone Density and Stress Transfer

Crestal bone loss and early implant failure after loading results may occur from excess stress at the implant-bone interface.[48-51,56-59,80-84] A range of bone loss has been observed in implants with similar load conditions.[59] Misch noted in 1990 that part of this phenomenon may be explained by the evaluation of finite element analysis stress contours in the bone for each bone density.[2] As a result of the correlation of bone density, elastic modulus bone strength, and bone-implant contact percent, when a load is placed on an implant, the stress contours in the bone are different for each bone density. In D1 bone, highest strains are concentrated around the implant near the crest, and the stress in the region is of lesser magnitude. D2 bone, with the same load, sustains a slightly greater crestal strain, and the intensity of the stress extends farther apically along the implant body (Figure 7-26). D4 bone exhibits the greatest crestal strains, and the magnitude of the stress on the implant proceeds farthest apically along the implant body (Figure 7-27). As a result, the magnitude of a prosthetic load may remain similar and yet give one of the following three different clinical situations at the bone-implant interface, based on bone density: (1) physiologic bone loads in the adapted window

Figure 7-25 D4 bone has the least bone-implant contact. As a result, the stress is greatest for the D4 bone-implant interface. Trabecular bone is fine, the strength is poor, and the modulus of elasticity microstrain difference is greatest. The microstrain difference for each bone density is not the same. D4 bone is most at risk, whereas D1 bone is least at risk.

zone and no marginal bone loss, (2) mild overload to pathologic overload bone loads and crestal bone loss, or (3) generalized pathologic overload and implant failure. Therefore, to obtain a similar clinical result in each implant prosthesis, the variables in each patient must be either eliminated or accounted for in the treatment plan. As the myriad of variables cannot be

WWW.HIGHDENT.IR

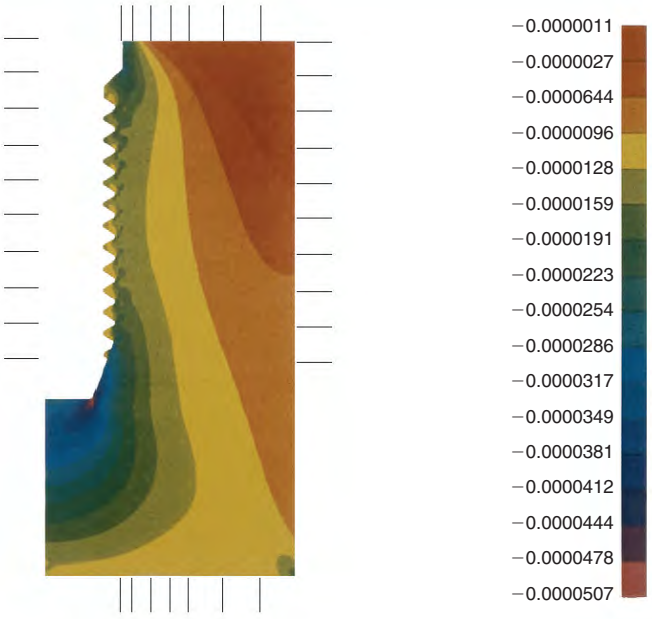


Figure 7-26 Stress transfer around the implant interface is different for each bone density. In this two-dimensional finite element analysis, D2 bone has an intermediate stress intensity around the implant.

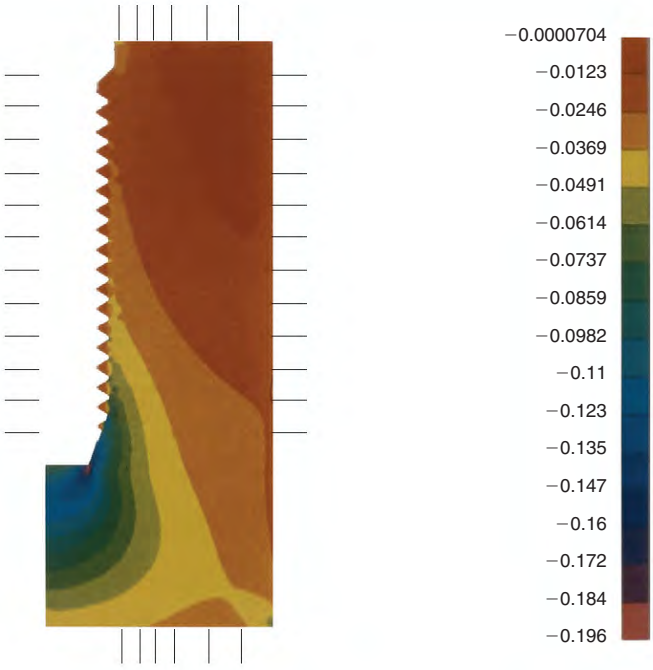


Figure 7-27 A two-dimensional finite element analysis demonstrates that D4 bone has a higher stress intensity around the implant, and the higher intensity even extends to the zone around the apical threads.

eliminated relative to bone density, the treatment plans (including implant number, size, and design) should be modified.

TREATMENT PLANNING

When a patient is first examined, the most common radiographic evaluation is with a panoramic radiograph. The initial treatment plan is presented to the patient using the anatomical location as an index of the bone density: anterior mandible and single tooth replacement is D2, anterior maxilla and posterior mandible is D3, and posterior maxilla is D4. After the following treatment steps are taken into consideration (e.g., implant key position and number, implant size and design, and available bone), a more complete treatment plan relative to bone density is obtained by a CT scan or modified during the surgical procedure using the tactile method to determine bone density.

Four facts form the basis for treatment plan modification in function of the bone quality: (1) each bone density has a different strength; (2) bone density affects the elastic modulus; (3) bone density differences result in different amounts of bone-implant contact percent; and (4) bone density differences result with a different stress-strain distribution at the implant-bone interface. Bone density is an implant treatment plan modifier in several ways—prosthetic factors, implant size, implant design, implant surface condition, implant number, and the need or method of progressive loading.

As the bone density decreases, the strength of the bone also decreases. To decrease the incidence of microfracture of bone, the strain to the bone should be reduced.

Box 7-2 Treatment Plans

Bone density/Treatment plan
Stress = Force/Area
↓ Bone density = ↑ Implant area

- Implant number
- Implant width
- Implant length
- Implant design
- Implant surface condition

Strain is directly related to stress. Consequently, the stress to the implant system should also be reduced as the bone density decreases (Box 7-2). One way to reduce the biomechanical loads on implants is by prosthesis design to decrease force. For example, cantilever length may be shortened or eliminated, narrower occlusal tables designed and offset loads minimized, all of which reduce the amount of load.[44] RP-4 restorations, rather than fixed prostheses, permit the patient to remove the restorations at night and reduce nocturnal parafunctional forces. RP-5 prostheses permit the soft tissue to share the occlusal force and reduce the stress on the implants. Night guards and acrylic occlusal surfaces distribute and dissipate parafunctional forces on an implant system. As the bone density decreases, these prosthetic factors become more important.

The load on the implant may also be influenced by the direction of force to the implant body.[79] A load directed along the long axis of the implant body decreases the amount of stress in the crestal bone

WWW.HIGHDENT.IR

region compared with an angled load. Therefore as the bone density decreases, axial loads on the implant body become more critical. Bone grafting or bone spreading to increase the width of bone and to better position the implant relative to the intended load is considered for soft bone types.

Stress may also be reduced by increasing the functional area over which the force is applied. Increasing implant number is an excellent way to reduce stress by increasing functional loading area. Three implants rather than two may decrease applied implant moments in half and bone reaction forces by two thirds, depending on implant position and size.[44] An implant prosthesis with normal patient forces in the bone should have at least one implant per tooth. In the molar region, two implants for each missing molar may be appropriate. In D2 bone with normal patient forces, a pontic may replace a tooth between two implants. In D3 bone, one implant per tooth is often appropriate.

The surface area of the implant macrogeometry may be increased to decrease stress to the implant-bone interface.[60,86] The width of the implant may decrease stress by increasing the surface area.[60,87] This may also reduce the length requirement. For every 0.5 mm increase in width, there is an increased surface area between 10% and 15% for a cylinder implant, and even more difference is found with threaded-implant body designs. Because the greatest stresses are concentrated at the crestal region of the implant, width is more significant than length for an implant design, once adequate length has been established. D4 bone should often require wider implants compared with D1 or D2 bone. This may require onlay grafts or bone spreading to increase the width of bone, when other stress factors are high. Based on long-term clinical experience of V-shaped threaded implant bodies, the minimum bone height for initial fixation and early loading for D1 bone is 7 mm; for D2 bone, 9 mm; and for D3 bone, 12 mm using the classic V-thread screw implant design and titanium surface condition. D4 bone benefits from relatively longer implants for initial fixation and early loading compared with other bone densities, not only for initial fixation, but also because the stress/strain transfer of occlusal forces extends farther down the implant body. This implant length requirement may require sinus grafts in the posterior maxilla. However, because the crestal region is where pathologic overload of bone most often occurs after prosthetic loading, once initial healing is complete the length of the implant is not as effective to solve crestal bone loss (and the quality of implant health) as other factors (e.g., implant design, implant width).

The macro design affects the magnitude of stresses and their impact on the bone-implant interface[10,80,88,89] and can dramatically change the amount and contour of the bone strains concentrated at the interface. Different implant design criteria respond to different bone densities. Bone densities exhibit a tenfold difference in strength, and the elastic modulus is significantly different between D1 and D4. Implants designed for D4 bone should have the greatest surface area. For example, a classic V-thread screw design has 30% more surface area than a cylinder implant. A thread design with more threads has more surface area than one with fewer threads. The depth of the thread is also a variable that may be controlled, based on the surface area desired. The deeper the thread depth in the implant body, the more functional surface area for the bone-implant contact. An implant body designed for the soft bone should have more and deeper threads than an implant body designed for hard bone (Figure 7-28). A D1 implant, on the other hand, may be designed for easy surgical placement, as the strains under load are minimized, but the surgical failure rates are greater.

Figure 7-28 Several features may be incorporated in the implant design to decrease stress after loading. The implant on the right has more threads than the implant on the left. The thread depth may be greater to increase surface area. A rough surface condition will also increase bone-implant contact. As a result, the implant on the right (BioHorizons D4 Maestro Implant) has more functional surface area than the implant on the left (BioHorizons D2 Maestro Implant). In soft bone types, the implant on the right has a loading advantage.

Coatings or the surface condition on an implant body can increase the bone-implant contact percentage and therefore the functional surface area. A rougher surface is strongly suggested in soft bone and has resulted in improved short-term survival rates when compared with machined titanium.[11] After 1 to 2 years, the mechanical load on the overall implant design is more critical to the amount and type of bone contact compared with the surface condition on the implant body. Rough surface conditions also may have some disadvantages. Plaque retention when exposed above the bone, contamination, and increased cost are a few of the concerns with roughened surfaces. The benefit and risk of surface conditions suggests the roughest surfaces are most often used in only softer bone types.

Progressive bone loading provides for a gradual increase in occlusal loads, separated by a time interval

WWW.HIGHDENT.IR

to allow the bone to mature and accommodate to the local strain environment.[2] Over time, progressive loading changes the amount and density of the implant-bone contact. The increased density of bone at the implant interface improves the overall support system mechanism. The softer the bone, the more important the need for progressive loading.[1,2]

SUMMARY

A key determinant for clinical success is the diagnosis of the bone density in a potential implant site. The strength of bone is directly related to bone density. The modulus of elasticity is related to bone density. The percentage of bone-implant contact is related to bone density, and the axial stress contours around an implant are affected by the density of bone. As a consequence, past clinical reports that did not alter the protocol of treatment related to bone density had variable survival rates. To the contrary, altering the treatment plan to compensate for soft bone types has provided similar survival rates in all bone densities. Once the prosthetic option, key implant position, and patient force factors have been determined, the bone density in the implant sites should be evaluated to modify the treatment plan. The treatment plan may be modified by reducing the force on the prosthesis or increasing the area of load by increasing implant number, implant position, implant size, implant design, or implant body surface condition.

References

1. Misch CE: Bone character: second vital implant criterion, *Dent Today* 7:39-40, 1988.
2. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing, and progressive loading, *Int J Oral Implant* 6:23-31, 1990.
3. Friberg B, Jemt T, Lekholm U: Early failures in 4,641 consecutively placed Brånemark dental implants: a study from stage I surgery to the connection of completed prostheses, *Int J Oral Maxillofac Implants* 6:142-146, 1991.
4. van Steenberghe D, Lekholm U, Bolender C et al: Applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study on 558 fixtures, *Int J Oral Maxillofac Implants* 5:272-281, 1990.
5. Hutton JE, Heath MR, Chai JY et al: Factors related to success and failure rates at 3 year follow up in a multicenter study of overdentures supported by Brånemark implants, *Int J Oral Maxillofac Implants* 10:33-42, 1995.
6. Esposito M, Hirsch JM, Lekholm U et al: Biological factors contributing to failures of osseointegrated oral implants. (II) Etiopathogenesis, *Eur J Oral Sci* 106:721-764, 1998.
7. Morris HF, Ochi S, Crum P et al: AICRG, Part I: a 6-year multicentered, multidisciplinary clinical study of a new and innovative implant design, *J Oral Implantol* 30: 125-133, 2004.
8. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 6:387-416, 1981.
9. Schnitman PA, Rubenstein JE, Whorle PS et al: Implants for partial edentulism, *J Dent Educ* 52:725-736, 1988.
10. Minsk L, Polson A, Weisgold A et al: Outcome failures of endosseous implants from a clinical training center, *Compend Contin Educ Dent* 17:848-859, 1996.
11. Fugazzotto PA, Wheeler SL, Lindsay JA: Success and failure rates of cylinder implants in type IV bone, *J Periodontol* 64:1085-1087, 1993.
12. Snauwaert K, Duyck D, van Steenberghe D et al: Time dependent failure rate and marginal bone loss of implant supported prostheses: a 15-year follow-up study, *Clin Oral Investig* 4:13-20, 2000.
13. Herrmann I, Lekholm U, Holm S et al: Evaluation of patient and implant characteristics as potential prognostic factors for oral implant failures, *Int J Oral Maxillofac Implants* 20:220-230, 2005.
14. Brånemark P-I, Hansson BO, Adell R et al: *Osseointegrated implants in the treatment of the edentulous jaw—experience from a 10-year period*, Stockholm, 1977, Almquist and Wiksell International.
15. Jaffin RA, Berman CL: The excessive loss of Brånemark fixtures in the Type IV bone: a 5-year analysis, *J Periodontol* 62:2-4, 1991.
16. Engquist B, Bergendal T, Kallus T et al: A retrospective multicenter evaluation of osseointegrated implants supporting overdentures, *Int J Oral Maxillofac Implants* 3:129-134, 1988.
17. Johns B Jr, Jemt T, Heath MR et al: A multicenter study of overdentures supported by Brånemark implants, *Int J Oral Maxillofac Implants* 7:513-522, 1992.
18. Smedberg JI, Lothigius E, Bodin L et al: A clinical and radiological two-year follow-up study of maxillary overdentures on osseointegrated implants, *Oral Clin Implants Res* 4:39-46, 1993.
19. Higuchi KW, Folmer T, Kultje C: Implant survival rates in partially edentulous patients: a 3-year prospective multicenter study, *J Oral Maxillofac Surg* 53:264-268, 1995.
20. Weng D, Jacobson Z, Tarnow D et al: A prospective multicenter clinical trial of 3i machined surface implants: results after 6 years of follow up, *Int J Oral Maxillofac Implants* 18:417-423, 2003.
20a. Jemt T, Chai J, Harnett J: A 5-year prospective multicenter follow-up report on overdentures supported by osseointegrated implants, *Int J Oral Maxillofac Implants* 11:291-298,1996.
21. Misch CE, Hoar JE, Hazen R et al: Bone quality based implant system: a prospective study of the first two years of prosthetic loading, *J Oral Implantol* 25:185-197, 1999.
22. Kline R, Hoar JE, Beck GH: A prospective multicenter clinical investigation of a bone quality based dental implant system, *Implant Dent* 11:224-234, 2002.
23. Misch CE, Poitras Y, Dietsh-Misch F: Endosteal implants in the edentulous posterior maxilla—rationale and clinical results, *Oral Health* 90:7-16, 2000.
24. Misch CE, Steigenga J, Cianciola LJ et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 5-year case series study, *J Periodontol* 77: 1340-1347, 2006.
25. Roberts EW, Turley PK, Brezniak N et al: Bone physiology and metabolism, *J Calif Dent Assoc* 15:54-61, 1987.

WWW.HIGHDENT.IR

26. Klemetti E, Vaino P, Lassila V et al: Trabecular bone mineral density and alveolar height in postmenopausal women, *Scand J Dent Res* 101:166-170, 1993.
27. Mercier P, Inoue S: Bone density and serum minerals in cases of residual alveolar ridge atrophy, *J Prosthet Dent* 46:250-255, 1981.
28. Atwood DA, Coy WA: Clinical cephalometric and densitometric study of reduction of residual ridges, *J Prosthet Dent* 26:280-295, 1971.
29. Lavelle CLB: Biomechanical considerations of prosthodontic therapy: the urgency of research into alveolar bone responses, *Int J Oral Maxillofac Implants* 8:179-184, 1993.
30. Meier GH: Die architektur der spongiosa, *Arch Anat Physiol Wess Med* 34:615-628, 1887.
31. Kulmann C: *Die graphische Statik 1*, Aufl, Zurich, 1888, Meyer and Zeller.
32. Wolff J: *Das Gesetz der Transformation der Knochen*, Berlin, 1892, A Hirshwald.
33. Murry PDF: Bones: *A study of development and structure of the vertebral skeleton*, Cambridge, 1936, Cambridge University Press.
34. MacMillan HA: Structural characteristics of the alveolar process, *Int J Ortho* 12:722-730, 1926.
35. Parfitt AM: Investigation of the normal variations in the alveolar bone trabeculation, *Oral Surg Oral Med Oral Pathol* 15:1453-1463, 1962.
36. Harris EF, Baker WC: Loss of root length and crestal bone height before and during treatment in adult and adolescent orthodontic patients, *Ann J Orthod Dentofac Orthop* 98:463-469, 1990.
37. Neufeld JO: Changes in the trabecular pattern of the mandible following the loss of teeth, *J Prosthet Dent* 685-697, 1958.
38. Orban B: *Oral histology and embryology*, ed 3, St Louis, 1953, Mosby.
39. Enlow DH: *Principles of bone remodeling: an account of post-natal growth and remodeling processes in long bones and the mandible*, Springfield, Ill, 1963, Thomas.
40. Roberts WE, Smith RK, Zilberman Y et al: Osseous adaptation to continuous loading of rigid endosseous implants, *Am J Orthod* 86:96-111, 1984.
41. Garretto LP, Chen J, Parr JA et al: Remodeling dynamics of bone supporting rigidly fixed titanium implants. A histomorphometric comparison in four species including human, *Implant Dent* 4:235-243, 1995.
42. Rhinelander FW: The normal circulation of bone and its response to surgical intervention, *J Biomed Mater Res* 8:87-90, 1974.
43. Currey JD: Effects of differences in mineralization on the mechanical properties of bone. *Philos Trans R Soc Lond B Biol Sci* 1121:509-518, 1984.
44. Bidez MW, Misch CE: Force transfer in implant dentistry: basic concepts and principles, *J Oral Implantol* 18: 264-274, 1992.
45. Frost HM: Mechanical adaptation. Frost's mechanostat theory. In Martin RB, Burr DB: *Structure, function, and adaptation of compact bone*, New York, 1989, Raven Press.
46. Kazarian LE, Von Gierke HE: Bone loss as a result of immobilization and chelation: preliminary results in Macaca mulatta, *Chin Orthop Relat Res* 65:67-75, 1969.
47. Minaire MC Neunier P, Edouard C et al: Quantitative histological data on disuse osteoporosis: comparison with biological data, *Calcif Tissue Res* 17:57-73, 1974.
48. Uhthoff HK, Jaworski ZF: Bone loss in response to long-term immobilisation, *J Bone Joint Surg Br* 60-B:420-429, 1978.
49. Simmons DJ, Russell JE, Winter F: Space flight and the non-weight bearing bones of the rat skeleton, *Trans Orthop Res Sco* 4:65, 1981.
50. Ingebretsen M: Out of this world workouts, *World Traveler* Feb:10-14, 1997.
51. Oganov VS: Modern analysis of bone loss mechanisms in microgravity, *J Gravit Physiol* 11:143-146, 2004.
52. Mori S, Burr DB: Increased intracortical remodeling following fatigue damage, *Bone* 14:103-109, 1993.
53. Linkow LI, Chercheve R: *Theories and techniques of oral implantology*, vol 1, St Louis, 1970, Mosby.
54. Lekholm U, Zarb GA: Patient selection and preparation. In Brånemark P-I, Zarb GA, Albrektsson T, editors: *Tissue integrated prostheses: osseointegration in clinical dentistry*, Chicago, 1985, Quintessence.
55. Truhlar RS, Orenstein IH, Morris HF et al: Distribution of bone quality in patients receiving endosseous dental implants, *J Oral Maxillofac Surg* 55:38-45, 1997.
56. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, *Postgraduate Dentistry* 2:3-17, 1995.
57. Oh T, Yoon J, Misch CE et al: The cause of early implant bone loss: myth or science? *J Periodontol* 73:322-333, 2002.
58. Misch CE, Suzuki JB, Misch-Dietsh FD et al: A positive correlation between occlusal trauma and peri-implant bone loss—literature support. *Implant Dent* 14:108-116, 2005.
59. Manz MC: Radiographic assessment of peri-implant vertical bone loss: DICRG interim report no. 9, *J Oral Maxillofac Surg* 55:62-71, 1997.
60. Misch CE, Bidez MW, Sharawy M: A bioengineered implant for a predetermined bone cellular response to loading forces: a literature review and case report, *J Periodontol* 72:1276-1286, 2001.
61. Misch C: Progressive bone loading, *Dent Today* 12:80-83, 1995.
62. Trisi P, Rao W: Bone classification: clinical-histomorphometric comparison, *Clin Oral Implants Res* 10:1-7, 1990.
63. Orenstein IH, Synan WJ, Truhlar RS et al: Bone quality in patients receiving endosseous dental implants: DICRG interim report no. 1, *Implant Dent* 3:90-94, 1994.
64. Quirynen M, Naert I, van Steenberghe D et al: A study of 589 consecutive implants supporting complete fixed prostheses. Part I: periodontal aspects, *J Prosthet Dent* 8:655-663, 1992.
65. Rothman SLG: Interactive Implant Surgical Planning with Sim/Plan. In Rothman SLG: *Dental applications of computerized tomography: surgical planning for implant placement*, Chicago, 1998, Quintessence.
66. Genant HK: Quantitative computed tomography: update. *Calcif Tissue Int* 41:179-186, 1987.
67. Cann CE: Quantitative CT for determination of bone mineral density: a review, *Radiology* 166:509-522, 1988.
68. Rothman SLG: Computerized tomography of the mandible. In Rothman SLG: *Dental applications of computerized tomography: surgical planning for implant placement*, Chicago, 1998, Quintessence.
69. Kircos LT, Misch CE: Diagnostic imaging and techniques. In Misch CE, editor: *Contemporary implant dentistry*, ed 2, St Louis, 1999, Mosby.

WWW.HIGHDENT.IR

70. Todisco M, Trisi P: Bone mineral density and bone histomorphometry are statistically related, *Int J Oral Maxillofac Implants* 20:898-904, 2005.
71. Norton MR, Gamble C: Bone classification: an objective scale of bone density using the computerized tomography scan, *Clin Oral Implants Res* 12:79-84, 2001.
72. Homolka P, Beer A, Birkfellner W et al: Bone mineral density measurement with dental quantitative CT prior to dental implant placement in cadaver mandibles: pilot study, *Radiology* 224:247-252, 2002.
73. Aranyarachkul P, Caruso J, Gantes B et al: Bone density assessments of dental implant sites: 2. Quantitative cone-beam computerized tomography, *Int J Oral Maxillofac Implants* 20:416-424, 2005.
74. Ikumi N, Tsutsumi S: Assessment of correlation between computerized tomography values of the bone and cutting torque values at implant placement: a clinical study, *Int J Oral Maxillofac Implants* 20:253-260, 2005.
75. Shahlaie M, Gantes B, Schulz E et al: Bone density assessments of dental implant sites: quantitative computed tomography, *Int J Oral Maxillofac Implants* 18:224-231, 2003.
76. Misch CE, Qu Z, Bidez MW: Mechanical properties of trabecular bone in the human mandible implications of dental implant treatment planning and surgical placement, *J Oral Maxillofac Surg* 57:700-706, 1999.
77. Carter DR, Hayes WC: Bone compressive strength: the influence of density and strain rate, *Science* 194: 1174-1176, 1976.
78. Rice JC, Cowin SC, Bowman JA: On the dependence of the elasticity and strength of cancellous bone on apparent density, *J Biomech* 21:155-168, 1988.
79. Misch CE, Bidez MW: Implant protected occlusion, *Pract Periodontics Aesthet Dent* 7:25-29, 1995.
80. Kitagawa T, Tanimoto Y, Nemoto K et al: Influence of cortical bone quality on stress distribution in bone around dental implants. *Dent Mater J* 24:219-224, 2005.
81. Sevimay M, Turhan F, Kilicarsian MA et al: Three-dimensional finite element analysis of the effect of different bone quality on stress distribution in an implant-supported crown, *J Prosthet Dent* 93:227-234, 2005.
82. Tada S, Stegaroiu R, Kitamura E et al: Influence of implant design and bone quality on stress/strain distribution in bone around implants: a 3-dimensional finite element analysis, *Int J Oral Maxillofac Implants* 18 357-368, 2003.
83. Crupi B, Guglielmino E, LaRosa G et al: Numerical analysis of bone adaptation around an oral implant due to overload stress, *Proc Inst Mech Eng [H]* 218:407-415, 2004.
84. Ichikawa T, Kanitani H, Wigianto R et al: Influence of bone quality in the stress distribution—an in vitro experiment, *Clin Oral Implants Res* 8:18-22, 1997.
85. Bassi F, Procchio M, Fava C et al: Bone density in human dentate and edentulous mandibles using computed tomography, *Clin Oral Implants Res* 10:356-361, 1999.
86. Steigenga JT, Alshammari KF, Nociti FH et al: Dental implant design and its relationship to long term implant success, *Implant Dent* 12:306-317, 2003.
87. Petrie CS, Williams JL: Comparative evaluation of implant designs: influence of diameter, length and taper on strains in the alveolar crest: a three-dimensional finite element analysis, *Clin Oral Implants Res* 16:486-494, 2005.
88. Rieger MR, Adams WK, Kinzel GL et al. Finite element analysis of bone-adapted and bone-bonded endosseous implants, *J Prosthet Dent* 62: 436-440, 1989.
89. Schenk RK, Buser D: Osseointegration: a reality, *Periodontology 2000* 17:22-35, 1998.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 8

Treatment Plans Related to Key Implant Positions and Implant Number

Carl E. Misch

In the past, treatment planning for implant dentistry was primarily driven by the existing bone volume in the edentulous sites. As a result, distal cantilevers were extended from anterior implants or shorter implants were placed in the posterior regions of the mouth. A second historical phase of treatment planning has since developed based on esthetics. In this scheme, implant positions were primarily controlled by the teeth being replaced.

The primary causes of complications in implant dentistry are related to biomechanics.[1] For example, early loading failures outnumber surgical healing failures, especially in soft bone, when forces are greater than usual or implant sizes are shorter than 10 mm. Misch developed a treatment plan sequence to decrease the risk of biomechanical overload, consisting of following[2]:

1. Prosthesis design
2. Patient force factors
3. Bone density in the edentulous sites
4. Key implant positions and number
5. Implant size
6. Available bone in the edentulous sites
7. Implant design

This chapter will consider the key implant positions for a prosthesis, followed by the overall number of implants to support the restoration.

ABUTMENT OPTIONS

Several treatment options are available for the adequate restoration of an edentulous segment. As a general rule, implant-supported prostheses independent from natural adjacent teeth are designed whenever possible. This concept reduces the risk of decay on the natural tooth margin next to the adjacent pontic or abutment. The incidence of decay on a tooth splinted in a fixed partial denture accounts for 22% of the complications within 10 years, whereas individual crowns have less than 1% risk of decay within this time frame.[3] Unrestored natural teeth have less risk of decay, and implants do not decay at all. A second common complication of teeth-supported fixed prosthetic restorations is endodontic-related factors that occur in approximately 15% of cases within 10 years. Implant abutments do not require endodontic procedures, and unsplinted natural tooth crowns have less endodontic procedures. Independent implant prostheses may also reduce or eliminate pontics, while simultaneously increasing the number of abutments and distributing forces more effectively. The increase in abutment number decreases the risk of an unretained restoration, which is the third most common fixed prosthesis complication supported by natural teeth. Therefore independent implant prostheses cause fewer complications and exhibit greater long-term success rates of the prosthesis and greater survival rates of the adjacent teeth.[4] However, when an implant restoration is joined to a natural tooth, an increased risk of abutment screw loosening, implant marginal bone loss, and unretained restoration occurs. In addition, the distribution of occlusal forces is optimized when independent implant prostheses are designed. As a result, the ideal treatment plan for a partially edentulous patient includes an independent implant restoration.

Key Implant Positions

Some implant positions are more critical than others in regard to force reduction. There are four general guidelines to determine key implant positions (Box 8-1):

1. Cantilevers on the prosthesis should be reduced and preferably eliminated; therefore the terminal abutments in the prosthesis are key positions.

Box 8-1 Guidelines for Key Implant Positions

No cantilevers
No three adjacent pontics
Canine-molar rule
Arch dynamics

WWW.HIGHDENT.IR

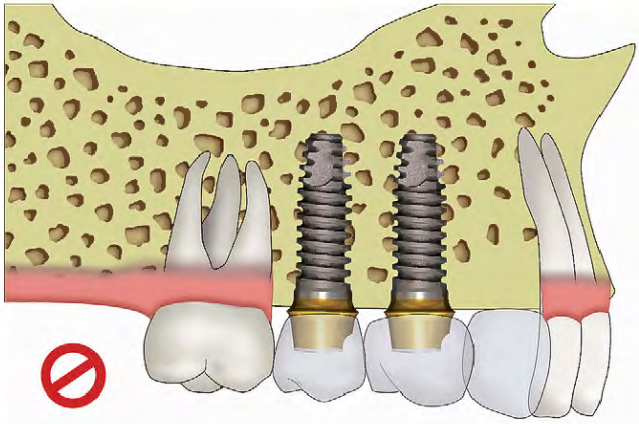


Figure 8-1 Ideal key implant positions include the terminal abutment positions when adjacent teeth are missing. Without a terminal abutment, a cantilever exists on the restoration, which increases complications. This is particularly important when a lateral load or heavy bite force is applied to the cantilever.

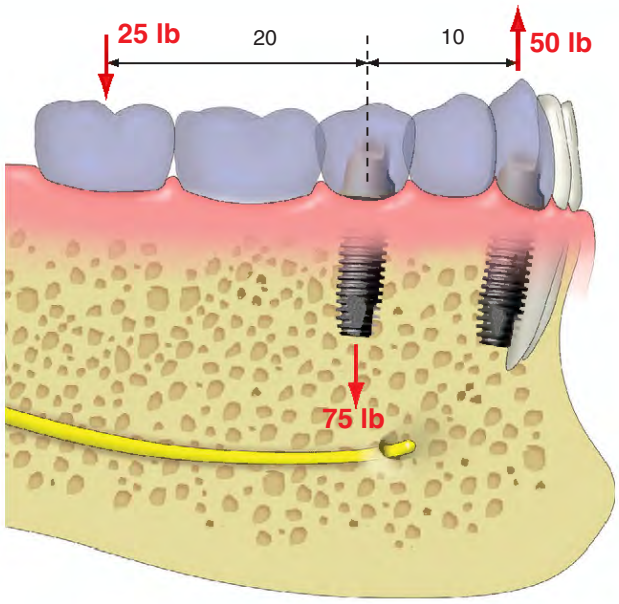


Figure 8-2 A cantilever on two implants may be considered a Class 1 lever. When the center of each implant is 10 mm apart, with a 20-mm cantilever, a mechanical advantage of 2 is created. Therefore the load on the cantilever will be multiplied by 2 on the far implant, and the implant close to the cantilever receives the total stress of the two loads.

2. Three adjacent pontics should not be designed in the prosthesis.
3. The canine and first molar sites are key positions, especially when adjacent teeth are missing.
4. An arch is divided into five segments. When more than one segment of an arch is being replaced, a key implant position is at least one implant in each segment.

No Cantilevers

The first rule for ideal key implant positions is that no cantilever should be designed in the prosthesis (Figure 8-1). Cantilevers are force magnifiers to the implants, abutment screws, cement or prosthesis screws, and implant-bone interface. Cantilevers on fixed partial dentures supported by teeth have a higher complication rate than prostheses with terminal abutments, including unretained restorations. This is especially noted with parafunction or reduced crown height spaces.[5] Therefore the ideal key implant positions include the terminal abutment positions when adjacent teeth are missing.

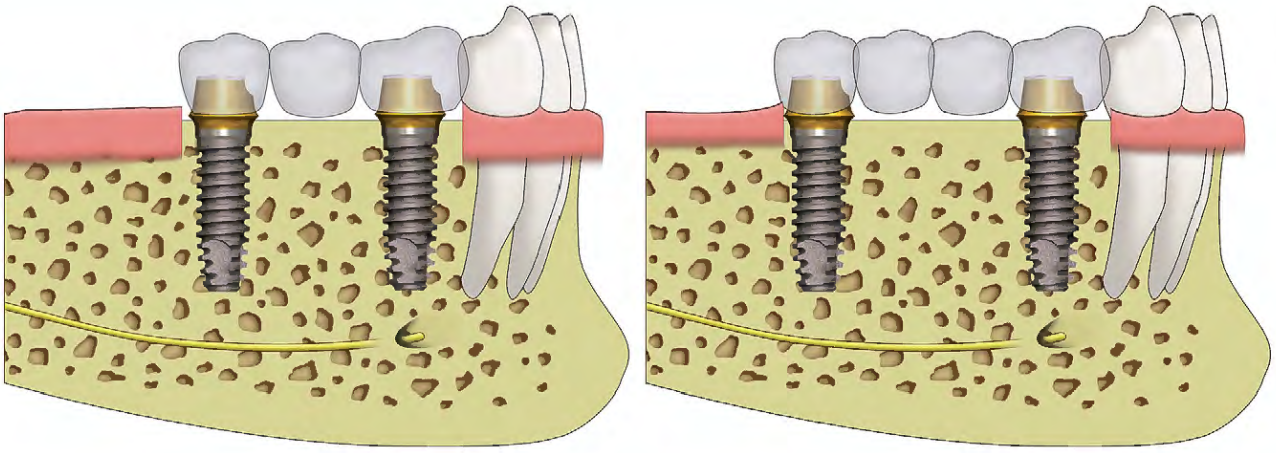
The length of the cantilever is directly related to the amount of the additional force placed on the abutments of the prosthesis. Therefore when a 25-lb force is placed along the long axis of an implant, the implant system (i.e., crown, cement, abutment, abutment screw, implant body, implant marginal bone, and implant bone interface) receives a 25-lb load. When a force of the same magnitude (25 lb) is applied on a 10-mm cantilever, the moment force on the abutment is increased to a 250-lb millimeter force. As a result, any part of the implant system is at an increased risk of biomechanical failure (e.g., porcelain fracture, uncemented prosthesis, abutment screw loosening, crestal bone loss, implant failure, implant component or body fracture).

A cantilevered restoration on multiple implants may be compared with a class I lever. The extension of the prosthesis from the last abutment is the *effort arm* of the lever. The last abutment next to the cantilever acts as a fulcrum when a load is applied to the lever. The distance between the last abutment and the farthest abutment from the end of the cantilever represents the *resistance arm* and may be called the anteroposterior distance or A-P spread of the implants. The length (usually in millimeters) of the cantilever (effort arm) divided by the resistance arm represents the *mechanical advantage.* Therefore, when two implants are 10 mm apart with a cantilever or extension of 20 mm, the mechanical advantage is two (20 mm/10 mm). In this example, a 25-lb force on the cantilever results with a 50-lb force on the farthest abutment from the cantilever (25 lb × 2 = 50 lb). The abutment closest to the cantilever (fulcrum) receives a force equal to the sum of the other two forces, or, in this example 75 lb (25 lb + 50 lb) (Figure 8-2). Therefore cantilevers magnify forces to all the abutments supporting the prosthesis.

To enforce the rule of no cantilever, the key implant positions when one or two adjacent teeth are missing indicate one implant per tooth. When 3 to 14 adjacent teeth are missing, the key implant positions include the two terminal abutments, one on each end of the prosthesis. A three- to four-unit prosthesis may be fabricated with only these abutments, when nearly all the force factors are low and the bone density is favorable (Figure 8-3). Restorations of 5 to 14 units require additional abutments.

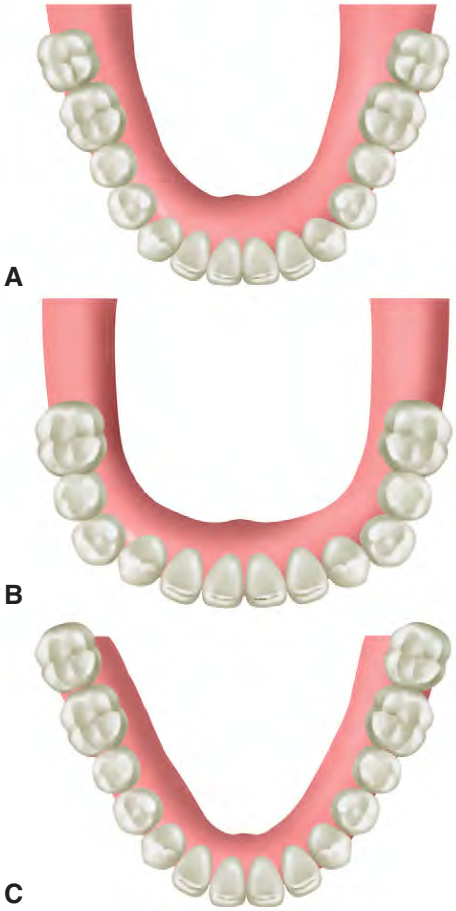
The ideal treatment plan should eliminate cantilevers. However, in some clinical conditions, a cantilever is the most prudent treatment option. For example, in

WWW.HIGHDENT.IR



A B

Figure 8-3 **A,** When three adjacent teeth are missing, the terminal abutments are key implant positions. When all patient force factors are low and bone density is good, these abutments may be adequate to replace the three missing teeth. **B,** When four adjacent teeth are missing, the terminal abutments are key implant positions. Rarely are these two implants sufficient to replace four posterior teeth.



A B C

Figure 8-4 The anteroposterior distance (A-P spread) of five implants in the mandible is measured from the distal of the last two implants to the mid position of the most anterior implant. Because these splinted implants form an arch, the cantilever may extend up to 2.5 times the A-P distance (when patient force factors are low and bone density is good). Arch shape affects the A-P distance. The ovoid arch form **(A)** often has an A-P distance of 6 to 8 mm. A square arch form **(B)** often has an A-P dimension of 2 to 5 mm. A tapered arch form **(C)** has the greatest A-P distance, larger than 8 mm.

an edentulous mandible, available bone in the posterior regions may be insufficient for root form implant placement, without advanced procedures (e.g., nerve repositioning, iliac crest bone grafts). An alternative treatment plan may be to cantilever pontics from anterior implants. However, when terminal abutments are not designed in the treatment plan and a cantilever is planned, other force and surface area factors should compensate for the increase in force. When this option is considered, the force factors of parafunction, crown height, masticatory dynamics, implant location, and opposing arch are closely scrutinized. In addition to force modifiers, the A-P distance (A-P spread) of the distal and anterior implants is also a factor. When the implants are in one plane, the cantilever should rarely extend farther than the A-P distance, regardless of how low the patient force factors (see Figure 8-1). When the force factors are unfavorable, the cantilever length should be reduced or eliminated, the implant number increased, the implant size increased, or the implant design surface areas increased. When five or more implants are positioned around an arch, and several different plans exist because of the arch form of the splinted implant, the cantilever may extend as far as 2.5 times the A-P distance when force factors are low and density is favorable (Figure 8-4). However, more than two pontics are not indicated on a posterior cantilever, even under ideal conditions.

When the cantilever on the prosthesis is represented by only a lateral incisor, the ideal implant position may not include the lateral incisor position when bite forces are low to moderate. The lateral incisor is the smallest tooth in the arch, so the cantilever is limited. The anterior bite forces are lowest in the central and lateral incisor region. The occlusal contact on the pontic may be eliminated and further reduce risk of overload.

WWW.HIGHDENT.IR

When a pontic replaces the lateral incisor, the soft tissue drape may be improved compared with an implant. As a result, the ideal implant position based on force and esthetics may be a larger-diameter implant in the canine position (i.e., 5-mm versus 4-mm diameter) and a cantilever pontic to replace the lateral incisor.

The fact that, on occasion, a cantilever may be acceptable when force factors are low and bone density is favorable does not negate the ideal goal that no cantilever should be designed in the prosthesis. Therefore when three or more adjacent teeth are missing, the terminal abutments at each end of the prosthesis are first designed in the treatment plan. When this option is not readily available, additional implants, larger implant sizes, and greater implant surface area design are indicated. In addition, the forces to the cantilevered portion of the prosthesis should be reduced.

No Three Adjacent Pontics

In most prostheses designs, three adjacent pontics are contraindicated on implants, just as they are contraindicated on natural abutments.[6] The adjacent abutments are subjected to considerable additional force when they must support three missing teeth, especially in the posterior regions of the mouth. In additions, all pontic spans between abutments flex under load. The greater the span between abutments, the greater the flexibility of the metal in the prosthesis. The greater the load, the greater the flexure. This metal flexure places shear and tensile loads on the abutments (see Chapter 25).[7] The greater the flexure, the greater the risk of porcelain fracture, uncemented prostheses, and abutment screw loosening.

A one-pontic span exhibits little flexure—8 µm or less with precious metal under a 25-lb load.[8] A two-pontic span flexes eight times more than a one-pontic span, all other variables being equal. A three-pontic span flexes 27 times more than a one-pontic span (Figure 8-5).[7-9] As a result, not only is the magnitude of the force increased to the adjacent abutments when the prosthesis has three pontics (because they are supporting two abutments and three pontics), but the flexure of the metal increases to a point that the incidence of complications make the treatment plan contraindicated, especially when forces are greater (as in the posterior region).

The flexure of materials in a long span is more of a problem for implants than natural teeth. Because natural roots have some mobility both apically and laterally, the tooth acts as a stress absorber and the amount of material flexure may be reduced. Because an implant is more rigid than a tooth (and also has a greater modulus of elasticity than a natural tooth), the complications of increased load and material flexure are greater for an implant prosthesis. Therefore because three posterior pontics are contraindicated in a natural tooth–fixed prosthesis, it is even more important not to have three pontics in an implant restoration. Angled forces magnify the amount of the force to the implant system; therefore most maxillary anterior prostheses should also limit the number of pontics in the restoration.

The span of the pontics in the ideal treatment plan should be limited to the size of two premolars, which is 13.5 to 16 mm. When a molar is one of the teeth missing between existing teeth, the missing molar space may be 10- to 14-mm long. Therefore when the span is greater than 14 mm, two pontics should be considered to replace the molar. As a result, when a second premolar and first molar are missing, this span is often treatment-planned to replace three teeth, rather than two. In other words, a missing tooth span is often related to the missing number of roots in the mandible and number of buccal roots in the maxilla. This is especially appropriate for greater patient forces (i.e., moderate to severe parafunction) or softer bone types (i.e., D3 and D4) (Figure 8-6). As a result of these

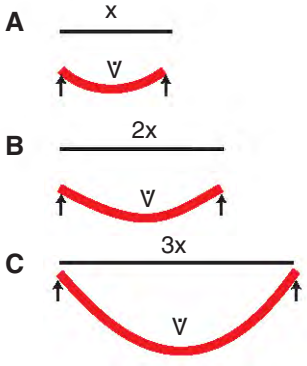


Figure 8-5 Metal flexure is related to the cube of the distance. **A,** When one pontic is present, the metal flexes (x). **B,** When the fixed prosthesis has two pontics ($2x$), the metal flexes $2 \times 2 \times 2 = 8$ times more. **C,** When three pontics exist ($3x$), the metal flexes $3 \times 3 \times 3 = 27$ times more than a one-pontic restoration.

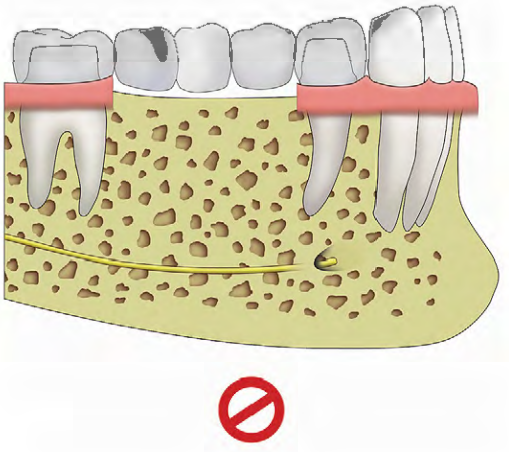


Figure 8-6 When adjacent missing posterior teeth include a molar, the missing molars may be regarded as two premolar teeth when the space is 14 mm or more. Therefore when a first molar and second premolar are missing, the treatment plan may consider replacement of three adjacent teeth.

WWW.HIGHDENT.IR

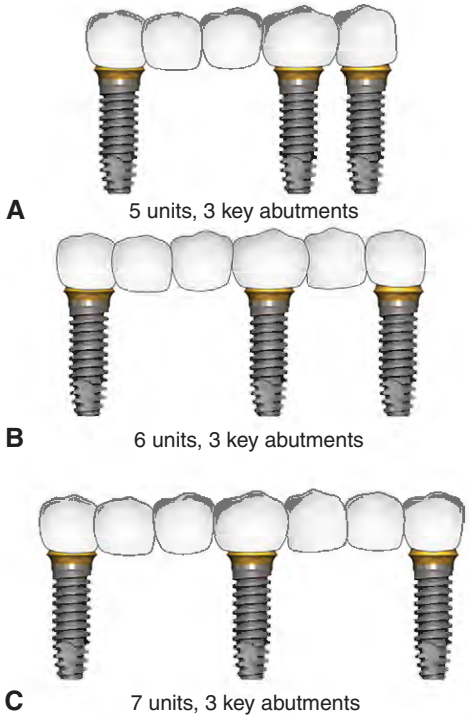


Figure 8-7 A five- to seven-unit fixed prosthesis has three key positions for the abutments. The terminal abutment follows Rule 1 (no cantilever), and a one-pier abutment is positioned following Rule 2 (no three adjacent pontics). Rarely are these three abutments sufficient to support the prosthesis in the long term. Additional abutments are required when force factors are moderate to severe or bone density is poor around the implant.

guidelines, an edentulous arch missing 14 natural teeth may have 18 potential implant sites. Rarely are second molars replaced; therefore when 12 natural teeth are considered in an edentulous arch, 14 potential implants sites are present.

To limit the effect of the complications of three adjacent premolar size pontics, additional key implant positions are indicated in prostheses missing more than four adjacent teeth. To have three potential pontics in a restoration, at least four adjacent posterior teeth or five adjacent anterior and premolar teeth must be missing (when terminal abutments are present). Therefore, when 4 to 14 missing adjacent teeth are to be replaced, key implant positions are located in the terminal abutments and additional pier or intermediary abutments are indicated to limit the pontic spans to 2 premolar-sized pontics or less. Following this rule, a five to seven premolar-sized unit prosthesis has three key abutments (two terminal and one pier) (Figure 8-7). An 8 to 10 premolar-sized unit prosthesis has 4 key implant positions (2 terminal and 2 pier). An 11- to 13-unit prosthesis has 5 key abutments (2 terminal and 3 pier), and a 14-unit prosthesis has 6 key abutment positions. In addition to these key abutments, additional abutments are usually needed to address force factors and bone density. Rarely is the force factor situation favorable and bone density ideal enough to be fulfilled with solely key abutments for a fixed prosthesis.

Canine and First Molar Sites

A fixed restoration replacing a canine is at greater risk than nearly any other restoration in the mouth. The maxillary or mandibular adjacent incisor is one of the weakest teeth in the mouth, and the first premolar is often one of the weakest posterior teeth. A traditional fixed prosthetic axiom indicates it is contraindicated to replace a canine and two or more adjacent teeth.[6,10] Therefore if a patient desires a fixed prosthesis, implants are required whenever the following adjacent teeth are missing in either arch: (1) the first premolar, canine, and lateral incisor; (2) the second premolar, first premolar, and canine; and (3) the canine, lateral, and central incisors (Figure 8-8). Whenever these combinations of teeth are missing, implants are required to restore the patient because: (1) the length of the span is three adjacent teeth, (2) the lateral direction of force during mandibular excursions increase the stress, and (3) the magnitude of the bite force is increased in the canine

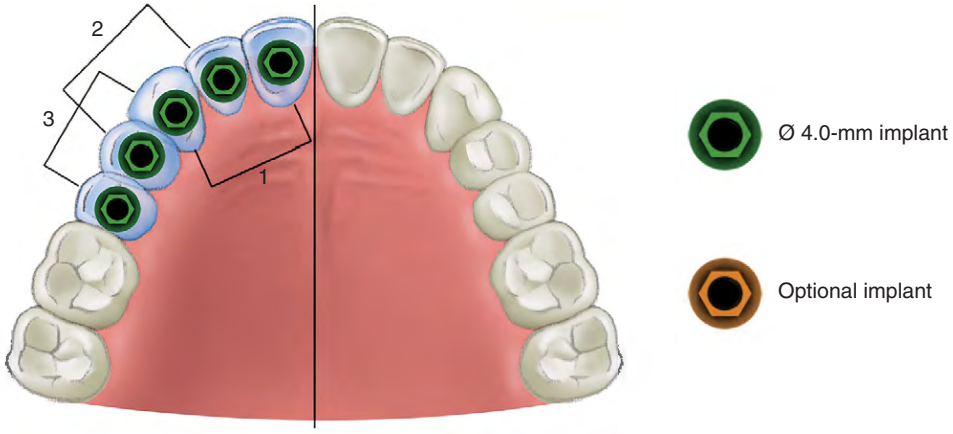


Figure 8-8 Whenever the canine and three adjacent teeth are missing, implants are required to support a fixed prosthesis. Therefore when *(1)* the canine, lateral, and central; *(2)* the lateral, canine, and first premolar; or *(3)* the canine, first, and second premolar teeth are missing, implants are indicated for a fixed prosthesis.

WWW.HIGHDENT.IR

Box 8-2 Implant Positions: Key Locations

3 to 4 units

Terminal abutments

5 to 14 units

Terminal abutments
Limit to two pontics

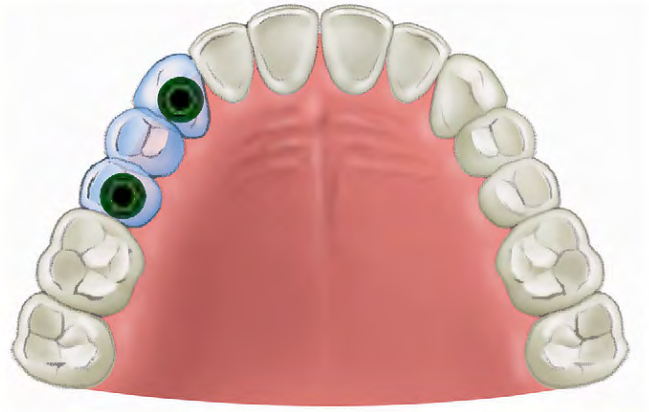


Figure 8-9 When the canine, first premolar, and second premolar teeth are missing, key implant positions are in the canine and second premolar positions to support these three teeth.

region compared with the anterior region. Therefore, under these conditions, at least two key implant positions are required to replace these three adjacent teeth, usually in the terminal positions of the span (especially when one of the terminal abutments is the canine position) (Box 8-2; Figures 8-9 and 8-10).

When the three adjacent teeth are the first premolar and canine and lateral incisors, the key implant positions are the first premolar and the canine. These positions result in an anterior cantilever to replace the lateral incisor. However, because the lateral incisor is the smallest tooth in the arch and in the anterior region has the least bite force, the cantilever is of limited negative impact. In addition, the canine implant is usually larger than a lateral incisor implant for the esthetic requirements of the restoration. This further reduces the effect of the cantilever (Figure 8-11). In addition, the occlusion is modified so that no occlusal contact is present on the lateral incisor pontic in centric occlusion or excursions of the mandible. When force factors are greater than usual, a small-diameter implant may also be used to support the lateral incisor, and three implants with no cantilever reduce the increased force factor risks.

When there are multiple missing teeth and the canine edentulous site is a pier abutment position, the canine position is a key implant position to help disocclude the posterior teeth in mandibular excursions. As a

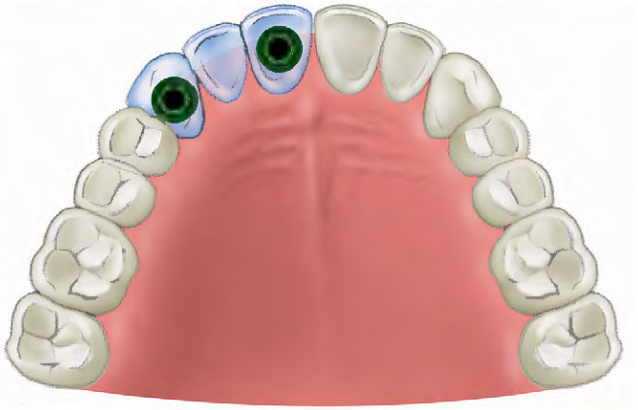


Figure 8-10 When the canine, lateral, and central incisor teeth are missing, key implant positions are in the central and canine positions to support these three teeth.

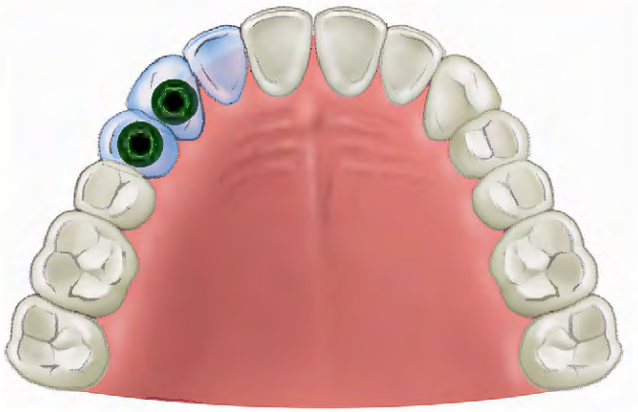
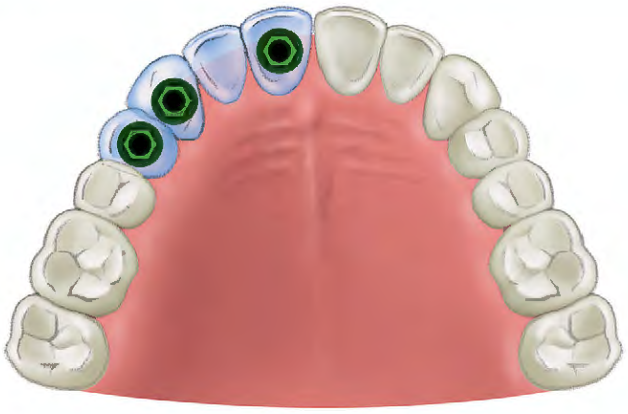


Figure 8-11 When the first premolar, canine, and lateral incisors are missing, the key implant positions are the first premolar and canine position. Although this may result in a cantilever on the restoration, the lateral incisor is the smallest tooth, the anterior region has less bite force, and the canine implant may be larger than the lateral incisor implant for esthetic requirements.

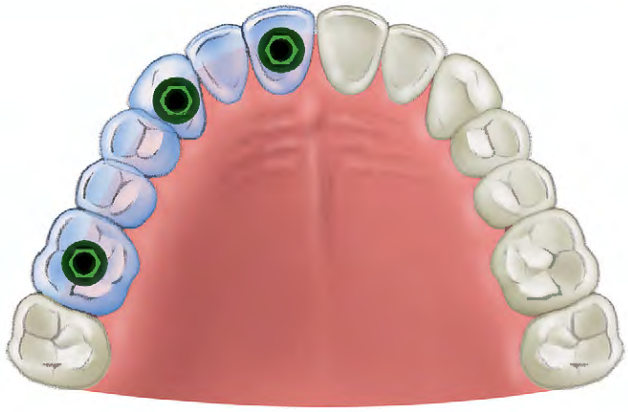
result, when four or more adjacent teeth are missing, including a canine and at least one adjacent posterior premolar tooth, the key implant positions are the terminal abutments, the canine position, and additional pier abutments, which limit the pontics spans to no more than two teeth (Figure 8-12).

The first molar is also a key implant position when three adjacent posterior teeth are missing. The bite force doubles in the molar position compared with the premolar position in both the maxilla and mandible. In addition, the edentulous span of a missing first molar is 10 to 12 mm, compared with a 7-mm span for a premolar. As a result, when three or more adjacent teeth are missing, including a first molar, the key implant positions include the terminal abutments and the first molar position (Figure 8-13). For example, in a patient missing the second premolar, first molar, and second molars, three key implant positions are needed to restore the full contour of the missing molars teeth: the

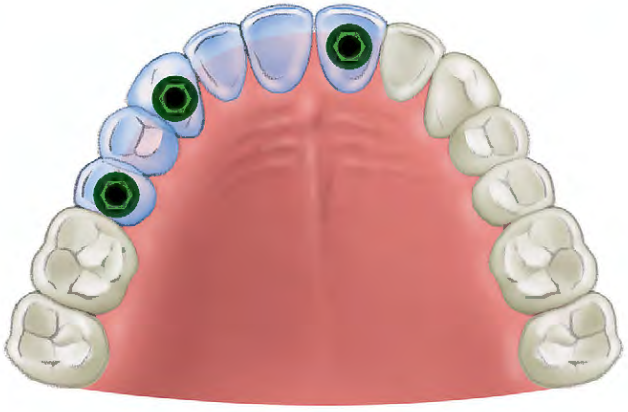
WWW.HIGHDENT.IR



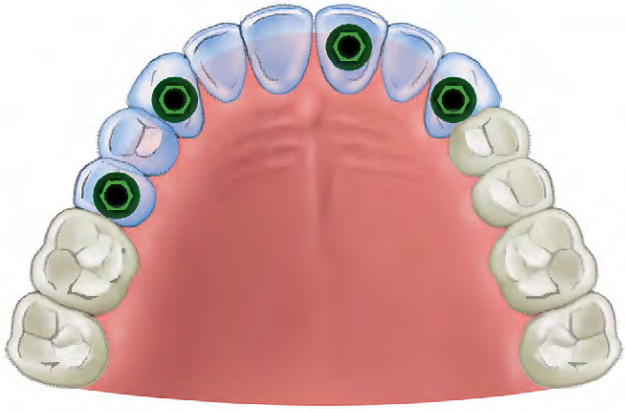
A



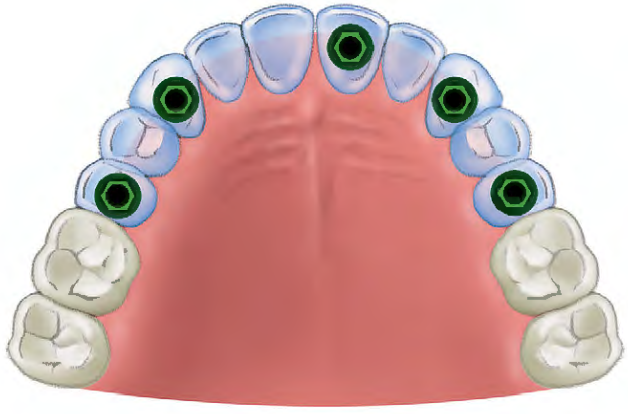
B



C



D



E

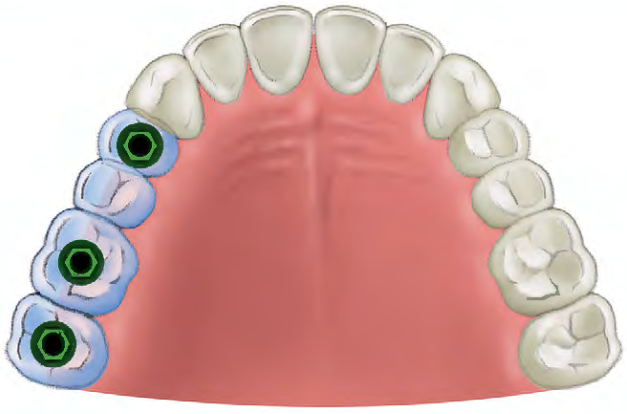
Figure 8-12 **A,** When the central, lateral, canine, and first premolar are missing, the ideal key implant positions are the central and first premolar (Rule 1, no cantilever) and the canine position (Rule 3, the canine and first molar position). **B,** When the central, lateral, canine, first premolar, second premolar, and first molar are missing, the three key implants positions are the central and first molar sites (Rule 1), and the canine site (Rules 2 and 3, no three adjacent pontic and canine and first molar position). **C,** When the central, central, lateral, canine, first premolar, and second premolar are missing, there are three key implant positions: the central and second premolar (Rule 1, no cantilever) and the canine position (Rule 3, the canine and first molar position). **D,** When eight adjacent teeth are missing from second premolar to the opposite canine, there are four key implant positions: The canine and second premolar position (Rule 1), the opposite canine (Rule 3), and one of the central incisor positions (Rule 2). **E,** When 10 adjacent teeth are missing from second premolar to second premolar, there are 5 key implant positions: the 2 second premolar sites (Rule 1), the 2 canine sites (Rule 3), and one of the central incisor positions (Rule 2).

second premolar and second molar terminal abutments and the first molar pier abutment (Figure 8-14). When one implant replaces a molar (for a span of less than 13 mm), the implant should be at least 5 mm in diameter. When a smaller-diameter implant is selected, the molar may be considered the size of two premolars.

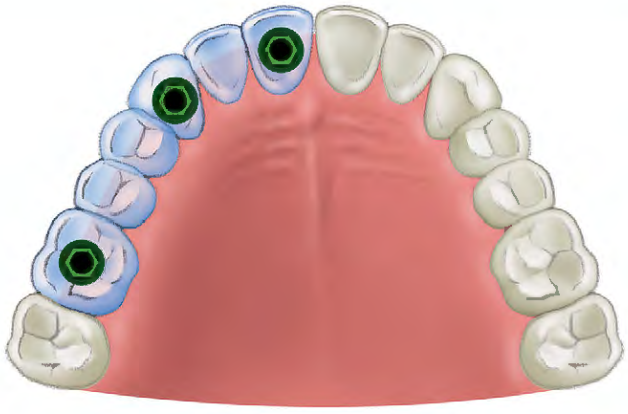
Key Arch Positions

An arch may be divided into five segments, similar to an open pentagon (Figure 8-15). The two central and two lateral incisors are one segment, the canines are independent segments, and the premolars and molars on each side form a segment. In other words,

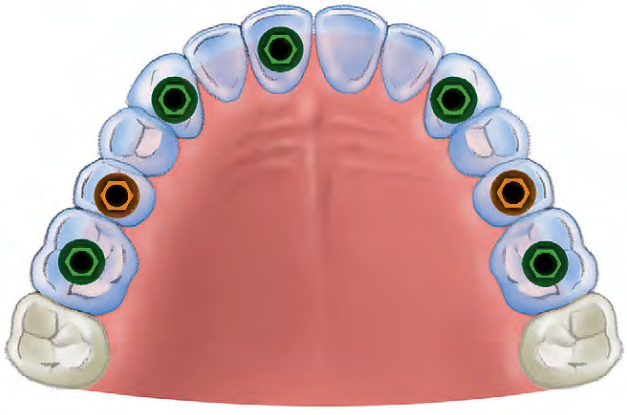
WWW.HIGHDENT.IR



A



B



C

Figure 8-13 **A,** When the patient is missing four teeth from the first premolar to second molar, there are three key implant positions to replace the four teeth: the first premolar and second molar sites (Rule 1) and the first molar position (Rule 3). **B,** When the patient is missing six adjacent teeth from the central incisors to the first molar, there are three key implant positions: The central and first molar position (Rule 1) and the canine position (Rule 3). When a larger implant cannot be inserted into the molar site, an additional implant is required to follow Rule 2. **C,** When the patient is missing teeth from first molar to first molar, there are five key implant positions: The two first molars (Rule 1), the two canines (Rule 3), and a central incisor (Rule 2). Additional implants in the posterior region are indicated when a larger-diameter implant is not positioned in the first molar sites (Rule 2). Implants in the second premolar site are also usually indicated when force factors are moderate or bone density is D3. Even more implant support is suggested when force factors are severe or bone density is D4.

each segment is essentially a straight line, with little inherent biomechanical advantage to a lateral force. However, when two or more segments of an arch are connected, the tripod effect is greater, and, as a benefit, an A-P distance (A-P spread) is created from the most distal terminal abutments to the most anterior pier abutment.

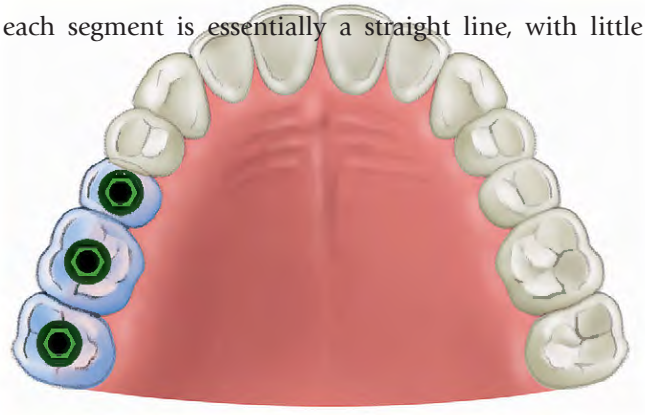


Figure 8-14 In a patient missing the second premolar and first molar and second molars, three key implant positions are needed to restore the full contour of the missing molars teeth: the second premolar and second molar terminal abutments and the first molar pier abutment.

When multiple adjacent missing teeth extend beyond one of the open pentagon segments, a key implant position needs to be situated within each segment. Therefore if the patient is edentulous from first premolar to first premolar, the key implant positions include the terminal abutments (the two first premolars), the two canines, and either of the central incisor positions (Figure 8-16). These implant positions follow the rules of: (1) no cantilever, (2) no three adjacent pontics, (3) the canine position, and (4) at least one implant in each edentulous segment of an arch.

IMPLANT NUMBER

In the past, the number of implants most often was determined in function of the amount of available bone in the mesiodistal dimension. For example, in an edentulous arch, six implants were used in abundant bone between the mental foramina anterior to the maxillary sinuses for a full-arch fixed prosthesis, whereas four implants were used in moderate to severe resorption for a fixed full-arch prosthesis (Figure 8-17).[11] However, this treatment option does not consider the force magnifiers of crown or height space or the A-P

WWW.HIGHDENT.IR

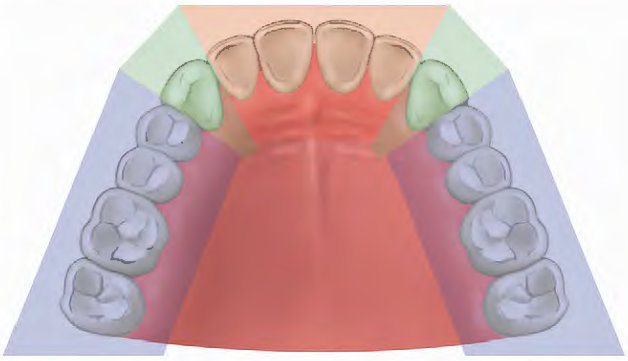


Figure 8-15 An arch may be considered as an open pentagon: the two premolar and molar sites, the two canine sites, and the central and lateral incisors represent the five sides.

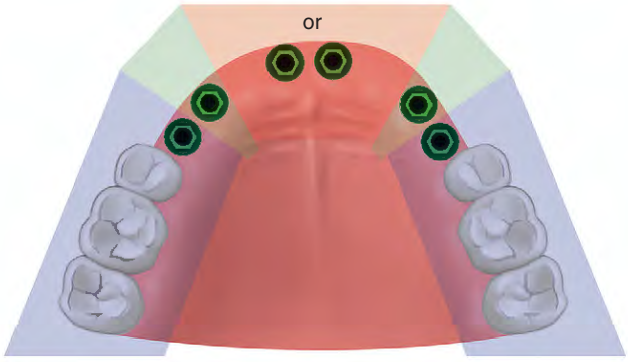


Figure 8-16 When a patient is missing eight teeth from first premolar to first premolar, there are five key positions: the first premolar sites (Rule 1, no cantilever and Rule 4, one implant in each open pentagon segment), the two canines (Rule 3, the canine and first molar rule and Rule 4, an implant in each pentagon segment), and an implant in one of the central incisor positions (Rule 2, no three adjacent pontics and Rule 4, an implant in each open pentagon segment).

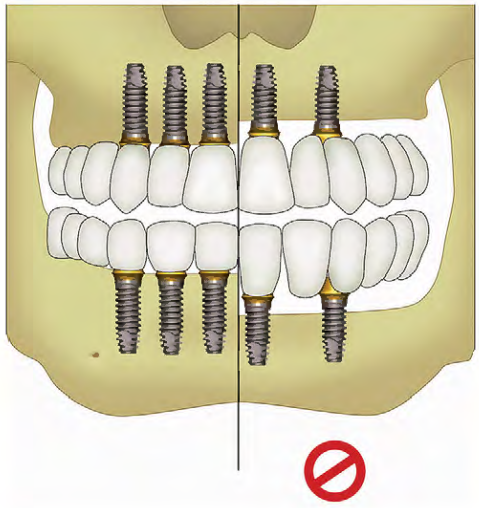


Figure 8-17 In the past, an edentulous arch used six implants to support a fixed prosthesis in abundant bone situations and four implants to support a complete full-arch prosthesis when minimal bone volumes were present.

Table 8-1 Implants versus Prosthesis Success: Four Implants per Prosthesis for 25 Patients (100 Implants for 25 Prostheses)

IMPLANT NUMBER (SUCCESS RATE)	FOUR IMPLANTS PER PROSTHESIS	PROSTHESIS SUCCESS RATE
100 (100%)	25	100%
90 (90%)	15	60%
80 (80%)	5	20%
75 (75%)	0	0%

distance (A-P spread) of the implants in relationship to the bilateral posterior cantilevers replacing the posterior teeth.

When four implants support a 12-unit fixed prosthesis, the position of the implants cannot follow the four key implant position rules, and include either four pontics between the anterior implants or three pontics cantilevered from the most distal implants. In addition, the number of implants in a treatment plan should rarely use a minimum number. There is no safety factor if an implant fails. For example, if 25 patients receive four implants to support a fixed prosthesis, there would be 25 fixed prostheses and 100 implants. If each patient lost one implant, there would only remain three implants and, as a result, nearly all 25 fixed prostheses would be at risk of overload failure. If 20% of the implants fail (with 1 failure per patient), only five of the 25 patients would have 4 implants to support the restoration (20% prosthesis success). This type of treatment planning may initially be less expensive for the patient, but an implant failure any time after implant surgery places the patient's restoration at considerable risk (Table 8-1).

If the 25 edentulous patients have eight implants to support a full-arch, 12-unit fixed prosthesis, the risks of prosthesis failure is significantly reduced (Table 8-2). If each patient loses one implant, most likely all patients would still be able to function with their original prosthesis. Even if all 25 patients lost two implants, the 25 prostheses may still function without risk (depending on the implant failure location). The additional implants also reduce the cantilever length or reduce the number of pontics in the prosthesis, providing more abutments for greater retention of the restoration, with reduced risk of screw loosening or uncemented prosthesis.

The key implant positions are often not enough support for the implant restoration, unless all patient force factors are all low (e.g., parafunction, masticatory dynamics, crown height) and the bone density is good (D1, D2). Therefore most often additional implants (besides the key implants) are added to the treatment plan.

WWW.HIGHDENT.IR

Table 8-2 Implants versus Prosthesis Success: Eight Implants per Prosthesis for 25 Patients (200 Implants for 25 Prostheses)

IMPLANTS (SUCCESS RATE)	SIX TO EIGHT IMPLANTS PER PROSTHESIS	PROSTHESIS SUCCESS RATE
200 (100%)	25	100%
175 (87.5%)	25	100%
150 (75%)	25	100%

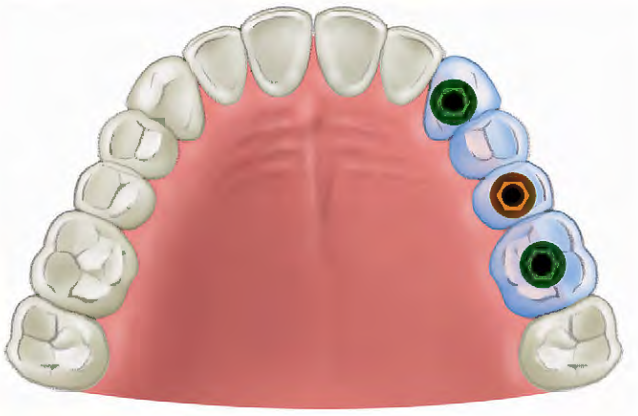


Figure 8-18 When four adjacent teeth are missing from the canine to first molar, the two terminal key implant positions are usually inadequate to support the prosthesis. One or two additional implants are required in most clinical situations (depending on patient force factors and bone density). The additional implant of choice is in the second premolar site, especially when a larger-diameter implant is not placed in the molar site.

One of the most efficient methods to increase surface area and decrease stress is to increase the implant number. For example, only two implant key positions as terminal abutments for a four-unit implant prosthesis in the canine and posterior region represent inadequate implant support, unless patient force factors are low, bone density is ideal, and implant size is not compromised. In most situations, three implants to replace four missing teeth is an ideal implant number. When force factors are high and bone density is poor (i.e., posterior maxilla), four implants to replace four teeth is often appropriate (see Figure 8-13; Figure 8-18).

Previous studies have shown that three abutments for a five-tooth span distribute stress more favorably than do two abutments for the same span. The one additional implant may decrease the implant reaction force by two times and reduce metal flexure fivefold. In addition, in the three-abutment scenario, moment forces are reduced.[12,13] In full-arch prostheses, studies comparing six implants and four implant abutments show better distribution and reduced stress on the six-implant system components (crown, cement, abutment, abutment screw, marginal bone, implant-bone interface, and implant components).[14]

The decision on the number of implants in the treatment plan begins with the implants in the ideal key positions. Additional numbers are most often required, and primarily related to the patient force factors or to bone density in the edentulous sites. Therefore in a young, large male who bruxes severely, with greater than normal crown height space in the posterior regions of the mouth, opposing an implant restoration will require one implant for each missing root (two implants for each molar). Likewise, patients with moderate force factors and poor bone density (D4 bone) in the implant sites may also require this many implants.

As a general observation, the number of implants to replace all of the mandibular teeth range from five to nine, with at least four between the mental foraminae. When fewer than six implants are used, a cantilever must be designed in a fixed prosthesis as a result of the mandibular flexure. Cantilevers in the mandible should ideally be projected in only one posterior quadrant to increase the A-P distance and reduce the force to the implants (Figure 8-19, *A*). When implants are positioned in four of the five open pentagon positions in the mandible, a cantilever is at a reduced risk of overload because of favorable dynamics of an arch, increased large A-P distance, and favorable bone density. When seven or more implants are used, two separate restorations may be fabricated with no posterior cantilever to permit mandibular flexure and torsion (Figure 8-19, *B*). Usually the second molar is not replaced in the edentulous mandible. A greater number of implants are generally required in the maxilla to compensate for the less dense bone and more unfavorable biomechanics of the premaxilla, and range from 7 to 10 implants with at least 3 implants from canine to canine (Figure 8-20).

In most situations, an implant should be positioned at least 1.5 mm from an adjacent natural tooth and 3 mm from an adjacent implant.[15-21] Using these guidelines, each 4-mm-diameter implant requires 7 mm of mesiodistal space (Figure 8-21). Therefore the maximum number of implants between adjacent teeth can be calculated by taking the crest module of an implant (e.g., 4.0 mm) and adding these dimensions (Figure 8-22). For example, an edentulous span of 21 mm is required for three adjacent implants 4 mm in diameter and 28 mm for four adjacent implants between two teeth. As a general rule, it is better to err on the side of safety in numbers than on the side of too few implants. Therefore, when in doubt, add an additional implant to the treatment plan.

Commonly, implant-supported crowns in the posterior regions of the mouth are the size of premolars. This concept often permits the placement of two implants to replace an intratooth molar, when the span is at least 14 mm for 4-mm-diameter implants (3 mm between each implant and 1.5 mm from the adjacent teeth). When the missing molar is the most distal in the arch,

WWW.HIGHDENT.IR

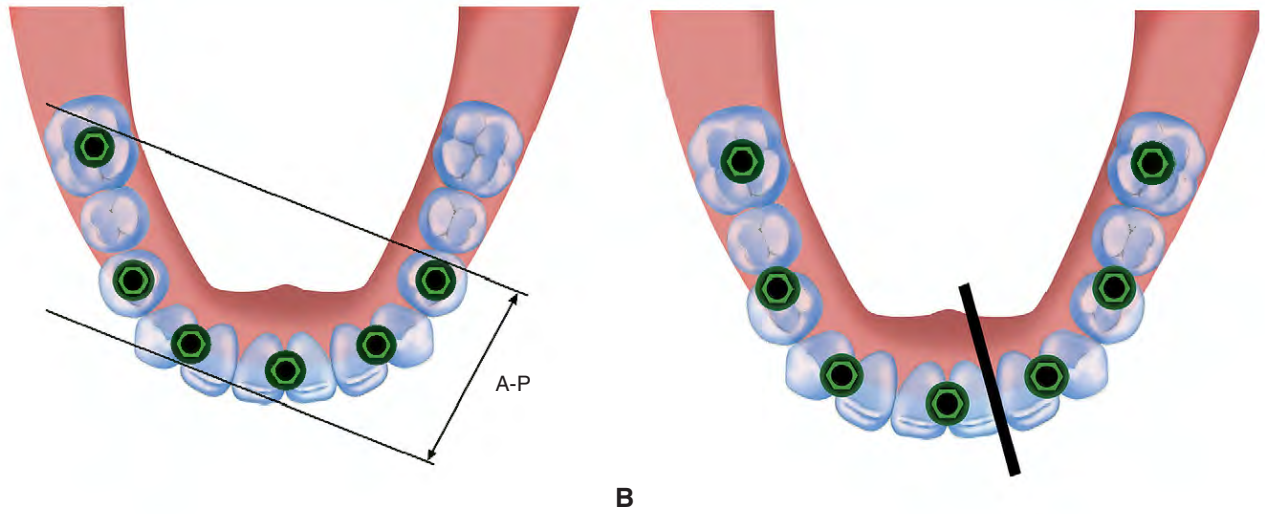


Figure 8-19 **A,** When six or more implants are positioned in the mandible (first molar, two first premolar, two canine, and one central incisor) a cantilever may be designed in the mandible because of the dynamics of an arch, with four of the five sections of an open pentagon joined, the large anteroposterior distance, and the favorable mandibular bone density. **B,** When seven or more implants are placed in an edentulous mandible, two separate prostheses with no posterior cantilever may be designed. The mandible flexure and torsion is free to occur when the separation between the two prostheses is between the mental foraminae. *A-P,* Anteroposterior distance.

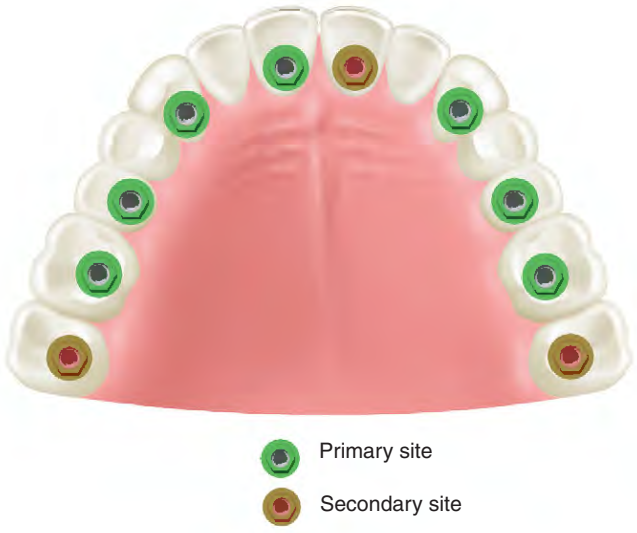


Figure 8-20 The ideal seven-implant positioning for a maxillary edentulous arch includes at least one central incisor position, bilateral canine positions, bilateral second premolar sites, and bilateral sites in the distal half of the first molars. In the case of heavy stress factors, an additional anterior implant and bilateral second molar positions (to increase the anteroposterior distance) may be of benefit.

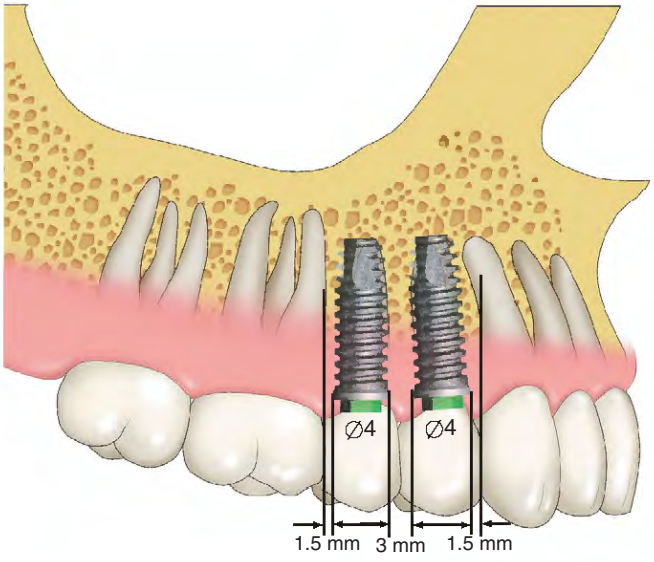


Figure 8-21 The minimum mesiodistal dimension for two standard 4-mm diameter implants is 1.5 mm + 4 mm + 3 mm + 4 mm + 1.5 mm = 14 mm.

a 12.5-mm span is required for two 4-mm-diameter implants (3 mm between each implant and 1.5 mm from the anterior tooth), because the 1.5-mm distance from the last tooth is no longer required.

There are several advantages of a 7- to 8-mm-wide premolar and a molar-sized crown. More implants may be used to restore the missing teeth. Implants may range from 4 to 5 mm in diameter, which are the most common sizes, and often the available bone has adequate buccolingual bone dimension in this region. The emergence of the crown contours on implants of this dimension permit sulcular probing. In addition, the occlusal table width decreases mesial and distal moment forces compared with a molar-sized crown.[22]

SPLINTED IMPLANTS

The primary advantage of restornig implants as independent units would be interproximal hygiene. However, this concept is not relevant with implants for two reasons. First, a very low percentage of the population flosses regularly.[23] Therefore this advantage would, at best, affect 1 or 2 of 10 patients. Because the implants are usually 3 mm or more apart, if a patient does wish

WWW.HIGHDENT.IR

to perform interproximal hygiene, most aids (e.g., floss threader, proxy brush) could easily clean this region.

A second advantage of separate dental units is the ability to replace a single unit to repair porcelain fracture. However, when dental implants are splinted together, the crown marginal ridges between the implants are supported by metal connectors; therefore the porcelain is placed under compression. As independent units, the margins of porcelain-to-metal crowns are most often placed under shear loads, which increase the risk of porcelain fracture. In addition, splinted dental units provide greater prosthesis retention and transfer less force to the cement interface. As a result, the restoration is less likely to become uncemented. This is especially significant when the abutments are short or lateral forces are present.

A third reason for independent crowns is primarily related to natural teeth. A single crown has a caries risk of less than 1% within 10 years. However, when natural teeth are splinted together, decay at the interproximal margin often occurs at a rate of approximately 22%.[24] In addition, the endodontic risk is increased when crowns are splinted. A single crown has an endodontic risk of 3% to 5.6%. Splinted teeth have an endodontic risk of 18%.[3] Therefore independent units reduce the incidence of complications and allow the practitioner to more readily treat these complications. However, implants do not decay or need endodontic therapy. As a result, independent units would not be required to address these complications.

On the other hand, there are many advantages to splinting implants together. To maximally benefit from an increased number of implants, the implants should be splinted together. Splinted implants increase functional surface area of support, increase the A-P distance (A-P spread) to resist lateral loads, increase cement retention of the prosthesis, decrease the risk of abutment screw loosening, decrease the risk of marginal bone loss, and decrease the risk of implant component fracture. In other words, the entire system benefits.[25-28]

In addition to biomechanical reasons, if an independent implant fails over time, the implant is removed, the site bone is grafted, the site is reimplanted, and a new crown is fabricated. When multiple splinted implants have an implant that fails, the affected implant may often be cut below the crown, and the implant or crown site converted to a pontic using the same prosthesis. As a result, rather than several surgical and prosthetic procedures over an extended period when independent units are restored, the problem may be solved in one relatively short appointment when the crowns are splinted together.

The splinted implants distribute less force to the implant bodies, which decreases the risk of marginal bone loss or implant body fracture. In a report by Sullivan, a 4-mm single implant replacing a molar had implant body fracture in 14% of the cases.[29] In comparison,

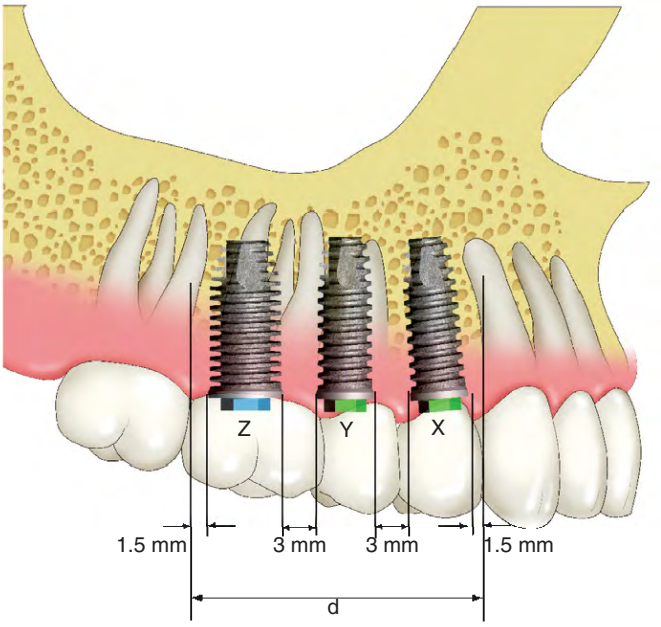


Figure 8-22 When three adjacent teeth are missing (two premolars and first molar), the mesiodistal dimension averages 7.1 mm + 6.6 mm + 10.4 mm = 24.1 mm. In this situation, planning 4-mm-diameter implants to fabricate two premolars of 7 mm each (1.5 mm + 4 mm + 1.5 mm) and one 5-mm-diameter implant for the first molar allows more bone around each implant.

multiple implants splinted together had a 1% implant body fracture.[1] Splinted implants reduce the risk of screw loosening. The highest prosthetic complication with single-tooth implants is abutment screw loosening. In a report by Balshi, single-tooth implants replacing a molar had 48% screw loosening over a 3-year period.[30] When two implants were splinted together to replace a molar, the incidence of screw loosening was reduced to 8% over the same period.

The exception to the splinted implant rule is a full-arch mandibular implant prosthesis. The body of the mandible flexes distal to the foramen on opening and has torsion during heavy biting with potential clinical significance for full-arch implant prostheses.[31,32] As a result, a full-arch implant prosthesis replacing the first or second molars should not be splinted to molars on the opposite side. Therefore full-arch mandibular restorations should have a cantilever or be made in two or three sections to accommodate the mandibular dynamics during function. The concept of flexure and torsion does not affect the maxilla, where all implants often are splinted together, regardless of their positions in the arch.

SUMMARY

A biomechanical-based treatment plan reduces complications after implant loading with the prosthesis. To

WWW.HIGHDENT.IR

reduce stress conditions, there are key implant positions for a prosthesis replacing missing teeth: (1) no cantilevers should be ideally designed on the restoration, (2) three adjacent pontics should be eliminated, (3) the canine and first molar sites are important positions in an arch, and (4) an arch is divided into five segments. When more than one segment of an arch is being replaced, a key implant position is at least one implant in each missing segment.

Increasing the number of implants is the most efficient method to increase surface area and reduce overall stress. Therefore, after the key implant positions are selected, additional implants are indicated to reduce the risks of overload from patient force factors or implant sites with reduced bone density. When in doubt of the number of implants required, add an additional implant.

References

1. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implant and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
2. Misch CE: Consideration of biomechanical stress in treatment with dental implants, *Dent Today* 25:80-85, 2006.
3. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
4. Priest GE, Priest J: The economics of implants for single missing teeth, *Dent Econ* 130-138, May 2004.
5. Rosenstiel SF, Land MF, Fujimoto J: *Contemporary fixed prosthodontics*, ed 4, St Louis, 2006, Mosby.
6. Shillinburg HT, Hobo S, Lowell D et al: Treatment planning for the replacement of missing teeth. In Shillinburg HI, Hobo S, editors: *Fundamentals of fixed prosthodontics,* ed 3, Chicago, 1997, Quintessence.
7. Smyd ES: Mechanics of dental structures: guide to teaching dental engineering at undergraduate level, *J Prosthet Dent* 2:668-692, 1952.
8. Phillips RW: Personal communication, 1990.
9. Bidez MW, Lemons JE, Isenberg BR: Displacements of precious and nonprecious dental bridges utilizing endosseous implants as distal abutments, *J Biomed Mater Res* 20:785-797, 1986.
10. Tylman SD: *Theory and practice of crown and fixed partial prosthodontics,* St Louis, 1965, Mosby.
11. Adell R, Lekholm U, Rockler B et al: A 15 year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 6:387-416, 1981.
12. Bidez MW, Misch CE: The biomechanics of interimplant spacing. Proceedings of the 4th International Congress of Implants and Biomaterials in Stomatology, Charleston, SC, May 24-25, 1990.
13. Rangert B, Renouard F: *Risk factors in implant dentistry,* Chicago, 1999, Quintessence.
14. McAlarney ME, Stavropoulos DN: Theoretical cantilever lengths versus clinical variables in fifty-five clinical cases, *J Prosthet Dent* 83;332-342, 2000.
15. Tarnow DP, Cho SC, Wallace SS: The effect of interimplant distance on the height of interimplant bone crest, *J Periodontol* 71:546-569, 2000.
16. Hatley CL, Cameron SM, Cuenin MF et al: The effect of dental implant spacing peri-implant bone using the rabbit *(Oryctolagus cuniculus)* tibia model, *J Prosthodont* 10:154-159, 2001.
17. Gastaldo JF, Cury PR, Sendyk WR: Effect of the vertical and horizontal distances between adjacent implants and between a tooth and an implant on the incidence of interproximal papilla, *J Periodontol* 75:1242-1246, 2004.
18. Novaes AB Jr, Papalexiou V, Muglia V et al: Influence of interimplant distance on gingival papilla formation and bone resorption: clinical-radiographic study in dogs, *Int J Oral Maxillofac Implants* 21:45-51, 2006.
19. Papalexiou V, Novaes AB Jr, Ribeiro RF et al: Influence of the interimplant distance on crestal bone resorption and bone density: a histomorphometric study in dogs, *J Periodontol* 77:614-621, 2006.
20. Desjardins RP: Tissue-integrated prostheses for edentulous patients with normal and abnormal jaw relationships, *J Prosthet Dent* 59:180-187, 1988.
21. Hobo S, Ichida E, Garcia LT: *Osseointegration and occlusal rehabilitation,* Chicago, 1989, Quintessence.
22. Allahyar G, Morgano SM: Finite element analysis of three designs of an implant-supported molar crown, *J Prosthet Dent* 5:434, 2004.
23. Segelnick S: A survey of floss frequency, habit and technique in a hospital dental clinic and private periodontal practice, *N Y State Dent J* 70:28-33, 2004.
24. Scurria MS, Bader JD, Shugars DA: Meta-analysis of fixed partial denture survival: prostheses and abutments, *J Prosthet Dent* 79:459-464, 1998.
25. Skalak R: Biomechanical considerations in osseointegrated prostheses, *J Prosthet Dent* 49:843, 1983.
26. Rangert B, Jemt T, Jorneus L: Forces and moments on Brånemark implants, *Int J Oral Maxillofac Implants* 4:241, 1998.
27. Wang TM, Leu IJ, Wang J et al: Effects of prosthesis materials and prosthesis splinting on peri-implant bone stress around implants in poor quality bone: a numeric analysis, *Int J Oral Maxillofac Implants* 17:231, 2002.
28. Guichet DL, Yoshinobu D, Caputo AA: Effects of splinting and interproximal contact tightness on load transfer by implants restorations, *J Prosthet Dent* 87:528, 2002.
29. Sullivan D, Siddiqui A: Wide-diameter implants: overcoming problems, *Dent Today* 13:50-57, 1994.
30. Balshi TJ, Wolfinger GJ: Two-implant-supported single molar replacement: interdental space requirements and comparison to alternative options, *Int J Perio Rest Dent* 17:426-435, 1997.
31. Abdel-Latif H, Hobkirk J, Kelleway J: Functional mandibular deformation in edentulous subjects treated with dental implants, *Int J Prosthodont* 13:513-519, 2000.
32. Hobkirk JA, Havthoulas TK: The influence of mandibular deformation, implant numbers, and loading position on detected forces in abutments supporting fixed implant superstructures, *J Prosthet Dent* 80:169-174, 1998.

WWW.HIGHDENT.IR

Chapter 9

Implant Body Size: A Biomechanical and Esthetic Rationale

Carl E. Misch

The initial treatment plan for implant dentistry should include the ideal implant size, based primarily on biomechanic and esthetic considerations. In traditional prosthetics, when a tooth is replaced, the abutment teeth are already provided by nature with wide posterior abutments for posterior teeth. When teeth are replaced with dental implants, the implant team should preselect the ideal abutment size, based on the ideal size for an esthetic restoration within biomechanical guidelines.

The size of an implant used to be determined primarily by the existing bone volume in height, width, and length. The surgeon would select longer implants in the anterior regions of the mouth and shorter ones in the posterior areas because of the limits of the mandibular canal and maxillary sinus. The width of the implant, also determined at surgery, would relate to the existing width of available bone, and one diameter implant (4 mm) would be used in most all situations.[1]

Over the years, dental implant treatment plans incorporating biomechanics have been advocated by Misch to decrease the most common complications—those related to stress.[2] The prosthesis first is planned, including whether the restoration is fixed or removable, how many teeth are replaced, and the esthetic demands. The patient force factors are then considered to evaluate the magnitude and type of force applied to the restoration. The bone density is evaluated in the regions of the potential implant placement. The key implant positions and the implant number are then selected in relation to the patient force factors and the bone density in the implant sites. For example, when the patient has parafunction or the bone is less dense or when a cantilever is present, the greater force exerted on the implant abutments will transmit greater stresses to the implant-bone interface. The next consideration in this ideal treatment plan sequence is the implant size.[1]

Dental implants function to transfer loads to surrounding biological tissues.[3,4] Biomechanical load management is dependent on two factors: the character of the applied force and the functional surface area over which the load is dissipated.[5] The implant size directly affects the functional surface area that distributes a load transferred through the prosthesis. A comprehensive approach to the overall dental implant size begins with the identification of clinical problems to be addressed. Fundamental scientific principles related to force and surface area are then combined with engineering principles to pursue the desired clinical goals. When relevant, esthetic considerations in regards to implant size are part of the evaluation. This chapter will build on and apply basic biomechanics and demonstrate how these principles also relate to the ideal dental implant size to support a prosthetic load. The esthetic guidelines related to implant size will also be addressed.

CHARACTER OF FORCES APPLIED TO DENTAL IMPLANTS

Stress and Strain

The presence of fibrous tissue has long been known to decrease the long-term survival of a root form implant.[1] Excessive loads on an osteointegrated implant may result in mobility of the supporting device, even after a favorable bone-implant interface has been obtained.[6] Although several conditions may cause crestal bone loss, one of these may be prosthetic overload.[7] Excessive loads on the bone result in increased strain conditions in the bone.[8] These microstrains on the bone may affect the bone remodeling rate and cause pathologic overload, which results in the loss of bone. The amount of bone strain is directly related to the amount of stress applied to the implant-bone interface. The greater the stresses throughout the implant-bone interface, the greater the risk factor for crestal bone loss and subsequent implant failure.[9] Therefore the stress and strain relationship has been shown to be an important parameter for crestal bone maintenance and implant survival.

Forces applied to dental implants may be characterized in terms of five distinct (although related) factors: magnitude, duration, type, direction, and magnification.[5] Each factor should be considered within the physiologic constraints on implant size. In addition, there are surgical and prosthetic considerations related to implant size.

WWW.HIGHDENT.IR

For example, the increase in stress to an implant body also increases the risk of abutment screw loosening or implant body fracture. As a result, a relative risk factor for different implant sizes may be established.

Force Magnitude

The physiology of the stomatognathic system imposes a range on the magnitude of forces that may be applied to an implant in the oral environment. The magnitude of bite force varies as a function of anatomical region and state of the dentition.[10] Average bite forces can range from 10 to 350 lb. The magnitude of force is greater in the molar region (200 lb), less in the canine area (100 lb), and least in the anterior incisor region (25 to 35 lb).[11] These average bite forces increase with parafunction to magnitudes that may approach 1000 lb in the posterior regions.[12]

After sustained periods of edentulism, the bone foundation often becomes less dense. Studies on dentate and edentulous jaws illustrate greater trabecular bone density in the anterior regions compared with the premolar or molar regions.[13] The bone's ultimate strength is highly dependent on its density.[14] As such, less dense bone may no longer be able to support normal physiologic bite forces on a dental implant. Careful treatment planning, including appropriate implant size selection, is imperative to lower the magnitude of loads imposed on the vulnerable implant-bone interface under these less ideal conditions. Thus the posterior regions with higher bite forces and lower bone densities should use a different parameter for implant size compared with the anterior regions. In addition, the anterior regions are more often in the esthetic zone and may influence the size of the implant as a consequence.

Force Duration

The duration of bite forces on the dentition has a wide range. Under ideal conditions, the teeth come together during swallowing and eating for only brief contacts. The total time of those brief episodes is less than 30 minutes per day. Patients who exhibit bruxism, clenching, or other parafunctional habits, however, may have their teeth in contact several hours each day.[12] Fatigue fractures increase in direct relationship to the amount of the force and the number of cycles of load. Therefore an increase in force duration directly increases the risk of fatigue load to the implant body when the force is higher than the endurance limit of these entities.[15] The implant body width is directly related to the strength of an implant, and wider diameter implants reduce the risk of fatigue fracture.

The duration of a force may also alter the implant-bone interface. Fatigue damage to cortical bone has been reported under relatively high-frequency loading rates (e.g., shin splints in runners).[16] Although fatigue damage to alveolar bone has not yet been reported in the literature, it is unlikely the alveolar bone reacts differently to parafunctional loads. Roberts et al. report the bone around an implant may be remodeled at a rate of 500% each year after loading, compared with normal trabecular physiologic remodeling around a tooth of 20% to 40% per year.[17] The dramatic increase in remodeling rates may eventually lead to fatigue damage and resultant bone loss.[18]

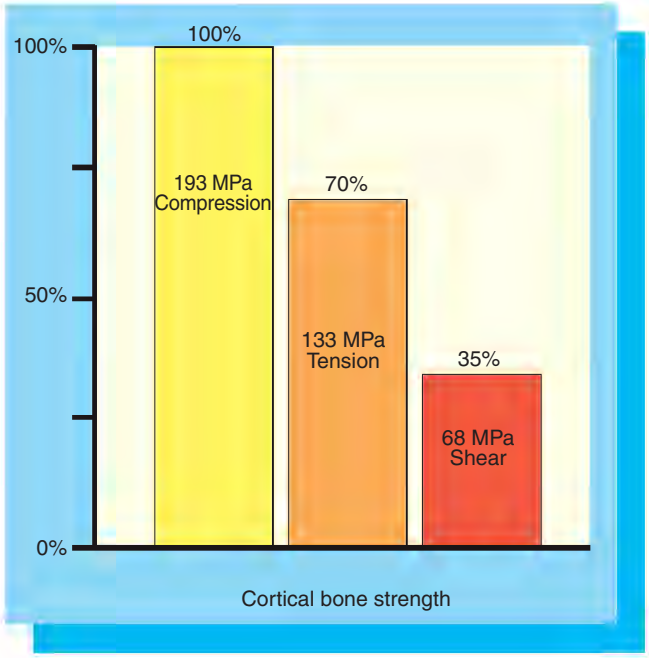


Figure 9-1 Bone is strongest under compression forces, 30% weaker to tensile forces, and 65% weaker to shear forces.

Force Type

Three types of forces may be imposed on dental implants within the oral environment: compression, tension, and shear. Bone is strongest when loaded in compression, 30% weaker when subjected to tensile forces, and 65% weaker when loaded in shear (Figure 9-1).[19] Therefore an attempt should be made to limit shear forces on bone, because it is least resistant to fracture under these loading conditions. This is most important in regions of decreased bone density, because the strength of bone is also directly related to its density.[14] An increased width of an implant may decrease offset loads and increase the amount of the implant-bone interface placed under compressive loads.

Force Direction

The forces to an implant body are typically greatest at the crestal bone interface.[20] Angled loads to the implant prosthesis produce angled loads to the crest module of the implant. The implant angulation is important to consider. For example, given an occlusal load with a 25-lb magnitude, the direction of the load has a significant effect on the magnitude of compressive and lateral load components. By increasing the angle of the

WWW.HIGHDENT.IR

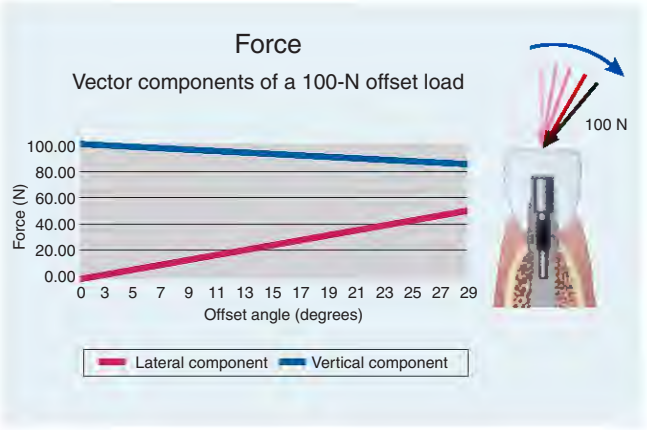


Figure 9-2 The force applied to an implant body with an angled load or angled direction of force is increased in direct relation to the force angle.

load by only 6 degrees, the lateral component of that load is increased by 233%. In addition, every degree of angled load greatly increases the damaging shear load component experienced by the implants, which is the most damaging component of the load (Figure 9-2).

Angled loads increase the amount of shear loads to the bone, and the bone is weakest to shear-type loads[21] (Table 9-1). Therefore, under ideal conditions, the implant body should be oriented to provide long axis compressive loads to the implant and to decrease shear loads to the crestal bone region. Occlusal loads in centric occlusion occur where the occlusal forces are usually the greatest. As such, the implant should be inserted perpendicular to the curve of Wilson and curve of Spee. Additionally, axial alignment places less stress on the overall implant system (i.e., abutment and abutment screw components) and decreases the risk of screw loosening and fatigue fractures of the implant or its components.

The anatomy of the mandible and maxilla places significant constraints on the ability to surgically place root form implants suitable for loading along their long axis. Bone undercuts further constrain implant placement and thus load direction imposed on the implant. Most all undercuts occur on the facial aspects of the bone, with the exception of the submandibular fossa in the posterior mandible.[22] Therefore implant bodies are often angled to avoid perforation of the lingual/facial bony undercut during insertion. Resorptive patterns after prolonged edentulism also exacerbates the normally occurring angulation challenges.[23]

The maxillary anterior region does not permit an ideal implant position, even under ideal conditions. The natural maxillary anterior teeth are 12 to 15 degrees off the long axis of load, and the bone of the premaxilla is in a similar relationship after tooth loss. Therefore implants in this region are often positioned with a greater relative angle to occlusal loads than any other region.[21] To decrease the effect of an angled load on the implant, the implant body may be increased in diameter.

Force Magnification

Force magnification increases the stress beyond the usual conditions of load; for example, a cantilevered prosthesis, a crown height greater than normal, or parafunction.[5] Multiple force magnifiers, such as a patient with parafunctional habits and an excessive crown height, may exceed the capability of any dental implant to withstand occlusal loads. Careful treatment planning with special attention to the implant position, implant number, occlusal loading, and an increase in implant size to increase functional surface area is indicated when a clinical case presents the challenge of force magnifiers.

A magnifier of force around an individual implant is also affected by the density of bone. Four distinct bone density categories within the maxilla and mandible exhibit a broad range of biomechanical strengths (i.e., ability to withstand physiologic loads).[13,14] Significantly

Table 9-1 Cortical Bone Strength Related to Angle of Load

TYPE OF FORCE	STRENGTH (MPa)	STANDARD DEVIATION	DIRECTION OF LOAD
Compressive	193	13.9	Longitudinal
	173	13.8	30 degrees off-axis
	133	15.0	60 degrees off-axis
	133	10.0	Transverse
Tensile	133	11.7	Longitudinal
	100	8.6	30 degrees off-axis
	60.5	4.8	60 degrees off-axis
	51.0	4.4	Transverse
Shear	68.0	3.7	Torsion

WWW.HIGHDENT.IR

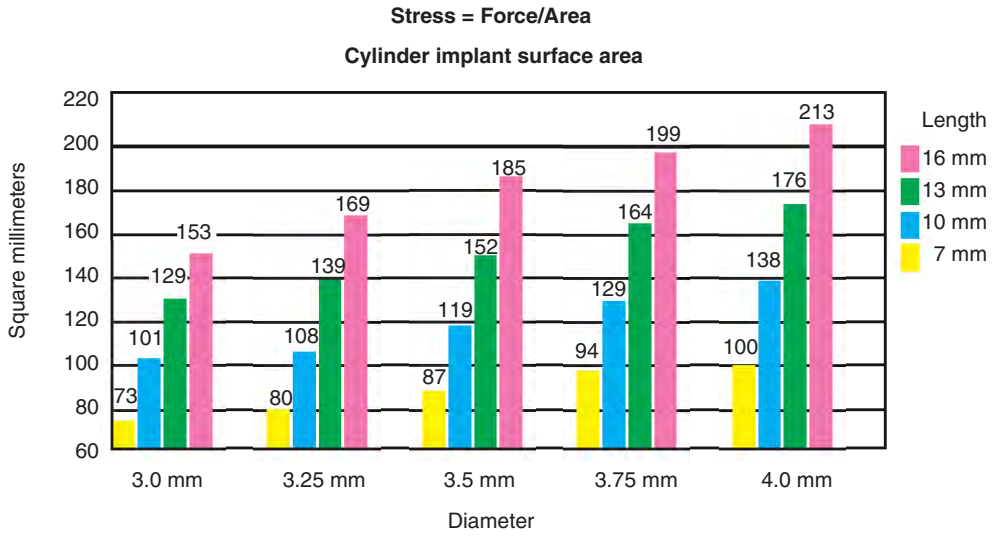


Figure 9-3 The length of a dental implant is directly related to its overall surface area. For every 3-mm increase in length, a cylinder implant has a 30% increase in surface area for shorter sizes and 20% for longer sizes.

increased clinical failure rates in poor-quality, porous bone compared with denser bone have been documented worldwide by multiple independent clinical investigators for more than a decade, with failure rates as high as 35% with implants in D4 (Type IV) bone.[24-28] Bone density is directly related to bone strength, and D4 bone may be more than 10 times weaker than D1 bone and 70% weaker than D2 bone.[14] Most implant failures in soft bone are from occlusal overload from a decrease in bone strength, a decreased bone-implant contact percentage, and the type of load transfer to the implant-bone interface on functional loading.[14,29] Therefore the effect of a resultant force is magnified as to its clinical result when placed in softer bone types.

Considerable effort should be made in the treatment plan to decrease the negative effects of compromised bone density, including implant size. The most important factor to decrease stress to the implant-bone interface is usually an increase in implant number, which dramatically increases the effective surface area over which the occlusal loads are dissipated, and in turn decreases stress. Implant number also relates to implant position, which can effectively reduce cantilever lengths and subsequent harmful bending loads and shear stresses. After the implant number has been increased in the treatment plan, the next beneficial step to decrease the risk of overload is to increase the implant size.

SURFACE AREA

The surface area over which the occlusal forces are applied is very relevant and is inversely proportional to the stress observed within the implant system (stress = force/surface area). It can be clearly seen from this basic engineering equation that, to reduce stress, the force must decrease or the surface area must increase. Therefore an increase in implant size is beneficial to decrease the stress applied to the system. The size of an implant may be modified in either length or diameter.

Rationale for Longer Implant Length

The length of the implant is directly related to the overall implant surface area, when all other variables are constant. A 10-mm–long cylinder implant increases surface area by approximately 30% over a 7-mm-long implant and has about 20% less surface area than 13-mm–long implants (Figure 9-3).[30,31] As a result, a common axiom has been to place an implant as long as possible. The length of the implant used for prosthetic support often corresponds to the height of available bone in the edentulous site.

Conventional thinking suggests that longer implants provide maximum functional surface area in a healed implant interface. However, when the length axiom is reevaluated, several challenges ensue. The available bone height is greater in the anterior regions of the mouth, especially the anterior mandible. Yet the bite forces are lower and the bone density is greater in these regions, especially in the mandible. The posterior regions have less bone height and the implant cannot engage the dense opposing cortical plate, because it is either nonexistent (maxilla) or beyond anatomical limits (neurovascular canal in the mandible). The posterior maxilla often has less height than the mandible, and the bone is less dense. As a result, poorer survival rates are reported for posterior implants, with the posterior maxilla associated with the highest failure rate (Figure 9-4).

WWW.HIGHDENT.IR

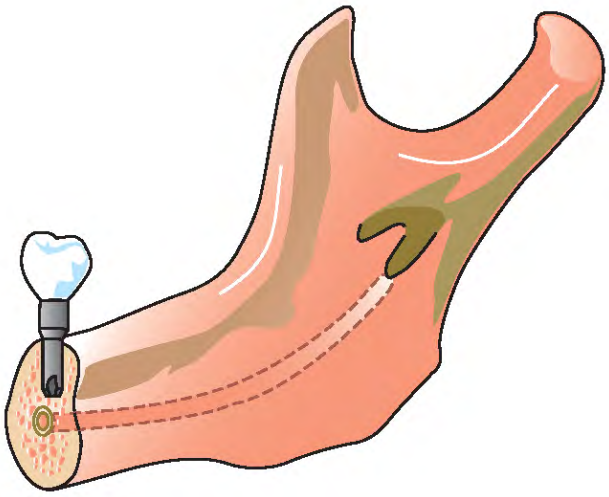


Figure 9-4 The longest implants are most often placed in the anterior mandible, followed by the anterior maxilla. The shortest implants are often placed in the posterior maxilla or mandible (or cantilevered from anterior implants). The survival rate of implants is related to length and anatomical location.

Implants longer than 15 mm have been suggested to provide greater stability under lateral loading conditions. However, reports suggest that increasing the length beyond a certain dimension may not reduce force transfer proportionately.[20,32] Finite element analysis provides an analytical means to investigate the influence of implant length relative to functional surface area under such extreme loading conditions. Misch and Bidez placed cylindrical implants of different lengths in a computer bone model with ideal bone density and volume (see Chapter 11). The embedded implant body lengths were 5, 10, 15, 20, and 30 mm. A 10-mm crown height was designed above the level of bone, and a lateral force of 50 N was applied to the top of the 10-mm crown. For these loading conditions, the percentage of maximum stress was plotted against the percentage of embedded length (Figure 9-5). Peak stresses were not completely dissipated in the 5-mm-long implant model, with approximately 30% of the maximum stress still present at the apex of the implant. The 5-mm implant model, therefore, did not provide sufficient length for lateral force dissipation in spite of good bone density and volume. In the 10-mm implant model, 80% of maximum stress was dissipated in approximately 95% of the embedded length. For the 15- and 20-mm lengths, 80% of the maximum stress was dissipated in approximately 90% of the embedded length. For the 30-mm length, 80% of the maximum stress was dissipated in approximately 70% of the embedded length. Therefore the length of the implant in favorable bone quality and crown height may range from 10 to 15 mm, and 12 mm is usually ideal. All implant lengths exhibited 80% to 100% of the stress in the crestal 40% of the implant length. The results of this analysis point to the fact that the majority of the maximum stress generated by a lateral load can be dissipated as well by implants in the range of 10 to 15 mm in length, compared with implants in the range

WWW.HIGHDENT.IR

of 20 to 30 mm in length. In addition, the highest stresses were observed in the crestal bone regions, regardless of the implant length. This biomechanical analysis supports the opinion that longer implants are not necessarily better in good bone volume and density, such as that found in the anterior regions of the mandible. An implant length of 12 mm is usually ideal under most patient force and bone density conditions and up to 15 mm is suggested in softer bone types.

Bicortical stabilization, a rationale often cited for longer implants, is not needed in D1 bone, which is primarily homogenous cortical bone.[13] Because bone overheating is a primary cause of surgical failure, attempting to engage the opposing cortical plate and preparing a longer osteotomy may result in overheating the bone when it is D1 or D2.[33] A threaded implant may not readily engage the denser bone of the apical cortical plate, and the implant threads may strip along the rest of the osteotomy, especially if D3 or D4 trabecular bone types. In addition, after the implant-bone interface is formed, excessively long implants do not transfer stress to the apical region, because most of the stresses are transmitted within the crestal 7 to 9 mm of bone (except in the softest bone types) and therefore are often not needed.[20] A further analysis relative to implant length is suggested, especially in good-quality bone.

D3 and D4 bone are primarily present in the posterior regions of the jaw, where less available bone height is observed compared with the anterior regions. Nerve repositioning is cited as an acceptable clinical treatment to facilitate placement of longer implants in the posterior mandible. However, this advanced surgical procedure represents an increased risk for paresthesia and is often not indicated, especially when other implant options are available.[34] To place the longest implants in the maxillary posterior regions, a sinus graft is often required. Sinus grafts have been shown to grow bone into the graft from the surrounding walls of bone, and bone does not form under the sinus mucosa. The apical end of longer implants will not benefit from the sinus bone graft procedure for some time, and the crest of the bone below the graft will remain at greater risk. Therefore increasing surface area primarily by length in the posterior regions of the jaws requires advanced grafting or nerve repositioning surgery and, in spite of this, does not benefit the primary regions of increased stress (i.e., the crestal bone region). However, implants from 12 to 15 mm in length are usually considered ideal for most situations.

Disadvantages of Short Implants

Different risk factors for implant longevity have emerged over the years. A review of the literature related to implant failure and implant length was published by Goodacre et al. in 2003.[35] In the majority of articles addressing implant length, implants 10 mm or smaller have increased failure rates. In the reports summarized, the failure rates of short implants were 10%, compared with a 3% failure rate of longer implants. The failure is even more apparent when the literature reviews implants smaller than 10 mm in the posterior regions of partially edentulous patients. Less than half the clinical reports had survival rates higher than 90%, and more than half of the reports had implant failure higher than 19.7% (Tables 9-2 and 9-3).[36-46]

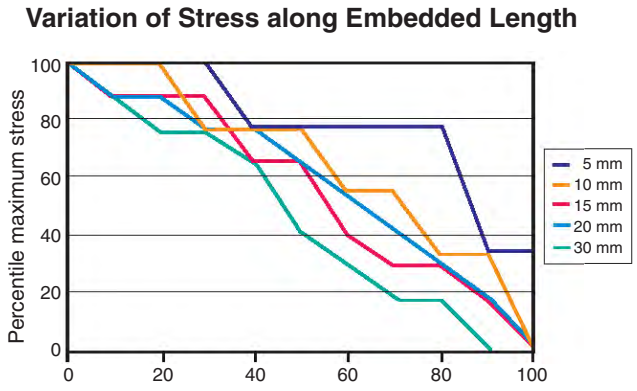


Figure 9-5 A computer bone model with implant lengths of 5, 10, 15, 20, and 30 mm was loaded with a lateral force of 50 N applied to the top of a 10-mm crown extension. Almost 80% of the stress was found in the crestal 40% of the implant, regardless of the length.

A review of several of the larger, multicenter reports of short implants is noteworthy. Minsk et al. reported the results of a training center in 1996, with 80 different operators using 6 different systems over a 6-year period.[27] Implants 7 to 9 mm in length were reported to have a 16% failure rate. The overall survival rate of all longer lengths was 95%. Winkler et al. published a multicenter report over a 3-year period in 2000.[37] The implant survival was directly related to the length of the implant. The 7-mm-long implants had a 26.4% failure rate, whereas 16-mm implants demonstrated only a 2.8% rate of failure. Implants of 8 mm had a 13% failure rate, whereas 10-mm implants in the report failed at a rate of 10.9%, and the 13-mm implants failed 5.7% of the time.

A multicenter report by Weng et al. in 2003 found 60% of all failed implants were 10 mm or less in length.[38] The overall failure rate of all implants in the study was 9%, yet the 7-mm implant failed 26% of the time and the 8.5-mm implant had a 19% failure rate[39] (Figure 9-6). Naert et al. reported in the literature on clinical outcomes of short dental implants.[6] Implants shorter than 10 mm had a survival rate average of 81.5%, whereas longer implants had a survival rate higher than 95%.

It should be noted the failure rates in most all these reports are not surgical failures or failures to osseointegrate. The failures associated with short implants often occurred after prosthetic loading. In other words, the surgical success was not affected by implant length. However, after the prostheses were

WWW.HIGHDENT.IR

Table 9-2 Short Implants (Literature Review Above 90%)

AUTHOR	NO. OF IMPLANTS	SUCCESS RATE (%)	IMPLANT SYSTEM
Higuchi et al., 1995[a]	109	94	Brånemark
Lekholm et at., 1999[b]	101	94	Brånemark
van Steenberghe et al., 2000[c]	16	100	Astra
Testori et al., 2001[d]	31	97	3i
Tawil et al., 2003[e]	116	93	Brånemark
Misch et al., 2005[f]	437	99	BioHorizons
Total implants	**810**		
Average success rate		**96.8**	

a. Higuchi KW, Folmer T, Kultje C: Implant survival rates in partially edentulous patients: a 3-year prospective multicenter study, *J Oral Maxillofac Surg* 53:264-268, 1995.
b. Lekholm U, Gunne J, Henry P et al: Survival of the Brånemark implant in partially edentulous jaws: a 10-year prospective multicenter study, *Int J Oral Maxillofac Implants* 14:639-645, 1999.
c. van Steenberghe D, De Mars G, Quirynen M et al: A prospective split-mouth comparative study of two screw-shaped self-tapping pure titanium implant systems, *Clin Oral Implants Res* 11:202-209, 2000.
d. Testori T, Wiseman L, Woolfe S et al: A prospective multicenter clinical study of the Osseotite implant: four-year interim report, *Int J Oral Maxillofac Implants* 16:193-200, 2001.
e. Tawil G, Aboujaoude, Younan R: Influence of prosthetic parameters on survival and complication rates of short implants, *Int J Oral Maxillofac Implants* 21:275-282, 2006.
f. Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study, *J Periodontol* 77:1340-7, 2006.

Table 9-3 Short Implants (Literature Review Below 90%)

AUTHOR	NO. OF IMPLANTS	SUCCESS RATE (%)	IMPLANT SYSTEM
Jemt and Lekholm, 1995[a]	298	76	Brånemark
Minsk et al., 1996[b]	50	84	Brånemark (6 systems)
Saadoun and Le Gall, 1996[c]	115	79	Steri-Oss
De Bruyn et al., 1999[d]	9	87	Screw-Vent
Winkler et al., 2000[e]	152	81	Screw-Vent (6 designs)
Naert et al., 2002[f]	1168	67	Brånemark
Weng et al., 2003[g]	97	79	3i
Total implants	**1889**		
Average success rate		**80.3**	

a. Jemt T, Lekholm U: Implant treatment in edentulous maxillae: a 5-year follow-up report on patients with different degrees of jaw resorption, *Int J Oral Maxillofac Implants* 10:303-311, 1995.
b. Minsk L, Polson A, Weisgold A et al: Outcome failures of endosseous implants from a clinical training center, *Compend Contin Educ Dent* 17:848-856, 1996.
c. Saadoun AP, Le Gall MG: An 8-year compilation of clinical results obtained with Steri-Oss endosseous implants, *Compend Contin Educ Dent* 17:669-668, 1996.
d. De Bruyn H, Collaert B, Linden U et al: Clinical outcome of screw vent implants: a 7-year prospective follow-up study, *Clin Oral Implants Res* 10:139-148, 1999.
e. Winkler S, Morris HF, Ochi S: Implant survival to 36 months as related to length and diameter, *Ann Periodontol* 5:22-31, 2000.
f. Naert I, Koutsikakis G, Duyck J et al: Biologic outcome of implant-supported restorations in the treatment of partial edentulism. Part I: a longitudinal clinical evaluation. *Clin Impl Res* 13:381-389, 2002.
g. Weng D, Jacobson Z, Tarnow D et al: A prospective multicenter clinical trial of 3i machined-surface implants: results after 6 years of follow-up, *Int J Oral Maxillofac Implants* 18:417-423, 2003.

in function, an increase in early loading failure was observed, especially within the first 12 to 18 months.

When an implant of 12 mm or more is unavailable without advanced surgical procedures to gain height, a shorter implant may be considered. However, four reasons linked to biomechanics may explain why the posterior short implant may have a higher failure rate after loading compared with longer implants[48]:

1. Higher bite forces
2. Bone density in the region
3. Increased crown height
4. Implant design considerations

IDEAL IMPLANT SIZE

To obtain predictable success in situations with most patient force factors or bone densities, there is a minimum implant length, depending on the implant body width and the implant design[49] (Figure 9-7). The softer the bone, the greater the implant body length and diameter suggested[50]. The literature observes a wide range of implant studies reporting higher implant survival rates when the implant is at least 12 mm in length. Shorter implants usually increase the risk of failure and therefore are not initially treatment planned as a first option. Rather than establish an ideal treatment

WWW.HIGHDENT.IR

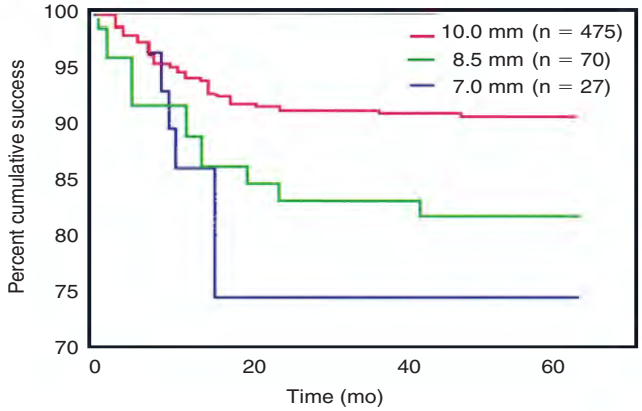


Figure 9-6 The 7-mm implant failed 26% of the time and the 8.5-mm-long implant had a 19% failure rate. Nearly all the failures occurred within 18 months after the initial loading period. (*Data from Weng D, Jacobsen Z, Tarnow D et al: A Prospectiv multicenter clinical trial of 3: machined-surface implants: results after 6 years of follow-up*, Int J Oral Maxillofac Implants *18:417-423, 2003*.)

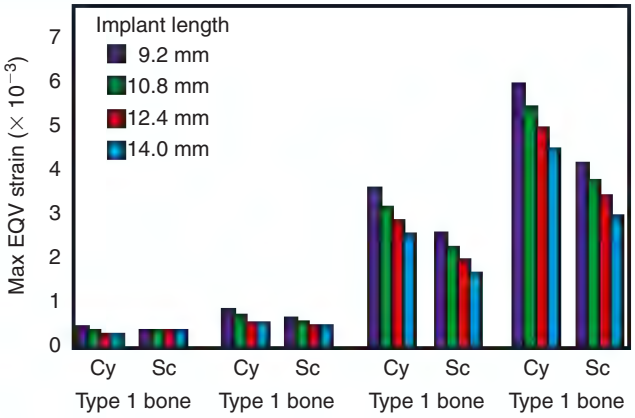


Figure 9-7 A finite element analysis found the strain increase around an implant was greatest in softer bone types (Type 3 and Type 4 bone). Longer implants had less strain than shorter implants and screw type implants less strain than cylinder implant in softer bone types. *(Data from Tada S, Stegariou R, Kitamura E et al: Influence of implant design and bone quality on stress/strain distribution in bone around implants: a 3-dimensional finite element analysis,* Int J Oral Maxillofa Implants *18:357-368, 2003.)*

plan that may be deficient in surface area, the minimum length of an implant for an unloaded healing protocol is usually 12 mm or more for most implant designs. Therefore, after the ideal treatment plan determines the key implant positions and implant number, the implant length selected for most treatment plan options is at least 12 mm long.

Rationale for a Shorter Implant Length

The height of existing available bone is often used by the implant dentist to determine the implant length after adequate width and mesiodistal space are confirmed. The height of available bone is measured from the crest of the edentulous ridge to the opposing landmark. The posterior regions of the jaws usually have the least height of existing bone, because the maxillary sinus expands after tooth loss and the mandibular canal is 10 mm or more higher than the inferior border of the mandibular body.[22] A radiographic study of 431 partially edentulous patients revealed that the posterior existing available bone height was 6 mm or greater in only 38% of maxillae and 50% of the mandibles.[34] The bite forces in the posterior regions of the mouth are greater than the anterior section.[11] As a result, in the posterior regions of the mouth with the highest bite forces, without sinus grafts or advanced surgical procedure, the implants are often shorter when compared with the anterior edentulous sites.

Implant length does affect the overall surface area of the implant body and is therefore theoretically desirable. However, unlike what occurs for a natural tooth and its periodontal membrane, stresses around implants during function and parafunction are typically concentrated on the marginal bone at the crest of the ridge.[9] Three-dimensional analysis demonstrate that for an implant with a direct bone contact, the greatest magnitude of stress is concentrated in the crestal 5 mm of the bone-implant interface[20,32] (Figure 9-8). Stresses

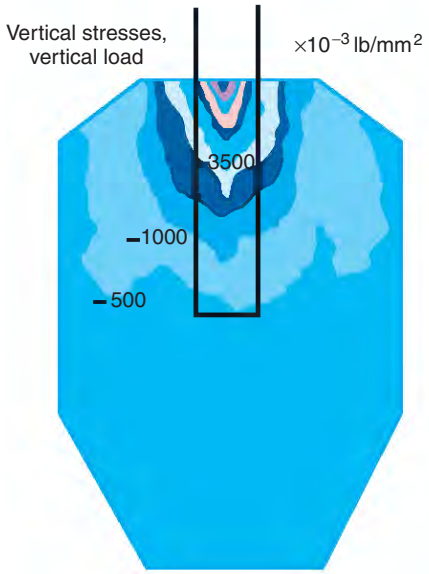


Figure 9-8 Three-dimensional finite element analysis demonstrates the highest strain conditions are around the crestal region of an implant.

distributed to the apical third of an implant are of much less magnitude than those in the crestal third. As a result, under some clinical conditions, stress transfer patterns to the bone may be similar between a short and a longer implant.[51]

Advantages of Short Implants

Shorter implants may offer several advantages compared with longer implants.[52] Bone grafting before and in conjunction with implant placement is not as frequent when short implants are used. The surgical procedure is reduced in complexity with decreased risk of overheating the bone and subsequent ease of site preparation and implant insertion. Therefore there

WWW.HIGHDENT.IR

Box 9-1 Advantages of Short Implants

1. Less bone grafting in height
   a. Less time for treatment
   b. Less cost of treatment
   c. Less discomfort
2. Less surgical risk of:
   a. Sinus perforation
   b. Paresthesia
   c. Osteotomy trauma from heat
   d. Damage to adjacent tooth root
3. Surgical ease
   a. Decreased interarch spaces
   b. Less inventory/cost

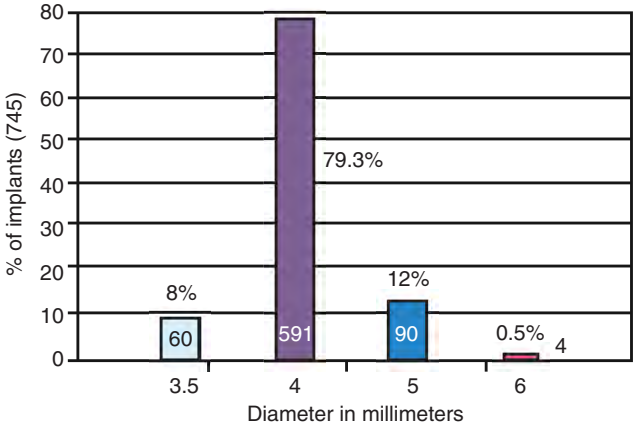


Figure 9-9 The implant diameters in this report were 3.5, 4, 5, and 6 mm. Of 745 implants, 79.3% were 4 mm wide, and the fewest in the report were 6 mm wide (0.5%). *(Data from Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study,* J Periodontol *77: 1340-1347, 2006.)*

is less surgical risk of sinus perforation during the grafting procedure in the posterior maxilla and less risk of mandibular canal violation and paresthesia in the lower posterior jaw. When an adjacent tooth root presents apical dilacerations, the shorter implant may be inserted above the apex of the tooth apex without compromising the implant position. In ridges with undercuts or fossae or when basal bone and the original alveolar ridge are not in the long axis of the missing teeth, a shorter implant may be inserted at a better angulation for occlusal load. From a surgeon's standpoint, less inventory and overhead in the office are additional benefits (Box 9-1).

In 2006, Misch et al. reported on a retrospective multicenter 6-year case series study,[46] during which 273 consecutive patients received 745 implants, either 7- or 9-mm long. These implants supported 338 restorations, all in the posterior regions of the mouth. There were 102 single tooth implants and 236 fixed restorations supported by multiple implants. In this multicenter report, the short implant dimension was dealt with by reducing biomechanical factors. Incisal guidance eliminated lateral forces in excursions. Multiple implants were always splinted together. Additional implants or wider implants were used when possible.

Box 9-2 Implant Size: Short versus Long

Guidelines

- Increase diameter
- Splint together
- Decrease crown height
- Decrease cantilever
- Increase surface area design
- OD versus FPD
- Minimize lateral force
- Improve bone density

OD, Overdenture; *FPD,* fixed partial denture.

From Stage I to Stage II healing, there were six failures (99.2% success rate). During the fabrication of the restorations, there were two implant failures (99.7% success rate). The 737 successful implants and 338 restorations were followed from 1 to 6 years after prosthesis delivery. A total of 140 implants were evaluated for at least 5 years and 263 implants for more than 4 years. No implants lost after prosthesis delivery. Therefore the early loading success rate after prosthesis delivery was 100% for up to 5 years (Figures 9-9 to 9-11). Therefore, when a biomechanical approach to decrease stress or increase area is followed, an implant survival similar to other reports may be obtained, at least in this report (Box 9-2).[46] Other recent reports in the literature also support the use of short implants, provided proper force orientation and load distribution are favorable.[43-45,52,53]

IMPLANT DIAMETER

Over several decades, implants have gradually increased in width. The pin implants of Scialom in the 1960s and 1970s were less than 2 mm wide (Figure 9-12).[54] Brånemark first introduced an implant body diameter of 3.75 mm.[1] An implant body of 4 mm in diameter was also available and was primarily used as a rescue or "back up" implant when the initial implant inserted lacked stability during surgical placement. In the late 1990s, several implant manufacturers provided implants with diameters as great as 8 mm. These larger diameter implants were primarily used to improve the emergence profile of the crown. It was rationalized that if the implant was similar in diameter to the replacement tooth size, the crown would be more esthetic.

The wide-diameter implant presents surgical, loading, and prosthetic advantages. The surgical advantages relate to the use of a wide implant as a rescue implant, when the regular body size does not adequately fixate to the surrounding bone (Box 9-3). The regular implant may be removed and replaced with the wide-body

WWW.HIGHDENT.IR

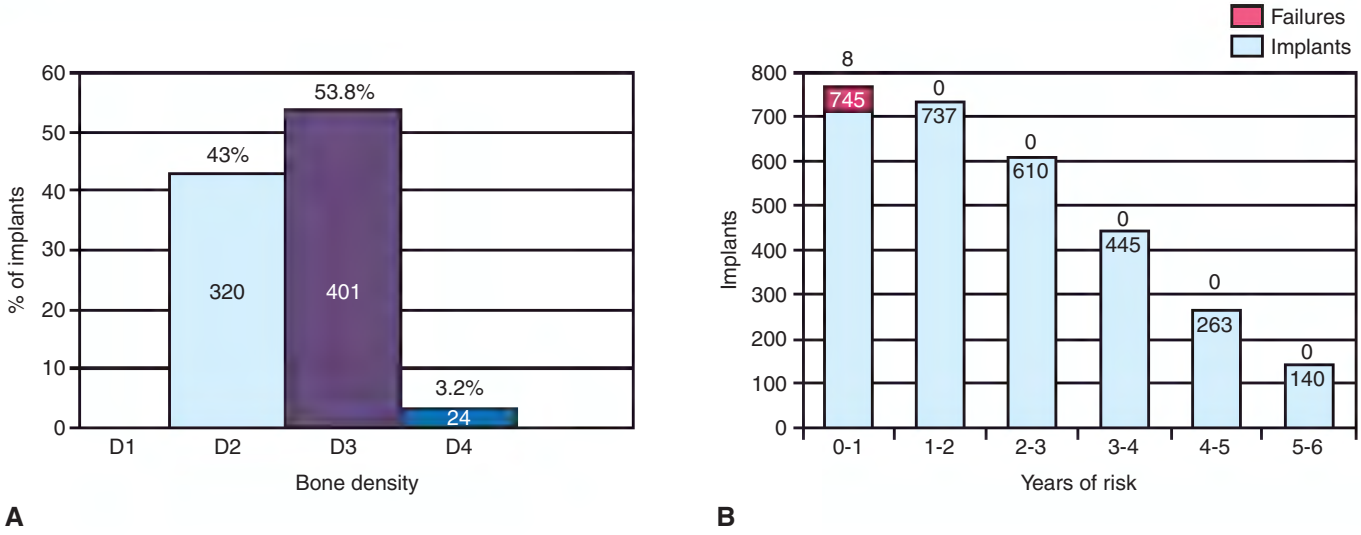


Figure 9-10 **A,** The majority of implant sites in the posterior regions were D3 bone (53.8%). The least-often observed bone types in this report were D1 and D4 bone, with 0% and 3.2% respectively. **B,** A total of 745 implants were surgically inserted during this study. There were eight implants lost (six implants from Stage I to Stage II surgery and two implants from Stage II to the prosthetic delivery). A total of 337 implants were followed after prosthetic delivery for as long as 6 years, with 263 implants having longer than a 4-year evaluation period.

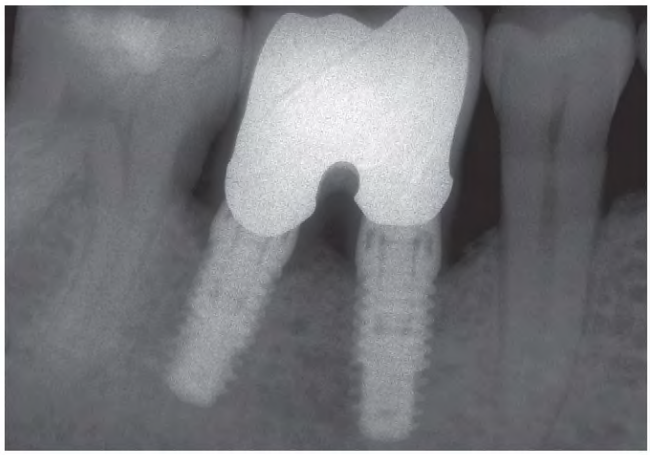


Figure 9-11 Short implants in the posterior regions were splinted together, with no lateral forces in excursions and no cantilevers less than 2 mm in diameter, to achieve clinical success.

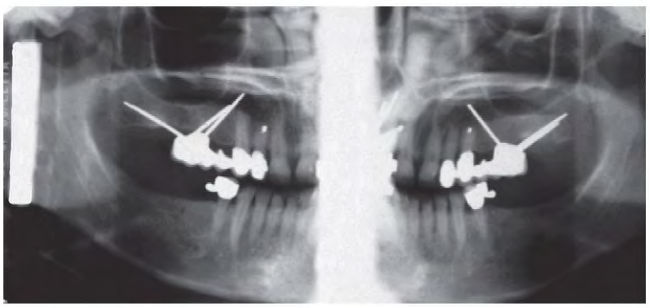


Figure 9-12 The pin implants of Scialom were used in a tripod approach to support fixed prostheses in the 1960s and 1970s.

Box 9-3 Surgical Advantages of Wide-Diameter Implants

- Surgical rescue implant
- Failed implant/immediate
- Tooth extraction/immediate

implant. When an implant fails, because of lack of osteointegration or fracture, the implant may be removed and the wide-body implant immediately inserted.[55] This eliminates the need for bone grafting, the time for bone augmentation healing, and the additional surgery to replace the implant. The same concept may be used for the immediate placement of an implant after the extraction of a tooth.[56] Because the diameter of most teeth are larger than 4 mm, a wider diameter implant has less space between the walls of the extraction site and the wide implant body.

The logical method to increase functional surface area in this region is to increase the implant diameter, because the opposing landmarks limit the implant length. Wider root form designs exhibit a greater area of bone contact than narrow implants of similar design, in part from an increase in circumferential bone contact. This trend is also noted in natural teeth to compensate for increased force; molar teeth are wider than incisors. For each millimeter implant diameter increase, the functional surface area is increased by 30% to 200%, depending on the implant design (i.e., cylinder versus thread) (Figure 9-13).

The loading advantages relate to a greater surface area, especially in the crestal region of the implant (Box 9-4). The greater surface area is a benefit when the patient force factors are greater; for example, parafunction, increased crown height, increased masticatory dynamics, and the molar regions in the posterior of the mouth.

WWW.HIGHDENT.IR

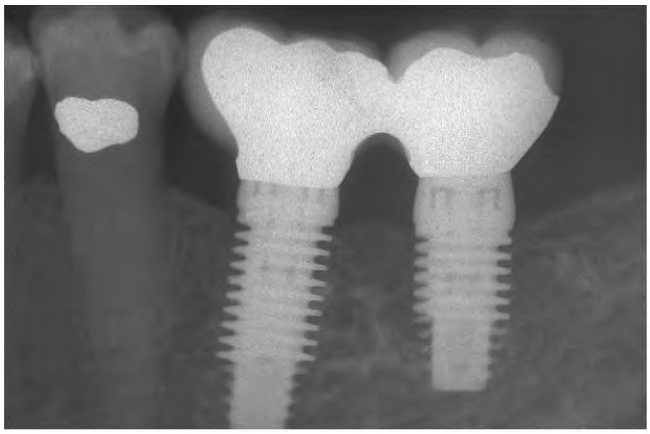


Figure 9-13 A wider diameter cylinder implant has a 5% to 8% greater surface area for every 0.25-mm increase in diameter. Therefore a 1-mm larger diameter cylinder implant may have a 30% increase in surface area.

Box 9-4 Loading Advantages of Wide-Diameter Implants

- Increase surface area
- Compensate unfavorable patient force factors
- Minimize cantilevers for angled implant
- Compensate for poor bone density
- Enhance surface for short implants

The greater surface area is an advantage when a cantilever is necessary to restore the dentition, either in a mesiodistal or facial-lingual direction. For example, the most distal implant with a posterior cantilever acts as a fulcrum and receives the greatest force. The wider diameter reduces the risk of overload. An angled load to the implant body increases the magnitude of the force at the crestal marginal bone and to the abutment screw of the implant. A larger diameter implant reduces the magnitude of forces to the entire implant system. In situations of poor bone density, there may be overload even with occlusal forces within physiologic limits. Wider diameter implants distributing forces over a greater surface area may decrease this risk.

An implant, smaller than 10 mm long, is often used in the posterior regions of the mouth. Under such conditions, the wide-diameter implant may compensate for the less than ideal implant length. Because occlusal stresses to the implant interface are concentrated at the crest of the ridge, width appears more important than height after a minimum height has been obtained for initial fixation and resistance to torque and bending loads. A comparative evaluation on strains in the alveolar crest of implants with different diameters was performed by Petrie and Williams.[57] In this three-dimensional finite element analysis, there was as much as 3.5-fold reduction in stress when wider diameter implants (up to 6 mm) were compared with narrow diameters (3.5 mm). In 2004, Himmlova et al. confirmed the findings on a three-dimensional computer model evaluating implant lengths and diameters from 2.9 to 6.5 mm.[58] The stresses were primarily at the marginal bone levels at the crest of the implant. The greatest decrease in stress was found between 3.6-mm and 4.2-mm implants (31.5%), whereas the 5-mm implant compared with the 4.2-mm diameter reduced the stress by half as much (16.4%). The length of the implant also reduced the amount of stress, but was not as pronounced as the diameter. A study by Aparicio and Orozco in 1998 used Periotest values to clinically confirm less stress transferred to the implant-bone complex.[59] The observed Periotest values from 5-mm-wide implants in the maxilla and mandible were 1.1 and 0.6 units lower than for 3.75-mm-diameter implants in the same patients.

Several studies in the literature report a similar or improved implant survival rates compared with the regular diameters. Graves et al., in 1994, reported 96% survival over a 2-year period with 268 wide implants in 196 patients.[60] All failures occurred before Stage II surgery from nonintegration of the implant. In 2000, Winkler et al. reported on the influence of implant diameter and length related to implant survival.[37] The survival of implants 3.0 to 3.9 mm in diameter was 90.7%, whereas the survival rate of implants from 4.0 to 4.9 mm was 94.6% over a 3-year period.

The 5.5-mm-wide Frialit-2 implant was evaluated by Krennmair and Waldenberger in 2004 with 121 implants in 114 patients.[61] The study yielded a 98.3% overall survival, with 100% in the mandible and 97.3% in the maxilla. Griffin and Cheung reported on short, wide implants in posterior areas with reduced bone height for 168 HA-coated implants 6 mm in diameter and 8 mm long in 167 patients.[52] There were 128 single crowns supported by these implants. The overall cumulative survival rate for up to 68 months (mean 34.9 months) after loading was 100%. Anner et al. in 2005, found a 100% survival rate in 45 implants with a mean loading period of 2 years with a 6-mm-wide, tapered, HA-coated implant.[62] In 2006, Misch et al. compared 4-mm and 5-mm implants that were 7- and 9-mm long, respectively, in the posterior maxilla and mandible.[46] The 5-year retrospective study yielded 100% implant success for the 5-mm implant and a 98% survival for the 4-mm implant. Therefore these later reports seem to indicate that larger diameter implants can achieve similar implant survival to the standard 3.75-mm-diameter implant body.

The prosthetic advantages of the wide-diameter implant include an improved emergence profile for the crown (Box 9-5). The larger the implant diameter, the more closely the emergence profile resembles the natural tooth, especially in the posterior region of the jaws. Most natural roots are greater than 4 mm in cross-section. The closer the implant diameter to the root diameter, 2 mm below the cement-enamel junction

WWW.HIGHDENT.IR

Figure 9-14 A molar implant crown has an improved emergence profile with a larger diameter implant. This may decrease food impaction in the interproximal region.

(CEJ) ideal crestal bone level (in ideal conditions), the more similar the crown emergence. This is especially noted in the maxillary first molar region, because the root diameter approaches 8 mm, or twice the size of a 4-mm implant (Figure 9-14). The wider crown contour may also decrease the interproximal space and decrease the incidence of food impaction during function. The wide-diameter implant may also improve sulcular daily oral hygiene by improving the crown emergence and avoiding the need for a ridge lap. This allows access to the sulcus for periodontal probing depths or sulcus cleaning.[52]

In 1997, Jarvis emphasized the biomechanical advantage of wide-diameter implants, particularly in reducing the magnitude of stress delivered to the various parts of the implant.[63] The diameter of the implant is related to the bending fracture resistance or moment of inertia, and the increase in diameter decreases the risk of fracture to the power of four, provided all other geometric features remain the same. Therefore, based on force factors, an implant placed in a young, bruxing male should be larger in diameter than an implant for an older, nonparafunctional female.

- Improve emergence profile
- Decrease screw loosening
- Minimize component fracture
- Facilitate oral hygiene

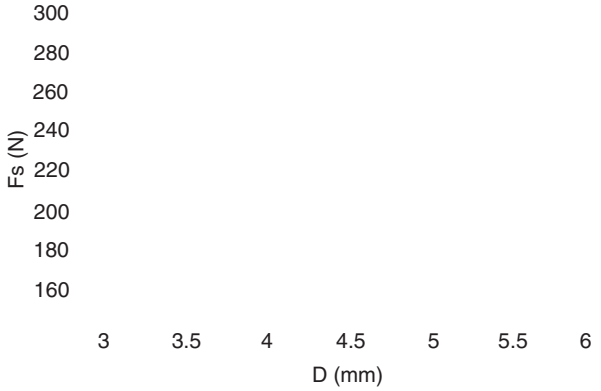


Figure 9-15 A larger diameter implant distributes less force to the abutment screw compared with a narrower diameter implant.

The force on an abutment screw is reduced with a large-diameter implant. A decrease in force to the abutment screw (and less screw loosening) and an increase in the strength of the implant body decrease the risk of fracture. The large-diameter implants, which have a larger prosthetic platform, exhibit less force transmission and stress to the abutment screw and therefore demonstrate less screw loosening (Figure 9-15).

By modeling the implant assembly as an engineering assembly, the following formula can be derived:

$$Fs = \frac{(P(H) - R2(h))^2}{D}$$

Where:

Fs = load on abutment screw
P = lateral load on the abutment
H = abutment height
R2 = reaction force on external hex
h = external hex height of the implant
D = platform diameter of the implant
Point A = point of abutment rotation (see Figure 9-16)

Therefore, by increasing *D* (platform diameter), the force on the screw is decreased. When a smaller abutment diameter is placed on an implant (platform switching), the force on the screw will increase, along with the risk of screw loosening (Figure 9-16).[64] In a clinical article by Cho et al. in 2004, wide-diameter

WWW.HIGHDENT.IR

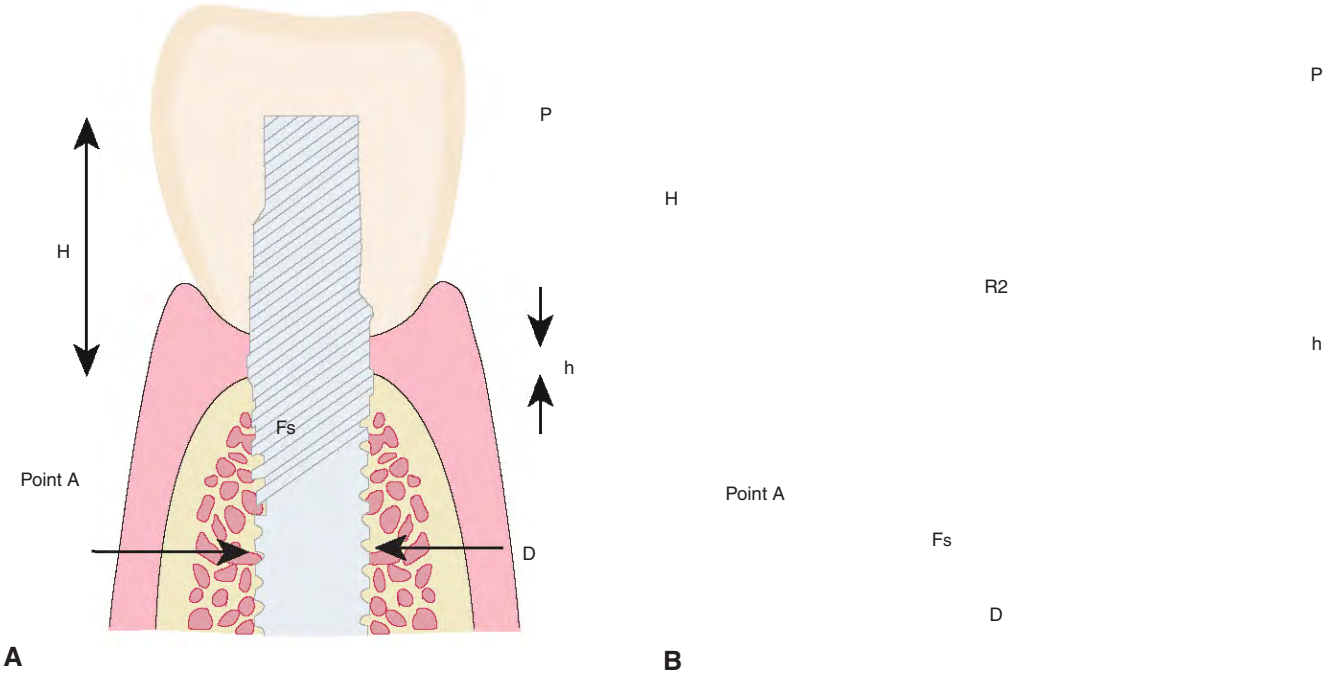


Figure 9-16 The implant assemby **(A)** modeled as an engineering assembly **(B).** *H,* Abutment height; *P,* lateral load on abutment; *Point A,* point of abutment rotation; *Fs,* load an abutment screw; *h,* external height of the implant; *D,* platform diameter of the implant; *R2,* reaction force on external hex.

- Bone trauma—drill sequence
- Decreased facial bone thickness may lead to recession
- Stress shielding
- Increased surgical failure rate
- Too close to adjacent tooth, PDL encroachment

PDL, Periodontal ligament.

implants had 5.8% screw loosening compared with 14.5% for standard-diameter implants.[65] When a larger diameter implant can not be inserted in the molar region, two regular-diameter implants rather than one wide-diameter implant is advantageous.[66-68]

Disadvantages of Wide-Bodied Implants

The disadvantages of a wide-bodied implant are primarily related to the surgical aspects and early healing period (Box 9-6). Several reports indicate a higher failure rate, not from loading failure, but during the early healing period.[69-72] It is hypothesized that the failure rate is related to the use of the implant as a rescue device in less than optimal conditions, such as when the regular size implant is not fixated or after extraction of a failed implant or tooth.

Alternatively, the higher bite forces and lower bone density of the posterior regions may not always be able to be addressed adequately by primarily implant width. The wider diameter, short implants in some of the literature had a higher failure rate than standard-diameter implants. For example, Ivanoff et al. in 1999, found 6-mm-long implants with a 5-mm diameter had a failure rate of 33% in the mandible and 10% in the maxilla.[70] The 8-mm-long, 5-mm-diameter implant failed 25% of the time in the maxilla and had a 33% failure rate in the mandible. In this study, the longer implants of 10-mm and 12-mm implants that were 5 mm in diameter reported 0% mandibular failure and a 10% failure in the maxilla. Therefore the increase in implant diameter was not enough to compensate for the shorter implants in this report.

There are other reports in the literature that question the use of wider diameter implants of any length. Eckert et al. in 2001 found 19% implant loss in the mandible and 29% in the maxilla of 85 wide-platform MK II implants in 63 patients.[71] In 2003, Attard and Zarb compared the success rate of the standard-diameter 3.75-mm-implant at 15 years and the 5-year survival of the wide-platform, 5-mm-diameter implant replacing posterior teeth. The standard-diameter implant had a 91.6% implant survival, whereas the 5-mm implant had a 76.3% rate.[72] In 2004, Shin et al. found an 80.9% survival rate of 5-mm-diameter, wide-bodied implants compared with 96.8% for the regular-diameter implant over a 5-year period.[73] These reports on the wide-body implant emphasized an elevated risk of failure compared with regular-diameter implants and the need for additional clinical trials. Ivanoff et al. stated the higher failure rate may be caused by an early learning curve, implants used in poor bone quality and the use of the wide-diameter implant as a rescue implant when the standard diameter did not reach stability or failed.[70]

A wide-bodied implant may be closer than 1.5 mm to the adjacent tooth or result with less than 1.5 mm of

WWW.HIGHDENT.IR

facial or palatal bone. As a result, the bone loss around the platform a "biologic width," microgap position, smooth metal below the bone, or loading bone loss from shear forces may cause bone loss on the adjacent tooth or facial bone loss and gingival recession.[67,73,74] Therefore abundant bone volume is necessary for wide-diameter implants to prevent these occurrences.

Stress shielding occurs when insufficient stress is transferred to the implant-bone interface, which results in disuse atrophy of the bone, similar to a condition when no strain is transferred to bone after tooth loss. Implants 6 mm or wider in the anterior regions of the mouth were initially inserted, because a central incisor root is approximately 6 mm at the cement-enamel junction. However, these implants often exhibited crestal bone loss and were too close to the adjacent labial incisors. As a result, bone loss and gingival recession were common. In the posterior regions, 8-mm-diameter implants were used in the molar sites. Again, the implant is so wide that the bone was often not strained enough to maintain a lamellar pattern. Instead, the lack of strain resulted in disuse atrophy and bone loss (titanium has 5 to 10 times the modulus of elasticity of cortical bone). The bending fracture resistance of an 8-mm-diameter implant is 16 times more than a 4-mm-diameter implant, and as a result, the stimulation of the interface may be too low to maintain bone. Therefore an implant should not be wider than 5 mm in the anterior when adequate mesiodistal space is present and force magnitude is also observed, and the implant should not be greater than 6 mm in the posterior, again when adequate forces exist to stimulate the bone.

NATURAL TEETH

The roots of the natural dentition optimize the amount and direction of forces found with the mouth. The periodontal ligament complex is a very effective organ that distributes occlusal loads over a large bone surface area, thereby reducing the stress on the bone and distributing the load away from the crestal bone and along the entire root surface. This is evidenced by the cribriform plate, which is cortical bone and disappears after the tooth loss.

The smallest diameter roots are in the mandibular anterior region, where the forces are less and the direction of force is along the long axis of the root. The maxillary anterior teeth have larger roots and a different cross-section shape to compensate for the off-axis loading that increases lateral forces on the structure. The canines have a greater root surface area in response to the higher bite forces (90 lb/in^2 compared with 35 lb/in^2) and the direction of force during mandibular excursions. The premolars have less surface area than the canine, because they do not receive a lateral load in excursions. The molars have multiple roots splinted together in one crown. The maxillary posterior region has the least bone density, whereas the mandibular counterpart has coarser trabecular bone. The maxillary molars have more roots than the mandibular components and therefore have more surface area to dissipate loads in the fine trabecular bone located in this region of the mouth. The molar crowns are almost twice as large in diameter and the root surfaces are twice those of the premolars. This compensates for the amount of load increase by two to three times and decreases the risk of damaging stresses.

The natural tooth roots may serve as an indicator for implant width requirements for prosthetic loads (Figures 9-17 and 9-18). In this light, the mandibular incisors regions and the maxillary lateral incisor may be replaced with 3- to 3.5-mm-diameter implants; the maxillary anteriors, premolars in both arches, and mandibular canine may use 4-mm-diameter implants. The molars may be restored with 5- or 6-mm-diameter implants in both arches. When larger diameter implants cannot be used in the molar region, two 4-mm-diameter implants for each molar should be considered.

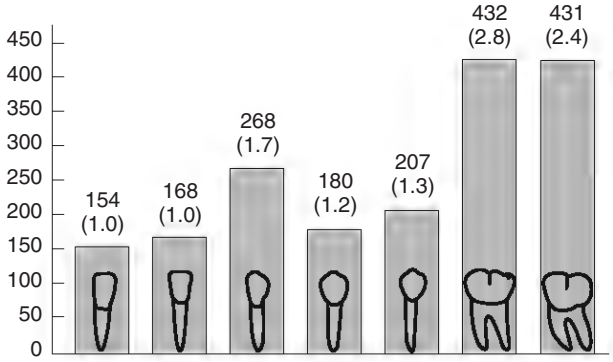


Figure 9-17 The implant diameter may be related to the natural teeth diameters, with narrower implants in the anterior and wider implants in the posterior regions.

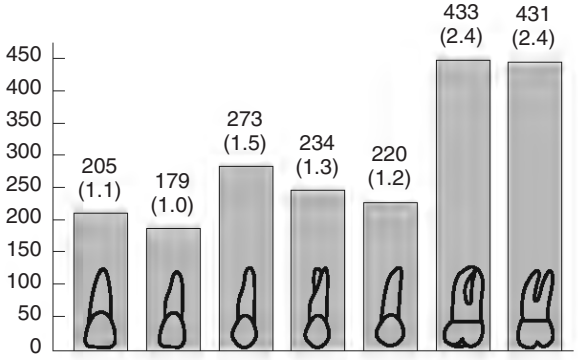


Figure 9-18 The maxillary molars have a larger diameter and surface area compared with any teeth in the mouth.

WWW.HIGHDENT.IR

Box 9-7 Implant Diameter Criteria

Esthetics

- Size of tooth (minimum implant size)
- Adjacent tooth-interproximal bone (maximum implant size)
- Faciopalatal bone (maximum implant size)

Function

- Functional surface area
- Fatigue strength

IMPLANT SIZE BASED ON ESTHETICS

Anterior Tooth Replacement

The average mesiodistal dimension for a central incisor is 8.6 mm for a male and 8.1 mm for a female, a lateral incisor is 6.6 mm for a male and 6.1 mm for a female, and a canine is 7.6 mm for a male and 7.2 mm for a female. However, the implant body should not be as wide as the natural tooth or clinical crown it replaces. Otherwise, the emergence contour and interdental papillae region cannot be established properly. The teeth become narrower as they proceed apically. The mesiodistal dimension of the maxillary central incisor at the cervix of the CEJ averages 6.4 mm, the lateral incisor dimension is 4.7 mm, and canine natural teeth at the cervix are 5.6 mm (Box 9-7).[75,76] This dimension is also too wide for an implant to restore the soft tissue and emergence profile of a crown associated with the implant. The bone level on natural teeth is 2 mm below the CEJ. The natural tooth dimensions at this bone level are reduced to 5.5 mm for central incisors, 4.3 mm for lateral incisors, and 4.6 mm for canines. Therefore the latter dimensions most closely approximate the widest implant diameter to mimic the emergence profile of a natural tooth as it emerges from the soft tissue drape.

The second factor that determines the mesiodistal ideal implant diameter to replace a tooth is the necessary distance from an adjacent tooth root or implant. The horizontal dimension of a wedge-shaped bone defect around an implant at the crest of the ridge from the biological width, implant design, or occlusal overload is about 1.4 mm wide.[77] When the implant is closer than 1.5 mm to an adjacent root of a tooth, the wedge-shaped vertical defect may become a horizontal defect, creating bone loss on the adjacent tooth root. Initial vertical bone loss around an implant during the first year of loading varies and ranges from 0.5 to more than 3 mm. This effect is of utmost importance because the interseptal bone height in part determines the incidence of presence or absence of the interdental papillae between the teeth or implants.[78] As a result, an implant should be at least 1.5 mm from the adjacent teeth whenever possible. Natural tooth roots are often closer than 1.5 mm to each other.

Therefore the ideal mesiodistal implant size is usually less than the natural root dimension. Most often, the ideal implant diameters used to replace the average size anterior tooth correspond to a 4.2-mm implant for a central incisor, a 3- to 3.5-mm implant for a lateral incisor, and a 4.2-mm implant for an average canine.

In summary, two conditions determine the ideal anterior tooth implant size in the mesiodistal dimension. The ideal diameter most often corresponds to the width of the missing natural tooth, 2 mm below the CEJ. In addition, the implant diameter plus 1.5 mm on each side should be equal to or less than the mesiodistal dimension between the two natural roots at the level of the crest of the residual ridge (see Box 9-7).

The implant dimension in question is the size of the crest module, not the implant body dimension. For example, a 4.1-mm crest module (on a 3.75-mm implant body) needs 7.1 mm of mesiodistal crestal bone, a 3.5-mm crest module (on a 3.25-mm implant body) is indicated for 6.5 mm of bone, and a 5.2-mm crest module requires 8.2 mm of bone.

The difference in the emergence profile between a 4-mm-diameter implant and a 5-mm-diameter implant is often not clinically relevant and provides a zone of safety related to the available bone around the implant. Therefore, when in doubt, a smaller size diameter implant should be selected. As such, a 4-mm-diameter implant often is used in the central implant position for a single-tooth replacement. Likewise, a 3-mm-diameter implant often is used for a lateral incisor single-tooth restoration. The exceptions to this rule may be in a bruxing patient. The wide-diameter implant will decrease abutment screw loosening, crestal bone loss, and risk of long-term implant body failure.

Multiple Anterior Implants

When implants are placed adjacent to each other, a minimum distance of 3 mm is suggested, especially when crestal bone loss is expected around the implants, to accommodate for eventual crestal bone loss and maintain interseptal bone levels. This distance permits a 1.5-mm defect to form on adjacent implants, without the vertical wedgelike defect becoming a horizontal defect and increasing the risk of tissue shrinkage between the implants.

The size dimension of two adjacent anterior implants should most often be reduced compared with the ideal dimensions of a single-tooth implant. The smaller implant diameters increase the amount of bone on the facial, increase the amount of soft tissue interdentally, and decrease the risk of esthetic complications.

Posterior Tooth Replacement

The natural maxillary first and second premolar teeth have an average mesiodistal size of 7.1 and 6.6 mm,

WWW.HIGHDENT.IR

respectively. The dimensions of these teeth at the CEJ are 4.8 and 4.7 mm.[75,76,79,80] At a distance of 2 mm below the CEJ, the teeth are also similar in size and average 4.2 and 4.1 mm. Therefore, although the first premolar is often slightly larger than the second premolar at the occlusal surface, both teeth are similar at the level of the bone.

The ideal implant size in the posterior maxilla depends on four criteria (Box 9-8):

1. The implant dimension should correspond to the natural tooth (2 mm below the CEJ).
2. The implant should be at least 1.5 mm from the adjacent teeth.
3. The implant should be at least 3 mm from an adjacent implant.
4. The implant should be at least 4 mm in diameter.

In this way, bone loss on one implant will not affect the adjacent tooth or implant, and the implant is strong enough to resist fatigue fracture and screw loosening under higher bite forces in the posterior region of the mouth.

The natural maxillary molars have the greatest diameter and largest surface area of any teeth (Figure 9-18). The maxillary molars have a 200% increase in surface area compared with the premolar teeth. The first molar is 10.4 mm in mesiodistal dimension, and the second molar is 9.8 mm. The CEJ dimensions of these teeth are 7.9 mm and 7.6 mm, respectively, and 2 mm below the CEJ these teeth are both 7 mm. However, the ideal implant diameter is 5 to 6 mm for the maxillary molars. Because titanium is 5 to 10 times more rigid than natural teeth, the modulus of elasticity for an implant of sizes greater than 6 mm may be too great and cause stress shielding in the bone-implant interface. As a result, bone loss around the implant may occur from inadequate stimulation of the bone. In addition, the 6-mm diameter should not be used in the anterior regions because the magnitude of the force is not large enough to strain the bone within the ideal physiologic zones next to such a wide-diameter implant.

When the diameters of molar implants do not provide sufficient surface area, the number of implants should be increased. Rather than two implants replacing the first and second molars, three or even four implants may be considered. Treatment plans for implant number to compensate for implant size may be used for very soft bone types or unfavorable force factors (i.e., parafunction). When multiple adjacent posterior teeth are missing, increasing the number of implants affects the overall surface area than the implant size.

SUMMARY

The ideal size of the implant body should be incorporated into a treatment plan, rather than the surgeon determining this dimension at the time of surgery. The initial size of an implant is determined in both length and diameter. In a two-stage healing protocol, the ideal implant length should be 12 to 16 mm. The softer the bone, the longer the implant requirements. The greater the bite force, the longer the implant dimension. Therefore the shortest implant length may be treatment planned in the anterior mandible, the anterior maxilla should have a slightly longer implant, the posterior mandible a longer implant, and the longest implant requirement for an ideal treatment plan is usually found in the posterior maxilla.

Box 9-8 Implant Size Selection Criteria in Posterior Maxilla

- Natural tooth 2 mm below cement-enamel junction
- 1.5 mm from adjacent tooth
- 3 mm from adjacent implant
- 4-mm-diameter minimum, for posterior maxilla

The diameter of the implant is also an important part of an ideal treatment plan. The diameter of an implant has a surgical, loading, and prosthetic consideration. In the initial treatment plan, the loading and prosthetic components are most important. The width of the implant is directly related to the overall functional surface area. Therefore, where the forces are greater or the bone is less dense, the implant is wider, ranging from 3 to 6 mm. As a general rule, the narrowest implant is found in the anterior mandible, followed by the anterior maxilla and the posterior mandible; the widest diameter requirements are found in the molar region of the posterior maxilla.

The prosthetic aspects of the implant width are primarily related to the esthetics of the emergence profile, the force on an abutment screw and the strength of the implant components. As a result, wider diameter implants are selected in the molar regions; standard diameters in the canines, premolars, and maxillary central incisors; and the smallest size implants in the maxillary lateral and mandibular incisors.

The natural dentition follows the guidelines established in the implant size treatment plan considerations. The correlation is most likely found because of the biomechanical relationship of the amount and type of the forces in the location of the jaws and the type of the bone in the region. Therefore, in the maxilla, fine trabecular bone is used to dissipate forces. The angle of the force to the premaxilla is 12 to 15 degrees, and the amount of force is the greatest in the molar region. The mandible is a force-absorbing unit and has coarse trabeculae and dense cortical bone. The tooth size difference is reflected in the diameter of the tooth, not in the overall length dimension. These guidelines are consistent when engineering principles determine tooth size.

WWW.HIGHDENT.IR

References

1. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaws, *Int J Oral Surg* 10:387-416, 1981.
2. Misch CE: Consideration of biomechanical stress in treatment with dental implants, *Dent Today* 25:80-85, 2006.
3. Brunski JB: Biomechanics of oral implants: future research directions, *J Dent Ed* 52:775-787, 1988.
4. Brunski JD, Hipp JA, Cochran GVB: The influence of biomechanical factors at the tissue-biomaterial interface. In Hanker JS, Gianmaria BC, editors: *Biomechanical materials and devices,* Pittsburgh, 1989, Materials Research Society.
5. Bidez MW, Misch CE: Force transfer in implant dentistry: basic concepts and principles, *J Oral Implant* 18:264-274, 1992.
6. Naert I, Koutsikakis G, Duyck J et al: Biologic outcome of implant-supported restorations in the treatment of partial edentulism. Part I: a longitudinal clinical evaluation, *Clin Impl Res* 13:381-389, 2002.
7. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, Dental Learning Systems Co., Inc., *Postgrad Dent* 3:3-17, 1995.
8. Frost HM: Mechanical adaptation: Frost's mechanostat theory. In Martin RB, Burr DB, editors. *Structure, function and adaption of compact bone,* New York, 1989, Raven Press.
9. Misch CE, Suzuki J, Misch-Dietsh FD et al: A positive correlation between occlusal trauma and peri-implant bone loss—literature support, *Implant Dent* 14:108-116, 2005.
10. Gibbs CH, Mahan PE, Mauderli A et al: Limits of human bite strength, *J Prosthet Dent* 56:226-229, 1986.
11. Scott I, Ash MM Jr: A six channel intra-oral transmitter for measuring occlusal forces, *J Prosthet Dent* 16:56, 1966.
12. Graf H: Bruxism, *Dent Clin North Am* 13:659-665, 1969.
13. Misch CE: Bone density: effect on treatment planning, surgical approach and progressive loading, *Int J Oral Implant* 6:23-31, 1990.
14. Misch CE, Qu Z, Bidez MW: Mechanical properties of trabecular bone in the human mandible: implications for dental treatment planning and surgical placement, *J Oral Maxillofac Surg* 57:700-706, 1999.
15. Misch CE: The effect of bruxism on treatment planning for dental implants, *Dent Today* 9:76-81, 2002.
16. Bidez MW, Misch CE: Issues in bone mechanics related to oral implants, *Implant Dent* 1:289-294, 1992.
17. Roberts WE, Garetto LP, De Castro RA: Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants with threaded or smooth surfaces: indications for provisional loading and axially directed occlusion, *J Indiana Dent Assoc* 68:19-24, 1989.
18. Misch CE, Bidez MW, Sharawy M: A bioengineered implant for a predetermined bone cellular response to loading forces: a literature review and case report: a case report demonstrating proof of principle, *J Periodontol* 72:1276-1286, 2001.
19. Cowin SC: *Bone mechanics,* Boca Raton, Fla, 1989, CRC Press.
20. Borchers I, Reidhart P: Three dimensional stress distribution around dental implants at different stages of interface development, *J Dent Res* 62:155-159, 1994.
21. Misch CE, Bidez MW: Implant protected occlusion: a biomechanical rationale, *Compend Contin Dent Educ* 15:1330-1343, 1994.
22. Misch CE: Divisions of available bone in implant dentistry, *Int J Oral Implantol* 7:9-17, 1990.
23. Cawood JJ, Howell RA: A classification of the edentulous jaw classes I to VI, *Int J Oral Maxillofac Surg* 17:232-279, 1988.
24. Engquist B, Bergendal T, Kallus T et al: A retrospective multicenter evaluation of osseointegrated implants supporting overdentures, *Int J Oral Maxillofac Impl* 3:129-134, 1988.
25. Friberg B, Jemt T, Lekholm U: Early failures in 4,641 consecutively placed Brånemark dental implants: a study from Stage I surgery to the connection of completed prostheses, *Int J Oral Maxillofac Impl* 6:142-146, 1991.
26. Jaffin RA, Berman CL: The excessive loss of Brånemark fixtures in Type IV bone: a 5-year analysis, *J Periodontol* 62:2-4, 1991.
27. Minsk L, Polson A, Weisgold A et al: Outcome failures of endosseous implants from a clinical training center, *Compend Contin Educ Dent* 17:848-856, 1996.
28. Orenstein IH, Syman WJ, Truhlar RS et al: Bone quality in patients receiving endosseous dental implants: DICRG Interim report no. 1, *Implant Dent* 3:90-94, 1994.
29. Sevimay M, Turhan F, Kilicarslan MA et al: Three-dimensional finite element analysis of the effect of different bone quality on stress distribution in an implant-supported crown, *J Prosthet Dent* 93:227-234, 2005.
30. Misch CE: Short versus long implant concepts—functional surface area, *Dent Today* 18:60-65, 1999.
31. Misch CE: Short versus long implant concepts—functional surface area, *Oral Health* 89:13-21, 1999.
32. Rieger MR, Adams WK, Kinzel GL et al: Finite element analysis of bone-adapted and bone-bonded endosseous implants, *J Prosthet Dent* 62:436-440, 1989.
33. Sharawy M, Tehemar M, Weller N et al: Heat generation during implant drilling, Report 1, the significance of motor speed, *J Oral Maxillofac Surg* 10:1160-1169, 2002.
34. Oikarinen K, Raustia AM, Hartikainen M: General and local contraindications for endosteal implants: an epidemiological panoramic radiographic study in 65-year-old subjects, *Comm Dent Oral Epidemiol* 23:114-118, 1995.
35. Goodacre CJ, Bernal G, Runcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
36. das Neves FD, Fones D, Bernardes SR et al: Short implants—an analysis of longitudinal studies, *Int J Oral Maxillofac Implants* 21:86-93, 2006.
37. Winkler S, Morris HF, Ochi S: Implant survival to 36 months as related to length and diameter, *Ann Periodontol* 5:22-31, 2000.
38. Weng D, Jacobson Z, Tarnow D et al: A prospective multicenter clinical trial of 3i machined-surface implants: results after 6 years of follow-up, *Int J Oral Maxillofac Implants* 18:417-423, 2003.
39. Jemt T, Lekholm U: Implant treatment in edentulous maxillae: a 5-year follow-up report on patients with different degrees of jaw resorption, *Int J Oral Maxillofac Implants* 10:303-311, 1995.
40. Saadoun AP, Le Gall MG: An 8-year compilation of clinical results obtained with Steri-Oss endosseous implants, *Comp Contin Educ Dent* 17:669-668, 1996.
41. Higuchi KW, Folmer T, Kultje C: Implant survival rates in partially edentulous patients: a 3-year prospective multicenter study, *J Oral Maxillofac Surg* 53:264-268, 1995.

WWW.HIGHDENT.IR

42. Lekholm U, Gunne J, Henry P et al: Survival of the Brånemark implant in partially edentulous jaws: a 10-year prospective multicenter study, *Int J Oral Maxillofac Implants* 14:639-645, 1999.
43. van Steenberghe D, De Mars G, Quirynen M et al: A prospective split-mouth comparative study of two screw-shaped self-tapping pure titanium implant systems, *Clin Oral Implants Res* 11:202-209, 2000.
44. Testori T, Wiseman L, Woolfe S et al: A prospective multicenter clinical study of the Osseotite implant: four-year interim report, *Int J Oral Maxillofac Implants* 16:193-200, 2001.
45. Tawil G, Aboujaoude, Younan R: Influence of prosthetic parameters on survival and complication rates of short implants, *Int J Oral Maxillofac Implants* 21:275-282, 2006.
46. Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study, *J Periodontol* 77: 1340-1347, 2006.
47. De Bruyn H, Collaert B, Linden U et al: Clinical outcome of screw vent implants: a 7-year prospective follow-up study, *Clin Oral Implants Res* 10:139-148, 1999.
48. Misch CE: Short dental implants: a literature review and rationale for use, *Dent Today* 24:64-68, 2005.
49. Tada S, Stegaroiu R, Kitamura E et al: Influence of implant design and bone quality on stress/strain distribution in bone around implants: a 3-dimensional finite element analysis, *Int J Oral Maxillofac Implants* 18:357-368, 2003.
50. Lum LB, Osier JF: Load transfer from endosteal implants to supporting bone: an analysis using statics. Part one: horizontal loading. *J Oral Implantol* 18:343-348, 1992.
51. Lum LB: A biomechanical rationale for the use of short implants, *J Oral Implantol* 17:126-131, 1991.
52. Griffin TJ, Cheung WS: The use of short, wide implants in posterior areas with reduced bone height: a retrospective investigation, *J Prosthet Dent* 92:139-144, 2004.
53. Rocuzzo M, Bunino M, Prioglio F et al: Early loading of sandblasted and acid-etched (SLA) implants: a prospective split-mouth comparative study, *Clin Oral Implants Res* 12:572-578, 2001.
54. Scialom J: Tripodial pin implants in oral implantology. In Cranin NA, editor: *Oral implantology,* Springfield, Ill, 1982, Charles C Thomas.
55. Langer B, Langer I, Herrmann I et al: The wide fixture: a solution for special bone situations and a rescue for the compromised implants, part I, *Int J Oral Maxillofac Implants* 8:400-408, 1993.
56. Jividen GI Jr: Immediate placement of wide-diameter implants in the premaxilla, *Dent Implantol Update* 9: 89-92, 1998.
57. Petrie CS, Williams JL: Comparative evaluation of implant design: influence on diameter, length and taper on strains in the alveolar crest. A three-dimensional finite element analysis, *Clin Oral Implants Res* 16:486-494, 2005.
58. Himmlova L, Dostaloua T, Kacovsky A et al: Influence of implant length and diameter on stress distribution: a finite element analysis, *J Prosthet Dent* 91:20-25, 2004.
59. Aparicio C, Orozco P: Use of 5-mm diameter implants: periotest values related to a clinical and radiographic evaluation, *Clin Oral Implants Res* 9:398-406, 1998.
60. Graves SL, Jansen CE, Siddiqui AA et al: Wide diameter implants: indications, considerations, and preliminary results over a two-year period, *Aust Prosthodont J* 8:31-37, 1994.
61. Krenmair G, Waldenberger O: Clinical analysis of wide diameter Frialit 2 implants, *Int J Oral Maxillofac Implants* 19:710-715, 2004.
62. Anner R, Better H, Chaushu G: The clinical effectiveness of 6 mm diameter implants, *J Periodontol* 76:1013-1015, 2005.
63. Jarvis WC: Biomechanical advantage of wide-diameter implants, *Compend Contin Educ Dent* 18:687-694, 1997.
64. Boggan S, Strong JT, Misch CE et al: Influence of hex geometry and prosthetic table width on static and fatigue strength of dental implants, *J Prosthet Dent* 82:436-440, 1999.
65. Cho SC, Small PN, Elian N et al: Screw loosening for standard and wide diameter implants in partially edentulous cases: 3- to 7-year longitudinal data, *Implant Dent* 13:245-250, 2004.
66. Luong SL, Brooks CN: Replacement of missing single molar with single wide-diameter implants or double standard endosseous implants, *Va Dent J* 76:45-46, 1999.
67. Handelsman M: Treatment planning and surgical considerations for placement of wide body implants, C*ompend Contin Educ Dent* 19:507-514, 1998.
68. Cehreli MC, Akca K: Narrow-diameter implants as terminal support for occlusal three-unit FODs: a biomechanical analysis, *Int J Periodontics Restorative Dent* 24:513-519, 2004.
69. Allahyar G, Morgano SM: Finite element analysis of three designs of an implant-supported molar crown, *J Prosthet Dent* 92:434-400, 2004.
70. Ivanoff CJ, Grondahl K, Sennerby L et al: Influence of variations in implant diameters: a 3- to 5-year retrospective clinical report, *Int J Oral Maxillofac Implants* 14:173-180, 1999.
71. Eckert SE, Meraw SJ, Weaver AL et al: Early experience with wide-platform MK II implants. Part I: implant survival. Part II: evaluation of risk factors involving implant survival, *Int J Oral Maxillofac Implants* 16:208-216, 2001.
72. Attard NJ, Zarb GA: Implant prosthodontic management of partially edentulous patients missing posterior teeth: the Toronto experience, *J Prosthet Dent* 89:352-359, 2003.
73. Shin SW, Bryant SR, Zarb GA: A retrospective study in the treatment outcome of wide-bodied implants, *Int J Prosthodont* 17:52-58, 2004.
74. Small PN, Tarnow DP, Cho SC: Gingival recession around wide-diameter versus standard-diameter implants: a 3- to 5-year longitudinal prospective study, *Pract Perio Aesthet Dent* 13:143-146, 2001.
75. Spray R, Black CG, Morris HF et al: The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering, *Ann Periodontol* 5:119-128, 2000.
76. Woelfe JB: *Dental anatomy: its relevance to dentistry,* ed 4, Philadelphia, 1990, Lea & Febiger.
77. Kraus BS, Jordan RE, Abrams L: *Dental anatomy and occlusion,* Baltimore, 1969, Williams and Wilkins.
78. Tarnow DP, Cho SC, Wallace SS: The effect of interimplant distance on the height of interimplant bone crest, *J Periodontol* 71:546-569, 2000.
79. Tarnow DP, Magner AW, Fletcher P: The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal papilla, *J Periodontol* 63:995-996, 1992.
80. Kois MC: Altering gingival levels: the restorative connection. 1. Biologic variable, *J Esthet Dent* 6:3-9, 1994.

WWW.HIGHDENT.IR

Chapter 10

Available Bone and Dental Implant Treatment Plans

Carl E. Misch

Long-term success in implant dentistry requires the evaluation of more than 50 dental criteria, many of which are unique to this discipline.[1] However, the doctor's training and experience and the amount and density of available bone in the edentulous site of the patient are arguably primary determining factors in predicting individual patient success. In the past, the available bone was not modified and was the primary intraoral factor influencing the treatment plan. Today the prosthodontic needs and desires of the patient should be first determined, relative to the number and position of missing teeth. After the intended prosthesis is designed, the patient force factors and bone density are then evaluated. The key implant positions, implant number, and size are determined. After these factors are considered, the most important element in the implant region is the available bone. Greenfield already appreciated its importance in 1913.[2] This chapter describes the three-dimensional concept of available bone and the implant treatment options for each type of bone anatomy.

Figure 10-1 Maxillary and mandibular atrophy after tooth loss was documented by J. Misch in 1922.

LITERATURE REVIEW

The process of bone volume atrophy after tooth loss and loss of alveolus has been fully documented (Figure 10-1).[3-19] Characteristic bone volume changes after tooth loss were evaluated in the anterior mandible by Atwood (Figure 10-2).[4-6] The six residual ridge stages are beneficial to appreciate the shapes and range of bone loss. Tallgren reported the amount of bone loss occurring the first year after tooth loss is almost 10 times greater than the following years.[7] The posterior edentulous mandible resorbs at a rate approximately four times faster than the anterior edentulous mandible.[8] It has been suggested that, in the mandibular synthesis, females present higher total reduction and more rapid bone loss during the first 2 years.[9] More recent studies in complete denture wearers have confirmed the higher rate of resorption in the first year of edentulouness.[10,11] The anterior maxilla resorbs in height slower than the anterior mandible. However, the original height of available bone in the anterior mandible is twice as much as the anterior maxilla. Therefore the resultant maxillary atrophy, although slower, affects

Figure 10-2 Atwood presented a classification of bone loss after tooth loss in the anterior mandible in 1963.

WWW.HIGHDENT.IR

the potential available bone for an implant patient with equal frequency.[7] The changes in the edentulous anterior maxillary ridge dimension can be dramatic in height and width (up to 70%), especially when multiple extractions are performed.[12] In addition, many patients lose additional bone by simultaneous alveolectomy procedures after tooth extraction before the delivery of a maxillary denture.[13] Although slight differences exist between different alveolectomy techniques, all are detrimental to the ridge volume.[14]

The residual ridge shifts palatally in the maxilla and lingually in the mandible as related to tooth position, at the expense of the buccal cortical plate in all areas of the jaws, regardless of the number of teeth missing.[15-19] However, after the initial bone loss, the maxilla continues to resorb toward the midline, whereas the mandibular basal bone is wider than the original alveolar bone position and results in the late mandible resorption progressing facially. This, in addition to a marked change in mandibular position, leads to the classical appearance of the denture wearer with a protruding chin and a mandibular lip.[20] The posterior maxilla loses bone volume faster than any other region. Not only does periodontal disease cause initial bone loss before the loss of teeth, the crestal bone loss is substantial after tooth extraction. In addition, the maxillary sinus after tooth loss expands toward the crest of the edentulous ridge. As a result, the posterior maxilla is more often indicated for bone augmentation compared with any other intraoral location.

Weiss and Judy[21] developed a classification of mandibular atrophy and its influence on subperiosteal implant therapy in 1974. Kent presented a classification of alveolar ridge deficiency designed for alloplastic bone augmentation in 1982.[22] Another bone volume classification was proposed by Lekholm and Zarb in 1985 for residual jaw morphology related to the insertion of Brånemark fixtures.[23] They described five stages of jaw resorption, ranging from minimal to extreme (Figure 10-3). The mandibular resorption was only described in loss of height. All the five stages of resorption in either arch used the same implant modality, surgical approach, and type of final prosthesis. In addition, as the bone volume decreased, the number of implants decreased.

A maxillary alveolar process of resorption after tooth loss after Atwood's description for the mandible was presented by Fallschüssel in 1986.[24] The six resorption categories of this arch ranged from fully preserved to moderately wide and high, narrow and high, sharp and high, wide and reduced in height, and severely atrophic. The classifications of Atwood, Zarb and Lekholm, and Fallschüssel do not describe the actual resorption process in chronological order and are more descriptive of the residual bone.[25] Another bone resorption classification, which included the expansion of the maxillary sinuses, was also proposed by Cawood and Howell in 1988.[26] Although similar to other categories, the bone volume changes are not reflective of the changes required for implant placement or bone grafting procedures.

In 1985, Misch and Judy established four basic divisions of available bone for implant dentistry in the edentulous maxilla and mandible, which follow the natural resorption phenomena of each region, and determined a different implant approach to each category.[1,27-33] The angulation of bone and crown height were also included for each bone volume, because they affect the prosthetic treatment. These original four divisions of bone were further expanded with two subcategories to provide an organized approach to implant treatment options for surgery, bone grafting, and prosthodontics (Figure 10-4). The ability to organize the available bone of the potential implant site into specific related categories of common treatment options and conditions is of benefit to both the beginning and experienced clinician alike. Improved communication among health professionals and the collection of relevant specific data for each category are also beneficial. The Misch-Judy bone classification has facilitated these processes during the past two decades

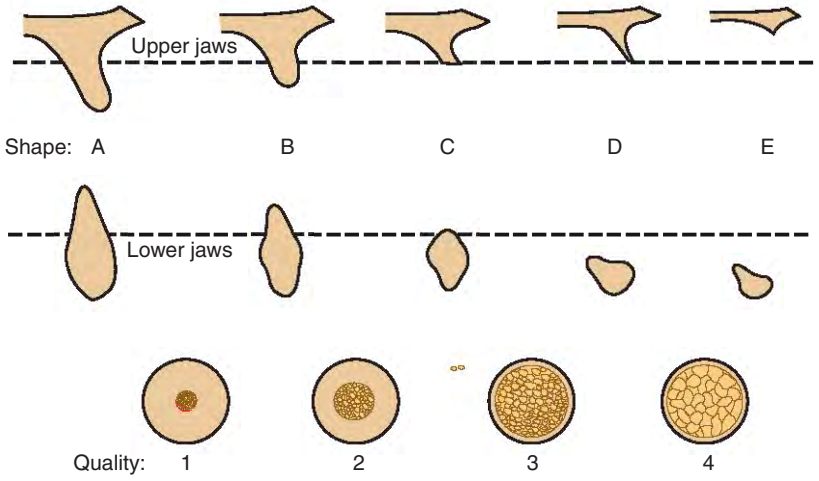


Figure 10-3 Lekholm and Zarb presented a classification of bone loss in the edentulous jaws in 1985.

WWW.HIGHDENT.IR

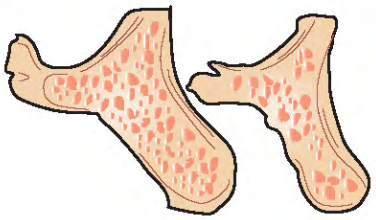


Figure 10-4 In 1985, Misch and Judy presented a classification of available bone (Divisions A, B, C, D), which is similar in both arches in 1985. Implant, bone grafting methods, and prosthodontic related treatment was suggested for each category of bone. *h*, Inadequate height; *w*, inadequate width.

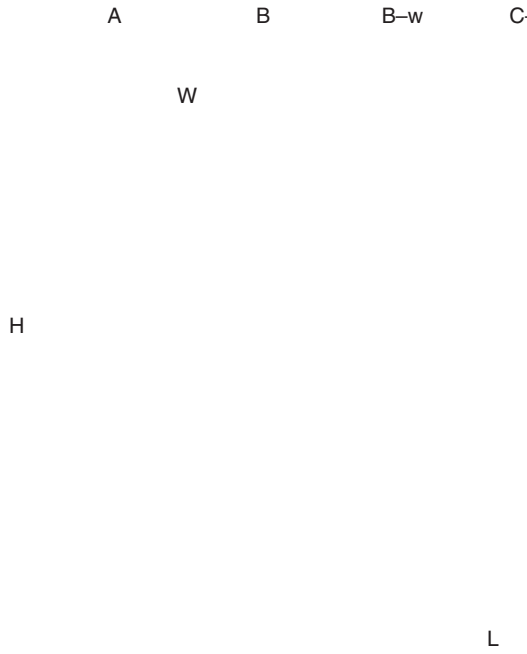


Figure 10-5 Available bone is measured in height *(H)*, width *(W)*, and length *(L)*. Also considered are crown height space and angulation of bone (direction of force to the implant body).

within the profession, universities, implant programs, and international implant societies.

AVAILABLE BONE

The category and design of the final prosthesis and key implant positions are first determined after a patient interview and evaluation of existing medical and dental conditions. The patient force factors and bone density are of particular note. The abutments necessary to support the restoration are then established in implant number and size and without initial regard to the available bone conditions.

Available bone describes the amount of bone in the edentulous area considered for implantation. It is measured in width, height, length, angulation, and crown height space (Figure 10-5). Historically, the available bone was not modified and dictated the implant position and size. Today, if the bone is inadequate to support an ideal abutment for the intended prosthesis or bone grafting, the ideal site is often indicated or an alternative site may be considered.

As a general guideline, 1.5 to 2 mm of surgical error is maintained between the implant and any adjacent landmark. This is especially critical when the opposing landmark is the mandibular inferior alveolar nerve. However, the implant may be placed without complication through the cortical plate of the maxillary sinus or inferior border of the mandible. The implant may also be positioned closer to the cribriform plate of a natural tooth. If the implant should become mobile or affected by periimplant disease, the adjacent landmark may be adversely involved. Likewise, if the sinus becomes infected or the adjacent tooth suffers from periodontal disease, the implant may be affected.

Manufacturers describe the root form implant in dimensions of width and length. The implant length corresponds to the height of available bone. Therefore this text refers to root form implant height or length. The width of a root form implant is most often related to the diameter and mesiodistal length of available bone. Most root form implants have a round cross-sectional design to aid in surgical placement; therefore the diameter of the implant corresponds to the implant width. Many manufacturers propose implants with a crest module wider than the implant body dimension.

WWW.HIGHDENT.IR

Yet the often stated dimension of the manufacturer is the smaller body width. For example, the Nobel Biocare 3.75-mm-diameter implant has a 4.1-mm-crest module. The clinician should be knowledgeable of all implant dimensions, especially because the crestal dimension of bone (where the wider crest module dimension is placed) is usually the narrowest region of the available bone and where the implant is closest to an adjacent tooth.

All teeth are not equal when considered as abutments for a prosthesis. The restoring dentist knows how to evaluate the surface area of the natural abutment roots. A healthy maxillary first molar with more than 450 mm^2 of root surface area constitutes a better abutment for a fixed prosthesis than a mandibular lateral incisor with 150 mm^2 of root support. The larger diameter teeth correspond to the regions of the mouth with greater bite force. It is interesting to note the increase in surface area for natural teeth is most dependent upon diameter and a change in design, more so than length.

Implant Width

All sizes and designs of implants do not have the same surface area and should not be considered as equals for prosthetic abutments. With a greater surface area of implant-bone contact, less stress is transmitted to the bone, and the implant prognosis improved. For a generic cylinder root form implant design, each 0.25-mm increase in diameter corresponds to a surface area increase of approximately 5% to 8%. Therefore a cylinder root form implant 1 mm greater in diameter will have a total surface area increase of approximately 20% to 30%. Because stress *(S)* equals force *(F)* divided by the functional area *(A)* over which it is applied *(S = F/A)*, the greater diameter decreases the amount of stress at the crestal bone implant interface. Because early bone loss relates to the crestal bone regions and prosthetic complications may be related to the crest module size of an implant, the width of the implant is much more critical than its height, after a minimum height has been obtained.

Implant Height

The height of the implant also affects its total surface area. A cylinder root form implant 3 mm longer provides 20% to 30% increase in surface area. The advantage of increased height does not express itself at the crestal bone interface but rather in initial stability of the implant, the overall amount of bone-implant interface, and a greater resistance to rotational torque during abutment screw tightening. The increased height of an implant in an immediate extraction site larger in diameter than the implant also increases the initial bone contact percent, which can decrease the initial risk of movement at the interface. In addition, the crestal bone and opposing anatomical landmark are often composed of cortical bone, which is denser and stronger than trabecular bone. As a result, it may help stabilize the implant while the trabecular woven bone forms. In this way, a direct bone-implant interface is encouraged. This may be of particular advantage when an immediate-loading protocol of implants is used for a transitional prosthesis. However, after the implant has healed, the crestal region is the zone that receives the majority of the stress. As a result, implant length is not as effective as the width to decrease crestal loads around an implant.

The minimum height for endosteal implants, long-term survival is in part related to the density of bone. The more dense bone may accommodate a shorter implant (i.e., 8 mm), and the least dense, weaker bone requires a longer implant (i.e., 12 mm). After the minimum implant height is established for each implant design and bone density, the width is more important than additional length. This chapter primarily presents the volume of bone requirements for ideal bone density situations or D2, which is coarse trabecular bone surrounded by porous to dense cortical bone.

Available Bone Height

The available bone height is first estimated by radiographic evaluation in the edentulous ideal and optional regions, where implant abutments are required for the intended prosthesis. A panoramic radiograph is the most common method for the preliminary determination of the available bone height.

The height of available bone is measured from the crest of the edentulous ridge to the opposing landmark. The anterior regions are limited by the maxillary nares or the inferior border of the mandible. The anterior regions of the jaws have the greatest height, because the maxillary sinus and inferior alveolar nerve limit this dimension in the posterior regions. The maxillary canine eminence region often offers the greatest height of available bone in the maxillary anterior.[34] In the posterior jaw region, there is usually greater bone height in the maxillary first premolar than in the second premolar, which has greater height than the molar sites because of the concave morphology of the maxillary sinus floor. Likewise, the mandibular first premolar region is usually anterior to the mental foramen and provides the most vertical column of bone in the posterior mandible. However, on occasion, this premolar site may present a reduced height compared with the anterior region, because of the anterior loop of the mandibular canal (when present) as it passes below the foramen and proceeds superiorly, then distally, before its exit through the mental foramen (Figure 10-6).

The dilemma of available bone in implant dentistry involves the existing anatomy of the edentulous mandible and maxilla. The initial mandibular bone

WWW.HIGHDENT.IR

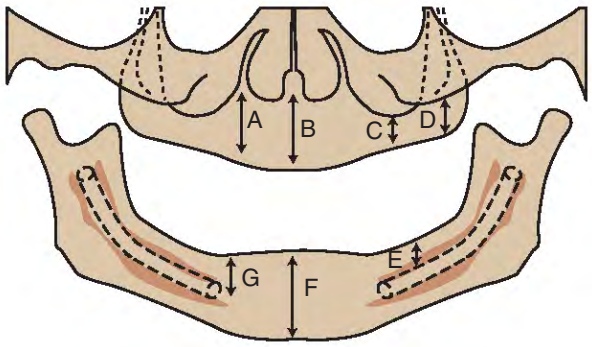


Figure 10-6 The height of available bone is measured from the crest of the edentulous ridge to the opposing landmark. The opposing landmark may be in the maxillary canine region *(A)*, floor of the nares *(B)*, maxillary sinus *(C)*, tuberosity *(D)*, mandibular canine region *(G)*, anterior mandible *(F)*, or bone above the inferior mandibular canal *(E)*.

height is influenced by skeletal anatomy, with angle Class II patients having shorter mandibular height, and angle Class III patients exhibiting the greatest height. The initial edentulous anterior maxillary available bone height is less than the mandibular available bone height. The opposing landmarks for the initial available bone height are more limiting in the posterior regions. The posterior mandibular region is reduced because of the presence of the mandibular canal, approximately situated 12 mm above the inferior border of the mandible. As a result, in the areas where greater forces are generated and the natural dentition has wider teeth with two or three roots, shorter implants, if any, are often used and in insufficient number because of the anatomical limiting factors. A study of 431 patients revealed that in the partially edentulous maxilla and mandible, the placement of posterior implants at least 6 mm in length was possible in only 38% and 50%, respectively. The anterior regions of edentulous arches could receive implants 55% and 61% of the time, respectively.[35] The existing bone anatomy of the implant patient often requires modification to enhance long-term implant success. For example, sinus grafts in the posterior maxilla permit the placement of posterior endosteal implants into restored bone height.

The suggested minimum bone height for predictable long-term endosteal implant survival approaches 12 mm. Before 1981 the Brånemark screw–type implant body and osteointegrated approach was provided as a single diameter (3.75 mm) and was used only in the completely edentulous anterior maxilla and mandible.[36] The implant drills cut 10 mm deep, and the "10-mm" implant was 9 mm in length. By 1990 this philosophy had been expanded to all jaw regions and many implant sizes. However, failure rates reported in the literature for implants shorter than 9 mm tend to be higher independently from the manufacturer design, surface characteristic, and type of application.[37-52] Therefore the initial implant length should approximate 12 mm, because the ideal treatment plan should first be designed. The inexperienced dentist may wish to have 14 mm of bone height to place a 12-mm-high implant body before proceeding with the surgery. This precaution allows 2-mm surgical error or permits an osteoplasty to gain additional width of bone. The 12-mm height minimum applies to most screw-shape endosteal implant designs in good-density (D2) bone. This minimum implant height requirement may be reduced in the very dense bone (D1) in the symphysis of an atrophic mandible, when the prosthesis has fewer forces (as an overdenture) or when the shorter dimension can be compensated by implant number, width, or design.[53-55]

The available bone height in an edentulous site is the most important dimension for implant consideration, because it affects both implant length and crown height. Crown height affects force factors and esthetics. In addition, bone augmentation is more predictable in width than height, so even when the width is inadequate for implant placement, bone grafting may be used to create a site ideal for restorative and implant insertion requirements.

Available Bone Width

The width of available bone is measured between the facial and lingual plates at the crest of the potential implant site. The crest of the edentulous ridge is most often supported by a wider base. In most areas, because of this triangular-shaped cross section, an osteoplasty provides greater width of bone, although of reduced height. However, the anterior maxilla often does not follow this rule, because most edentulous ridges exhibit a labial concavity in the incisor area, with an hourglass configuration. Crest reduction affects the location of the opposing landmark, with possible consequences for surgery, implant height selection, appearance, and the design of the final prosthesis. This is particularly important when an FP-1 prosthesis is planned, with the goal of obtaining a normal contour and proper soft tissue drape around a single tooth replacement.

After adequate height is available, the next most significant criterion affecting long-term survival of endosteal implants is the width of the available bone. Root form implants of 4-mm crestal diameter usually require more than 6 mm of bone width to ensure sufficient bone thickness and blood supply around the implant for predictable survival. These dimensions provide more than 1 mm of bone on each side of the implant at the crest. Because the bone usually widens apically, this minimum dimension rapidly increases. For root form implants, the minimum bone thickness is located in the midfacial and midlingual contour of the crestal region exclusively (Figure 10-7). The crestal aspect of the residual ridge is often cortical in nature and exhibits greater density than the underlying trabecular bone regions, especially in the mandible. This mechanical advantage permits immediate fixation

WWW.HIGHDENT.IR

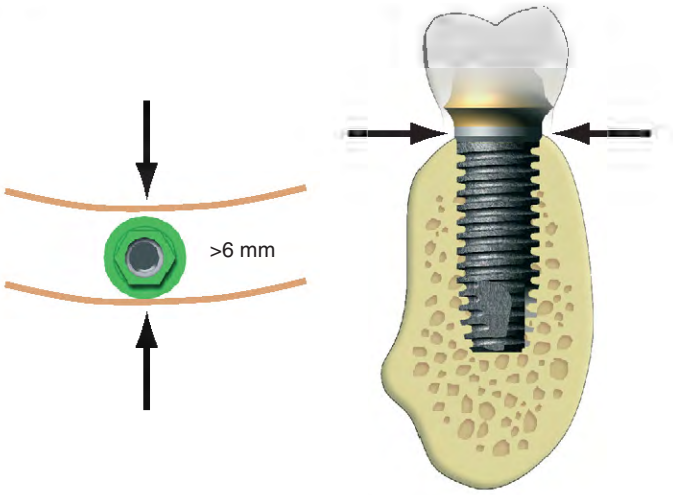


Figure 10-7 Minimum bone width for a 4-mm-diameter root form is 6 mm in the midfacial and lingual regions, because the round implant design results in more bone in all other dimensions (width and height).

of the implant, provided this cortical layer has not been removed by osteoplasty.

The initial width of available bone is related to the initial crestal bone loss after implant loading. Edentulous ridges that are greater than 6 mm in width have demonstrated less crestal bone loss than when minimum bone dimensions are available. Extraction sockets having more width at the crest also lose less bone during initial healing than sites with minimum width of cortical plates on the facial or lingual of the extraction site.

Available Bone Length

The mesiodistal length of available bone in an edentulous area is often limited by adjacent teeth or implants. As a general rule, the implant should be at least 1.5 mm from an adjacent tooth and 3 mm from an adjacent implant. This dimension not only allows surgical error, but also compensates for the width of an implant or tooth crestal defect, which is usually less than 1.4 mm. As a result, if bone loss occurs at the crest module of an implant or from periodontal disease with a tooth, the vertical bone defect will not spread to a horizontal defect and cause bone loss on the adjacent structure.[56] Therefore, in the case of a single-tooth replacement, the minimum length of available bone necessary for an endosteal implant depends on the width of the implant. For example, a 5-mm-diameter implant should have at least 8 mm of mesiodistal bone, so 1.5 mm is present on each side of the implant. A minimum mesiodistal length of 7 mm is usually sufficient for a 4-mm-diameter implant. Of course, the diameter of the implant is also related to the width of available bone and, in multiple adjacent sites, is primarily limited in this dimension. For example, a width of bone of 4.5 mm without augmentation requires a 3.5-mm or smaller implant, with inherent compromises (such as minimal surface area and greater crestal stress concentration under occlusal loads). Therefore in the narrower ridge, it is often indicated to place two or more narrow-diameter implants when possible to obtain sufficient implant-bone surface area to compensate for the deficiency in width of the implant. Because the implants should be 3 mm apart and 1.5 mm from each tooth, 13 mm or more in available bone mesiodistal length may be required when the narrower implant dimensions are used to replace a posterior tooth.

The ideal implant width for single-tooth replacement or multiple adjacent implants is often related to the natural tooth being replaced in the site. The tooth has its greatest width at the interproximal contacts, is narrower at the cement-enamel junction (CEJ), and is even narrower at the initial crestal bone contact, which is 2 mm below the CEJ.[57] The ideal implant diameter corresponds to the width of the natural tooth 2 mm below the CEJ, if it also is 1.5 mm from the adjacent tooth. In this way, the implant crown emergence through the soft tissue may be similar to a natural tooth. For example, a maxillary first premolar is approximately 8 mm at the interproximal contact, 5 mm at the CEJ, and 4 mm at a point 2 mm below the CEJ. Therefore a 4-mm-diameter implant (at the crest module) would be the ideal implant diameter, if it also is at least 1.5 mm from the adjacent roots (2 mm below the CEJ).

Available Bone Angulation

Bone angulation is the fourth determinant for available bone. The initial alveolar bone angulation represents the natural tooth root trajectory in relation to the occlusal plane. Ideally, it is perpendicular to the plane of occlusion, which is aligned with the forces of occlusion and is parallel to the long axis of the prosthodontic restoration. The incisal and occlusal surfaces of the teeth follow the curve of Wilson and curve of Spee. As such, the roots of the maxillary teeth are angled toward a common point approximately 4 inches away. The mandibular roots flare, so the anatomical crowns are more lingually inclined in the posterior regions and labially inclined in the anterior area compared with the underlying roots. The first premolar cusp tip is usually vertical to its root apex.

The maxillary anterior teeth are the only segment in either arch that does not receive a long axis load to the tooth roots, but instead are usually loaded at a 12-degree angle. As such, their root diameter is greater than the mandibular anterior teeth. In all other regions, the teeth are loaded perpendicular to the curves of Wilson or Spee.

Rarely does the bone angulation remain ideal after the loss of teeth, especially in the anterior edentulous arch (Figure 10-8). In this region, labial undercuts

WWW.HIGHDENT.IR

Figure 10-8 The angulation of bone may not be in the long axis of the missing tooth, especially in the anterior regions of the mouth. The computed tomography scan was obtained with a barium sulfate radiopaque template over the edentulous site of a lower anterior tooth. The available bone trajectory diverges 30 degrees from the tooth's angulation.

and resorption after tooth loss,[12,15,16] often mandate greater angulation of the implants or correction of the site before insertion. In the posterior mandible, the submandibular fossa mandates implant placement with increasing angulation as it progresses distally. Therefore, in the second premolar region, the angulation may be 10 degrees to a horizontal plane; in the first molar areas, 15 degrees; and in the second molar region, 20 to 25 degrees.

The limiting factor of angulation of force between the body and the abutment of an implant is correlated to the width of bone. In edentulous areas with a wide ridge, wider root form implants may be selected. Such implants may allow up to 25 degrees of divergence with the adjacent implants, natural teeth, or axial forces of occlusion with moderate compromise. The angled load to an implant body increases the crestal stresses, but the greater diameter implant decreases the amount of stress transmitted to the crestal bone. In addition, the greater width of bone offers some latitude in angulation at implant placement. The implant body may often be inserted so as to reduce the divergence of the abutments without compromising the permucosal site. Therefore an acceptable bone angulation in the wider ridge may be as much as 25 degrees.

The narrow yet adequate width ridge often requires a narrower design root form implant. Compared with larger diameters, smaller diameter designs cause greater crestal stress and may not offer the same range of custom abutments. In addition, the narrower width of bone does not permit as much latitude in placement regarding angulation within the bone. This limits the acceptable angulation of bone in the narrow ridge to 20 degrees from the axis of the adjacent clinical crowns or a line perpendicular to the occlusal plane.

Crown Height Space

The crown height space (CHS) is defined as the vertical distance from the crest of the ridge to the occlusal plane. It affects the appearance of the final prosthesis and the amount of moment force on the implant and surrounding crestal bone during occlusal loading. The CHS may be considered a vertical cantilever. Any direction of load that is not in the long axis of the implant will magnify the crestal stresses to the implant-bone interface and also to the abutment screws in the restoration. The greater the CHS, the greater the moment force or lever arm with any lateral force or cantilever. Esthetically, the prosthesis is less likely to replace the sole anatomical crowns of natural teeth when a greater CHS is present. The absence of a peri-implant ligament means that the bone-implant stresses cannot be managed by increasing the implant height. Therefore, as the CHS increases, a greater number of implants or wider implants should be inserted to counteract the increase in stress. For an ideal treatment plan, the CHS should be equal to or less than 15 mm for ideal conditions (see Chapter 6).

DIVISIONS OF AVAILABLE BONE

Division A (Abundant Bone)

Division A abundant bone often forms soon after the tooth is extracted. The abundant bone volume remains for a few years, although the interseptal bone height is reduced and the original crestal width is usually reduced by more than 30% within 2 years.[12] Division A bone corresponds to abundant available bone in all dimensions (Box 10-1 and Figures 10-9 to 10-11). It should be emphasized that the available bone height may be 20 mm for Division A, but this does not mean the implant length must be equal to the bone height. Because the stresses to the implant interface in good-density bone are captured at the crest of the ridge, an implant of 12 mm or more has been shown to

Box 10-1 Division A Dimensions

- Width > 6 mm
- Height > 12 mm
- Mesiodistal length > 7 mm
- Angulation of occlusal load (between occlusal plane and implant body) < 25 degrees
- Crown height space ≤ 15 mm

WWW.HIGHDENT.IR

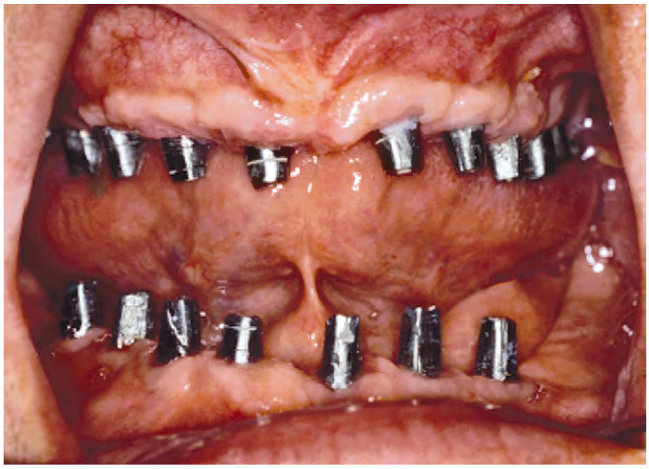


Figure 10-9 Eleven maxillary implants (4-mm diameter × 12 mm) and 7 mandibular implants (4 mm × 12 mm) are inserted into Division A available bone.

Figure 10-10 Both Division A arches were restored with fixed maxillary and mandibular implant restorations crown with heights inferior to 15 mm. In the FP-3 restoration, pink porcelain was used to replace the soft tissue drape.

be without compromise for long-term success, even though the implant does not engage the opposing cortical plate.

The Division A width of more than 6 mm is predicated on an implant diameter of at least 4 mm at the crest module, because abundant long-term data have been published regarding this implant size.[37,45] In abundant bone width (A+ bone) of greater than 7 mm, a wider (5-mm diameter) implant may be inserted, provided that 1 mm of bone remains around the buccal and lingual aspects of the implant. Osteoplasty may often be performed to obtain additional bone width.

The implant choice in Division A bone is a root form of 4 mm or greater in diameter. A larger diameter implant is suggested in the molar regions (5 to 6 mm in diameter). The length of the implant is 12 mm or greater. Longer implants are suggested in immediate-loading treatment options.

Division A bone should not be treated with smaller diameter implants for the final prosthesis. There are several advantages to the use of implants equal to or

Figure 10-11 A panoramic radiograph of the fixed maxillary and mandibular implant–supported restorations in Division A bone. The number of implants is related to the force conditions and bone quality of the patient.

greater than 4 mm in diameter, compared with smaller diameter implants (Box 10-2).

A patient with Division A bone should be notified that this is the most ideal time to restore their edentulous condition with implants. All too often, the doctor fails to educate the patient about the rapid decrease in bone volume width and the consequences of delaying treatment. When the bone volume is Division A, there is a decrease in treatment costs, with a reduction in the number and complexity of surgeries to the edentulous area with significant benefits to the patient. Unfortunately, these patients may not have significant problems with their existing restoration and therefore may not be motivated to address the situation. As the bone resorbs and the problems arise, a greater appreciation for the benefits of implant-supported restorations are realized. Just as the restoring dentist explains the need to replace a single tooth before tipping and extrusion of adjacent teeth and the risk of additional tooth loss, the patient should be educated as to the benefit of implant treatment while the area presents abundant bone.

The prosthetic options for Division A span the full gamut. An FP-1 restoration requires a Division A ridge. However, an FP-2 prosthesis most often also requires a Division A bone. An FP-2 restoration is the most common posterior restoration supported by multiple adjacent implants in partially edentulous patients, because of either bone loss or osteoplasty before implant placement. An FP-3 prosthesis is most often the option selected in the anterior Division A bone when the maxillary smiling lip position is high or a mandibular low lip line during speech exposes regions beyond the natural anatomical crown position.

For removable implant overdentures in Division A bone, the final position of the tooth and superstructure bar must be evaluated before surgery. A limited CHS is more common in Division A bone, and a final RP-4 or RP-5 result may require osteoplasty before implant placement. Division A bone may represent a contraindication for high profile O-ring attachments or

WWW.HIGHDENT.IR

Box 10-2 Division A Root Form Implant Advantages

- The larger the diameter of an implant, the greater the surface area and the less stress distributed through the crestal bone region.
- The larger diameter implants are closer to the lateral cortical plates of bone, which have greater density and therefore increased strength, modulus of elasticity, and bone-implant contact percentages.
- The larger diameter implants are less likely to fracture, because the strength of the material is increased by a power of four related to the radius of the implant. (A 4-mm-diameter implant is 16 times stronger than a 2-mm-diameter implant.)
- The smaller diameter implants are often one-piece to decrease the risk of fracture. The one-piece implants require an immediate restoration, rather than a submerged or one-stage approach. As such, likely loading and micromovement may occur at the bone-implant interface, with an increased risk of crestal bone loss and/or implant failure.
- The emergence profile angle of the crown is related to the implant diameter. The larger diameter teeth can be most esthetically restored with a wider diameter implant.
- The larger the implant diameter, the less stress applied to the abutment screw, and therefore complications such as screw loosening or fracture are less likely.
- The larger diameter abutment provides greater cement retention for the final restoration crown.
- Oral hygiene procedures are more compromised around smaller diameter implants restored with greater emergence profile angles and over contoured restorations.
- The crest module and crestal portion of many two-piece, small-diameter implants are smooth metal to increase the inter body wall thickness, thus creating shear loads to the crestal bone and an increased risk of bone loss.
- Implant costs to the patient are related to implant number, not diameter. Therefore increases in implant numbers for smaller diameter implants increase the cost to the patient (and the doctor).
- Division A root form implants are designed for variable bone density and can provide the greatest range of prosthetic options.

superstructures placed several millimeters above the tissue for hygiene considerations, because of a compromised CHS to accommodate prosthetic components (Figures 10-12 to 10-14).

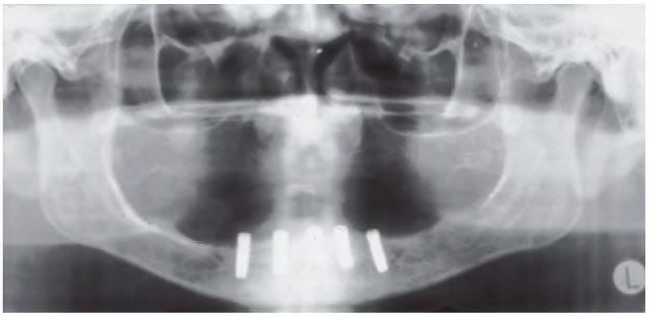


Figure 10-12 When planning implants in the anterior mandible for an overdenture, the following factors must be considered: crown height space for the denture teeth, acrylic, bar, and attachments. In Division A bone, the crestal bone may require osteoplasty to increase the crown height.

Figure 10-13 Intraoral view of an overdenture bar for an RP-4 restoration. The low-profile design is more common for Division A bone situations.

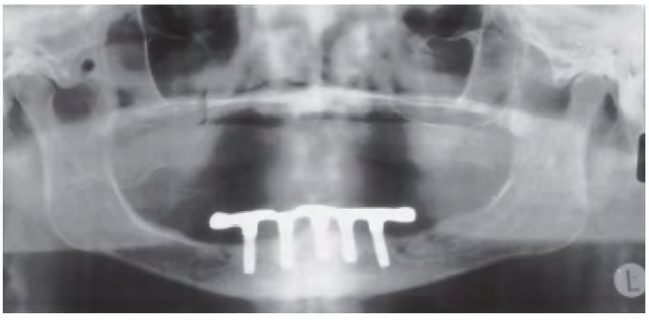


Figure 10-14 Panoramic radiograph of a Division A anterior mandible supporting an implant-retained and implant-supported overdenture (RP-4). The crown height is less than 15 mm.

Division B (Barely Sufficient Bone)

As the bone resorbs, the width of available bone first decreases at the expense of the facial cortical plate, because the cortical bone is thicker on the lingual aspect of the alveolar bone, especially in the anterior regions of the jaws. There is a 25% decrease in bone width the first year and a 40% decrease in bone width within the first 1 to 3 years after tooth extraction.[12,15,16] The resulting narrower ridge is often inadequate for many 4-mm-diameter root form implants. Slight to mild atrophy is often used to describe this clinical condition. After this Division B bone volume is reached, it may remain for more than 15 years in the anterior mandible.[5] However, the posterior mandibular height resorbs four times faster than the anterior region. The posterior maxillary regions exhibit less available bone height (as a consequence of sinus expansion) and have the fastest decrease

WWW.HIGHDENT.IR

Box 10-3 Division B Dimensions

- 2.5 to 6 mm wide
  - B+: 4 to 6 mm
  - B–w: 2.5 to 4 mm
- Height > 12 mm
- Mesiodistal length > 6 mm
- Angulation < 20 degrees
- Crown height space < 15 mm

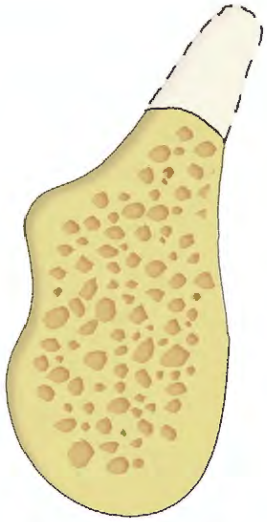


Figure 10-15 A Division B ridge may be converted to a Division A bone by osteoplasty.

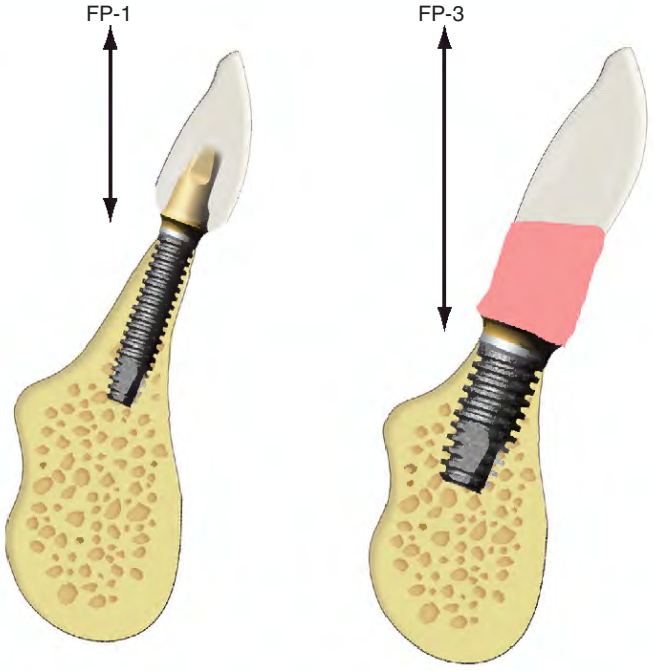


Figure 10-16 Options to treat a Division B ridge in the anterior mandible include a narrow implant with a final prosthesis closer to anatomical dimensions (FP-1) *(left)* or osteoplasty with Division A root forms and extended crown heights (FP-2 or FP-3) *(right)*.

of bone height of any intraoral region. As a result, the posterior regions of the jaws may become inadequate in height (C–h) earlier than the anterior regions.

Division B bone offers sufficient available bone height (Box 10-3). The Division B available bone width may be further classified into ridges 4 to 6 mm wide and B minus width (B–w) 2.5 to 4 mm wide, where bone grafting techniques are indicated (see Figure 10-4). Because the minimum mesiodistal length of a Division B ridge is less than that of Division A, a smaller diameter implant (i.e., 3 mm) may be used. Because the ridge width and implant diameter are narrower and forces increase as the angle of load increases, the angulation of occlusal load is also less. A CHS of 15 mm or less (similar to Division A) is necessary in Division B to decrease the moment of forces with lateral or offset loads, especially because of the smaller width dimension.

Three treatment options are available for the Division B edentulous ridge:

1. Modify the existing Division B ridge to another division by osteoplasty to permit the placement of root form implants 4 mm or greater in width (Figure 10-15). When more than 12 mm of bone height results, the bone converts to Division A. When less than 12 mm of bone height results, the bone converts to Division C–h.
2. Insert a narrow Division B root form implant.
3. Modify the existing Division B bone into Division A by augmentation.

To select the proper approach to this bone category, the final prosthesis must first be considered. When a Division B ridge is changed to a Division A by osteoplasty procedures, the final prosthesis design has to compensate for the increased CHS. For example, before surgery the available bone height may be compatible with an FP-1 prosthetic design. If, at the time of surgery, the ridge is found deficient in width for implant placement, it is not unusual to remove 3 mm of crestal bone before reaching a Division A width. This means the final restoration will require an additional 3 mm in height. Therefore it may result in an extended tooth (FP-2, FP-3) restoration (Figure 10-16). The osteoplasty option is less likely the treatment of choice for an FP-1 prosthesis with a B–w ridge, because even greater bone height reduction is required. The most common approach is to modify the narrower Division B ridge into another bone division by osteoplasty when the final restoration is an implant overdenture (Figures 10-17 to 10-19). The edentulous ridge crest may be reduced, thereby increasing the width of the ridge. If the CHS is less than 15 mm, the ridge division becomes Division A with a greater width than 6 mm. If the ridge height is reduced so that the crown height space is greater than 15 mm, the bone division is

WWW.HIGHDENT.IR

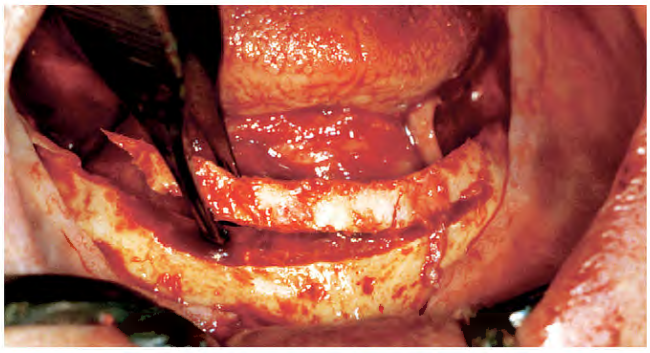


Figure 10-17 A Division B bone ridge may be modified by osteoplasty to increase the width of bone. The osteoplasty increases the crown height space for the prosthesis.

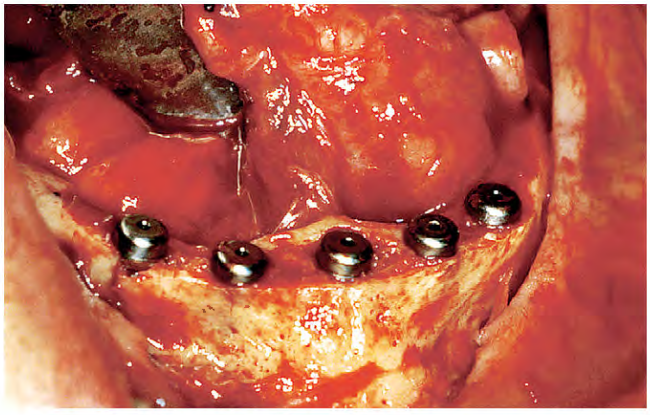


Figure 10-18 Five 4-mm-diameter anterior implants were placed after the osteoplasty in Figure 10-17. These implants were positioned with regard to the final restoration.

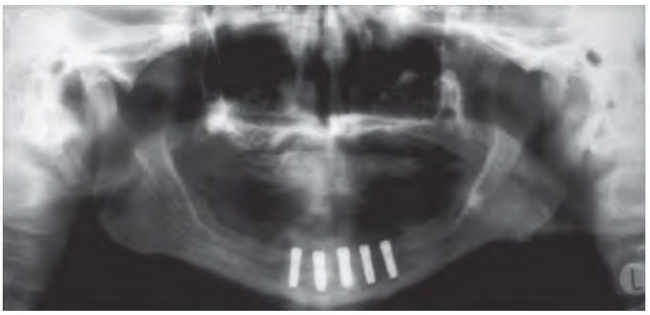


Figure 10-19 A panoramic radiograph of the Division B ridge modified to a Division A by osteoplasty and five root form implants between the mental foraminae.

Box 10-4 Disadvantages of Division B Root Forms

1. Almost twice the stress is concentrated at the top crestal region around the implant.
2. Less overall surface areas means that lateral loads on the implant result in almost three times greater stress than Division A root forms.
3. Fatigue fractures of the abutment post are increased, especially under lateral loads.
4. The crown emergence profile is less esthetic (except for maxillary lateral or mandibular incisors).
5. Conditions for daily care are compromised around the cervical aspect of the crown.
6. The implant design is often poor in the crestal region. To increase implant body wall thickness to reduce fracture, no threads or compressive force design are present, but this further increases stress and the amount of shear loads to bone.
7. The angle of load must be reduced to less than 20 degrees to compensate for the small diameter.
8. Two implants are often required for proper prosthetic support, unless anterior single tooth replacement for maxillary laterals or mandibular incisors, so surface area ends up being greater because of implant number, *not* size.
9. Implant costs are not related to diameter, so an increase in implant number results in greater cost to the doctor and patient.

not changed to a Division A. Instead, it has been altered to a Division C–h bone volume and, when cantilevers or lateral forces are present on the prothesis, is not as predictable for endosteal implant placement as Division A. An RP-4 or RP-5 restoration most often requires option 1—osteoplasty—where adequate CHS is created to permit the fabrication of the overdenture and superstructure bar with attachments without prosthetic compromise.

The second main treatment option for the narrow available bone Division B is the small-diameter root form implant. Smaller diameter root form implants (3.0 to 3.5 mm) are designed primarily for Division B available bone. The Division B bone is narrower, so the implant body of the implant must bisect the bone and implant angulation is less flexible. The Division B root form implants present several inherent disadvantages when compared with the larger diameter implants (Box 10-4).[56-61] As a result of these concerns for the Division B root form, this option is mostly used for single tooth replacement of a maxillary lateral incisor or mandibular incisors where the restricted available bone is in mesiodistal width.[62,63]

The third alternative treatment for Division B bone is to change the Division B ridge into a Division A by grafting the edentulous ridge with autogenous or a combination of allograft and alloplast with or without guided bone regeneration techniques (Figure 10-20). If this graft is intended for implant placement, a healing period of at least 4 to 6 months is needed for maturation of the graft and before endosteal implants should be placed. An FP-1 restoration most often mandates option 3: augmentation. The emergence profile angle of the final crown, which does not compromise hygiene, requires

WWW.HIGHDENT.IR

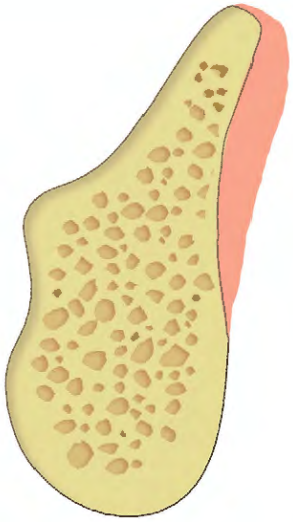


Figure 10-20 A Division B ridge anatomy may be modified to Division A by augmentation procedures.

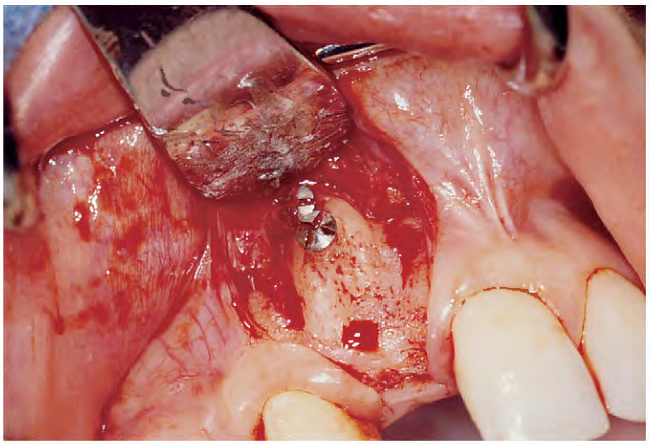


Figure 10-21 A reentry of an onlay ramus bone graft to a Divison B–w ridge. The ridge is now converted to Division A.

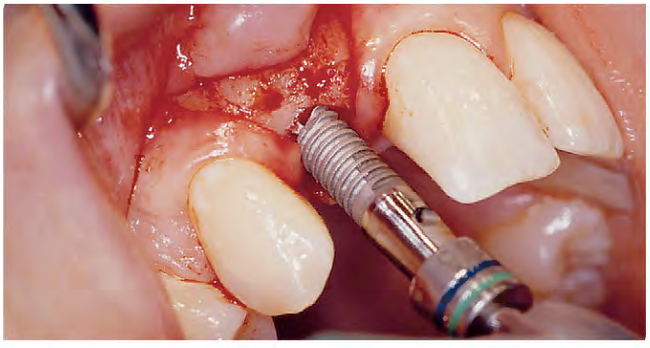


Figure 10-22 A root form implant may now be inserted without compromise to implant position or existing width of bone.

a Division A root form implant with the exception of maxillary lateral incisors or mandibular incisors. Stress factors may also dictate the surgical approach to Division B bone. In the presence of unfavorable stress factors, the number and width of abutments should be increased without increasing the CHS to provide a greater surface area of resistance to the magnified forces. To accomplish this goal, augmentation is indicated in Division B bone.

The success of alloplastic materials for augmentation correlates with the number of osseous walls in contact with the graft material.[64] Therefore a five-wall bony defect as a tooth socket forms bone more predictable with an alloplast than a one-wall defect as an onlay graft (see Chapter 37).

The distinction between B and B–w is especially important when augmentation is the method of choice. Bone augmentation is more predictable when the volume to augment is minimal and is for width, and least predictable when height is desired. For example, a width increase of 1 to 2 mm may be obtained with an alloplast and a guided bone regeneration, whereas more than 2 mm of width is more predictable with a autologous block graft. Some regions of the mouth are better suited than others for height augmentation (e.g., the floor of the maxillary sinus versus the posterior mandible).

An alternative for the augmentation approach for Division B bone is bone spreading. A narrow ostotomy may be made between the bony plates and bone spreaders are tapped into the edentulous site. The Division B ridge may be expanded to a Division A with this technique and allow a Division A implant or an alloplast to be inserted.[65]

The Division B–w ridge requires more than 2 mm of width increase, and therefore autologous bone is beneficial to predictably grow bone width. If the Division B–w ridge contour should be altered for improved prosthodontic relationships, an onlay particulate or block graft of autogenous bone is indicated. The autograft may be harvested from an intraoral region (such as the symphysis or ramus) and placed along the lateral aspect of the ridge that corresponds to ideal arch form (Figures 10-21 and 10-22). The implant placement should be delayed until after the augmentation process to permit ideal implant placement and to ensure complete bone formation before placing the implant. Division A root form implants may be placed 4 to 6 months after the autologous bone graft.[64-66]

The patient delaying treatment with a Division B bone situation should be notified of the future bone volume resorption. The augmentation of bone in height is much less predictable and requires more advanced techniques than the augmentation of width alone. For example, the patient may not be experiencing problems with a maxillary denture, but the Division B bone will resorb in height and decrease the stability and retention of the removable soft tissue–supported prosthesis. When

WWW.HIGHDENT.IR

treatment is delayed until patient problems begin, the overall result may be more difficult to achieve and more costly to the patient.

The final prosthesis type for Division B ridges is dependent on the surgical option selected. Grafted ridges will more often be used when a fixed prosthesis is desired, whereas ridges treated with osteoplasty before implant placement are likely to be supporting removable prostheses. The treatment option may be influenced by the region to be restored. For example, in the partially edentulous anterior maxilla, augmentation is most often selected because of esthetics. In the edentulous anterior mandible, osteoplasty is common. In the premolar region of the posterior mandible, Division B root form implants are often used because the bone density is adequate, available bone height is limited, and esthetics are not a major factor.

Division C (Compromised Bone)

The Division C ridge is deficient in one or more dimensions (width, length, height, or angulation) (Box 10-5) regardless of the position of the implant body into the edentulous site.

The resorption pattern occurs first in width and then in height. As a result, the Division B ridge continues to resorb in width, although height of bone is still present, until it becomes inadequate for any design of endosteal implant. This bone category is called Division C minus width (C–w) (Figure 10-23). The resorption process continues, and the available bone is then reduced in height (C–h). Moderate to advanced atrophy may be used to describe the clinical conditions of Division C. The posterior maxilla or mandible result with Division C–h more rapidly than the anterior regions because the maxillary sinus or mandibular canal limit vertical height sooner than the opposing cortical plates in the anterior regions. When the anterior mandible is C–h, the floor of the mouth is often level with the residual mandibular crest of the ridge. During swallowing, it may prolapse over the residual crest and implant sites, causing constant irritation of the permucosal implant posts and impairing proper design of the prosthetic superstructures.

The doctor must appreciate that the C–w bone will resorb to a C–h ridge as fast as the A resorbs to B and faster than B resorbs to C–w. In addition, without implant or bone graft intervention, the C–h available bone will eventually evolve into Division D (severe atrophy). Many completely edentulous patients are treated with implants in the mandible and conventional dentures in the maxilla, primarily because the mandibular C–h arch is more often the cause of patient complaint (Figure 10-24). However, the patient should be educated about the future maxillary bone loss that will render maxillary implant treatment almost impossible without advanced bone graft procedures before placement.

The Division C edentulous ridge does not offer as many elements for predictable endosteal implant survival or prosthodontic management compared with Divisions A or B. Anatomical landmarks to determine implant angulations or positions in relation to the incisal edge are usually not present; therefore greater surgical skill is required. The doctor and patient should realize that Division C ridge implant-supported prostheses are more complex and have slightly more complications in healing, prosthetic design, or long-term maintenance. On the other hand, the patients usually have greater need for increased prosthodontic support. In spite of the reduced bone volume, altered treatment plans that decrease stress can provide predictable, long-term treatment.

Box 10-5 Division C Bone

- Width (C–w bone): 0 to 2.5 mm
- Height (C–h bone) < 12 mm
- Angulation of occlusal load (C–a bone) > 30 degrees
- Crown height space (CHS) > 15 mm

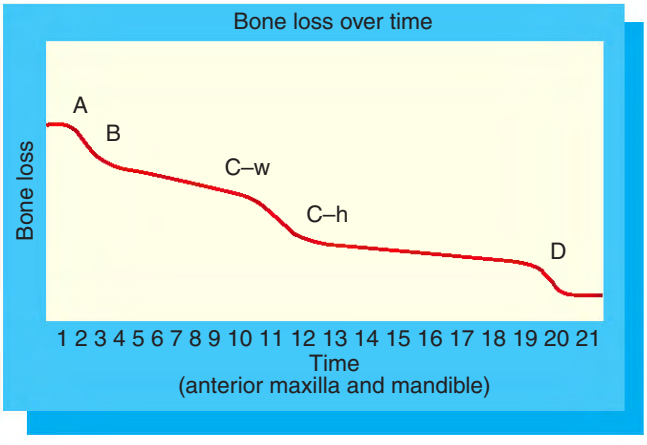


Figure 10-23 Bone resorbs from Division A to Division B and from Division C–w to C–h rapidly. Long plateaus are found for Division B and Division C–h.

Figure 10-24 Panoramic radiograph of a Division C–h anterior mandible with five root forms opposing a maxillary denture. The patient should be informed of the continued maxillary and posterior mandibular bone loss over time.

WWW.HIGHDENT.IR

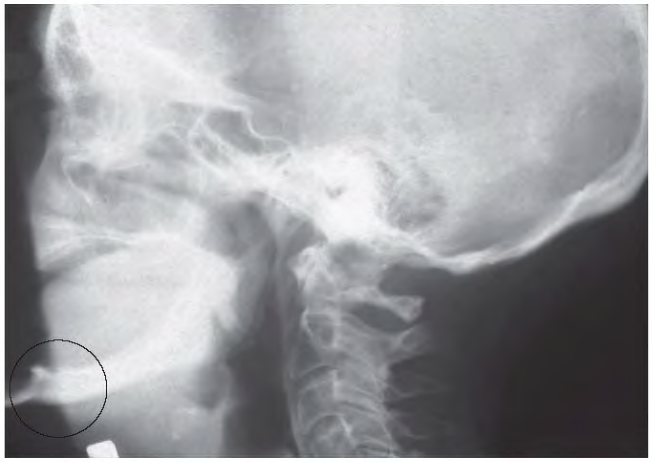


Figure 10-25 Lateral cephalogram of an anterior mandible with a 45-degree trajectory to the occlusal plane.

Figure 10-26 A C–a anterior mandible with four root form implants. The implants encroach on the floor of the mouth, complicate prosthesis fabrication, and impair speech and comfort during function.

There is one uncommon subcategory of Division C, namely C–a. In this category, available bone is adequate in height and width, but angulation is greater than 30 degrees regardless of implant placement (Figure 10-25). When present, this condition is most often found in the anterior mandible; other less-observed regions include the maxilla with severe facial undercut regions or the mandibular second molar with a severe lingual undercut. Root form implants placed in this bone category may be positioned within the floor of the mouth and compromise prosthetic reconstruction, speech, and comfort (Figure 10-26).

Implant treatment planning for the completely edentulous C–h arch is more complex than in Division A or B. There are seven implant treatment options for Division C bone (Box 10-6); all these options require greater skill than similar treatment modalities in Division A or B.

A C–w ridge may be treated by osteoplasty. An osteoplasty converts the ridge to C–h and, in the anterior mandibular region, most often to a width suitable for root form implants. The most common available bone division after osteoplasty of C–w is C–h available bone, not Division A, because the CHS is larger than 15 mm. On occasion, the C–w osteoplasty may convert the ridge to Division D, especially in the posterior mandible or maxilla. Care should be taken not to let this occur, because bone grafting procedures will be more challenging after the height has been reduced.

Another treatment option is to alter the Division C by grafting. After the ridge is augmented, it is treated with the options available in the acquired bone division. The patient who desires a fixed prosthesis often requires an autogenous graft prior to implant placement to acquire proper lip support and ideal crown height.

Augmentation of C–w is most often used when prosthetic guidelines require a fixed restoration or excess force factors require greater surface area implants and improved biomechanics for the prosthesis. The

Box 10-6 Division C Treatment Options

- Osteoplasty (C–w)
- Root form implants (C–h)
- Subperiosteal implant (C–h, C–a partial, or completely edentulous mandible)
- Augmentation procedures before implant insertion
- Disk design implants (posterior mandible, anterior maxilla)
- Ramus frame implant (C–h completely edentulous mandible)
- Transosteal implant (C–h anterior mandible)

C–w augmentation is more difficult than for Division B bone, because the need for bone volume is greater, yet the recipient bed is more deficient. Therefore block bone grafts are usually indicated.[66-68] Soft tissue complications, such as incision line opening, are also more common in C–w augmentations compared with Division B.

The C–h posterior maxilla is a common and unique edentulous condition. The residual ridge resorbs in width and height after tooth loss, similar to other regions. However, because of the initial ridge width dimension, a decrease of 60% in dimension still is adequate for 4-mm-diameter implants. In addition to the residual alveolar bone resorption, the maxillary sinus expands after tooth loss. As a result, the available bone height is decreased from both the crestal and apical regions. Sinus grafts, which elevate the maxillary sinus floor membrane and then graft the previous sinus floor region, were developed by Tatum in the mid-1970s.[65] This area is the most predictable intraoral region to augment in excess of 10 mm of vertical bone. Even alloplasts may be used for this technique. Therefore sinus grafting is often prescribed before placing endosteal implants in the C–h posterior maxilla (Figures 10-27 to 10-32).

Various implant approaches are used in the Division C–h available bone. Shorter endosteal implants are the

WWW.HIGHDENT.IR

Figure 10-27 A panoramic radiograph with a unilateral posterior maxilla missing a premolar and molars. The bone division is C–h in the premolar and Division D in the molar region.

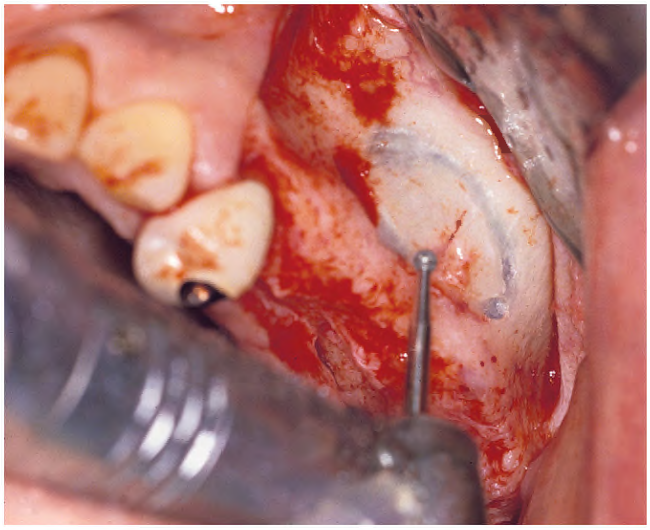


Figure 10-28 A Tatum lateral wall approach is prepared with a round diamond. The sinus mucosa will be elevated and a sinus graft will be placed to augment the bone height.

Figure 10-29 Postoperative radiograph of Figure 10-28. The posterior maxilla has been modified to Division A height requirement.

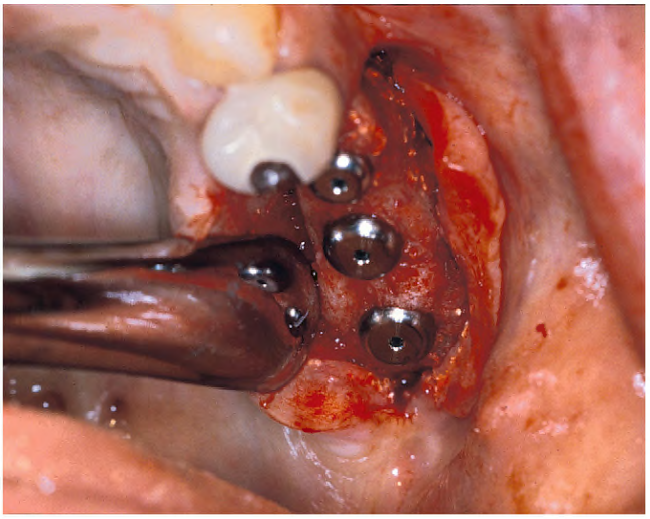


Figure 10-30 After the modification shown in Figure 10-29, three implants are inserted at reentry into the posterior maxilla. The second premolar implant is 4 mm in width; the molar sites are 5-mm-wide implants.

Figure 10-31 The three-unit fixed final restoration (FP-1) is cemented into position over the three implants.

most common options.[54,55] A C–h root form implant is usually 4 mm or greater in width at the crest module and 10 mm or less in height. Several studies indicate implant survival is decreased once an implant is 10 mm or less in height.[37,69,70] For example, a large multicenter study of 31 different sites and 6 different implant designs observed 13% failure with 10-mm implants, 18% failure with 8-mm implants, and 25% failure with 7-mm-long implants.[37] The implant failure does not occur after surgery, but rather after prosthetic delivery. This loading failure is a result of inadequate implant support combined with a magnification of force resulting from excessive CHS.

When endosteal root form implants are used in Division C–h bone with greater crown heights, additional implants should be placed to increase the overall

WWW.HIGHDENT.IR

Figure 10-32 Final panoramic radiograph of Figure 10-31 with the maxillary quadrant with three implants supporting a fixed prosthesis. The opposing mandibular plane of occlusion was corrected with two posterior crowns.

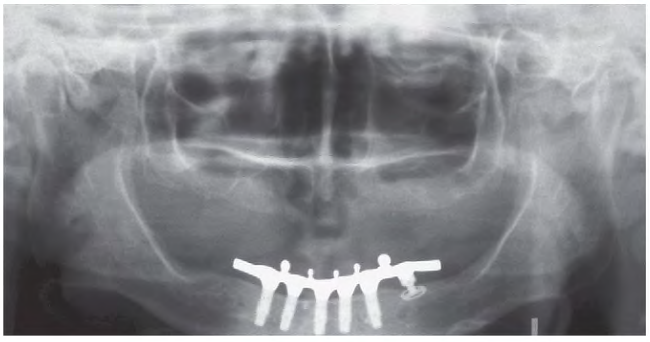


Figure 10-33 Panoramic radiograph of a posterior C–h mandible with a disc implant in place, connected to five anterior root form implants.

Figure 10-34 The five root forms and disc implant support a bar for a mandibular RP-4 overdenture opposing a conventional maxillary denture.

implant-bone surface area, and the prosthesis should load the implants in an axial direction. Because the CHS is greater than 15 mm, the design of a removable prosthesis should often reduce cantilever length and incorporate a stress relief mechanism. Reduced long-term predictability is usually expected if additional implants or less stressful prostheses are not used, because a greater moment force is transmitted to the implants.

Alternative designs to endosteal implants in the posterior mandibular edentulous Division C–h arch are subperiosteal and disk design implants (Figures 10-33 and 10-34).[71-73] Subperiosteal implants are more predictable in the mandibular arch than in the maxilla. The limitations of anatomy for root form implants may be bone angulation, a square arch form, or inadequate bone height. When the anterior bone angulation is unfavorable, root form implants may be positioned too far lingually for prosthodontic support, speech, or hygiene. The superstructure and abutment posts for the subperiosteal implant are designed and cast before implant placement. The permucosal posts may be designed with greater latitude than endosteal implants. When anterior root forms are placed in an edentulous mandible with a square arch form, the superstructure may not be cantilevered distally because of the poor anteroposterior distance. As a result, a fixed restoration or RP-4 overdenture prosthesis is contraindicated with anterior root forms in a square arch form. A subperiosteal implant may provide anterior and posterior bone support, and the square arch form does not contraindicate an RP-4 prosthesis. Autogenous grafts or nerve repositioning may be necessary to place endosteal implants in the posterior Division C–h mandible. The increase in treatment time, surgical risks and postoperative complications (such as paresthesia) are to be thoroughly discussed with the patient. Circumferential or unilateral subperiosteal implants permit the placement of posterior prosthodontic units without risks of paresthesia from nerve repositioning or lengthened treatment time associated with autogenous bone grafts and endosteal implants.[73]

Another alternative for the posterior mandible or premaxilla with Division C–h bone is a disk design implant that engages the lateral aspect of the cortical bone and may be used in available bone height of 3 mm or more. As a general rule, these implants are used in addition to other root form implants. Their inclusion in the treatment plan for C–h posterior sections of edentulous mandibles eliminate cantilevers in full-arch restorations.[71,74]

The prosthetic options for Division C ridges more often consist of removable prosthesis in the completely edentulous maxillary arch. A maxillary overdenture in a Division C ridge supports the upper lip without hygiene compromise. In the Division C mandible, the greater CHS often mandate an overdenture design with some soft tissue support (RP-5). A fixed restoration in the Division C mandible often requires both anterior and posterior implant support. The fixed prosthesis in Division C bone with greater than 15-mm CHS is most often a hybrid device, with denture teeth attached to a precious metal substructure with acrylic resin. In this way, the complications and costs of a porcelain-metal fixed restoration may be reduced.

WWW.HIGHDENT.IR

In general, Division C–h presents less favorable biomechanical factors to the implant support. Therefore, additional implants or teeth, cross-arch stabilization, soft tissue support, or an opposing removable prosthesis often need to be considered in the prosthetic design to improve the long-term prognosis. Treating the Division C ridge requires greater experience, caution, and training than does the previous two bone divisions; however, excellent results may be achieved.

The completely edentulous patient who does not have implant treatment should be well-educated that the bone resorption process will continue, with significant increased risks for the convention removable restoration. Waiting to treat the patient until irreparable problems develop is a poor treatment alternative that results in the need for more advanced procedures such as iliac crest grafts and significant risks of associated complications.

An alternative method of treatment for the maxilla is to fabricate a traditional denture in Division C arches after changing the division with nonresorbable hydroxyapatite. This treatment option is often indicated for a conventional maxillary denture on a C–w anterior maxilla. The patient and doctor should realize the augmented ridge is only a delay tactic for bone resorption, because it does not stimulate or maintain bone mass. Its primary intent is to provide additional retention and stability and delay the problems of the resorption leading to a Division C–h ridge. Bone augmentation for Division C–h bone to support complete dentures is rarely beneficial. Even more rarely should nonresorbable augmentation materials be used for additional support for a mandibular denture or for endosteal implant support.

In conclusion, as in all other bone divisions, the final prosthesis determines the treatment option. For mandibular RP-4 restorations, five root forms in the anterior mandible may be used (if the other dental criteria permit). However, the greater CHS or a square arch form may mandate an RP-5 prosthesis with anterior root form implants. The combination of anterior root forms and posterior subperiosteal implants (or disk implants) is a common treatment option for RP-4 or fixed prosthesis in the mandibular arch. Augmentation is often required for a fixed prosthesis in either of the Division C complete edentulous arches if stress factors are high and cannot be reduced.

Division D (Deficient Bone)

Long-term bone resorption may result in the complete loss of the residual ridge, accompanied by basal bone atrophy (Figure 10-35). Severe atrophy describes the clinical condition of the Division D ridge. At one time, it was believed only the alveolar process would resorb after tooth loss and the basal bone would remain. However, bone loss may continue beyond the previous roots of teeth and even include the bone over the inferior mandibular nerve or the nasal spine of the maxilla. Basal bone loss eventually results in a completely flat maxilla. In the mandible, the superior genial tubercles become the most superior aspect of the ridge. The mentalis muscle has lost much of its attachment, even though the superior portion of the muscle attaches near the crest of the resorbed ridge. The buccinator muscle may approach the mylohyoid muscle and form an aponeurosis above the body of the mandible. The mandibular arch also presents with mental foraminae and portions of the mandibular canal dehiscent. Therefore it is not infrequent that these patients complain of paresthesia of the lower lip, especially during mastication. The CHS is greater than 20 mm, which is a significant force multiplier and can rarely be reduced enough to render long-term success (Figure 10-36; Box 10-7).

The prosthetic result for Division D without augmentation is the poorest treatment outcome of all the divisions of bone. Fixed restorations are nearly always contraindicated, because the CHS is so significant.

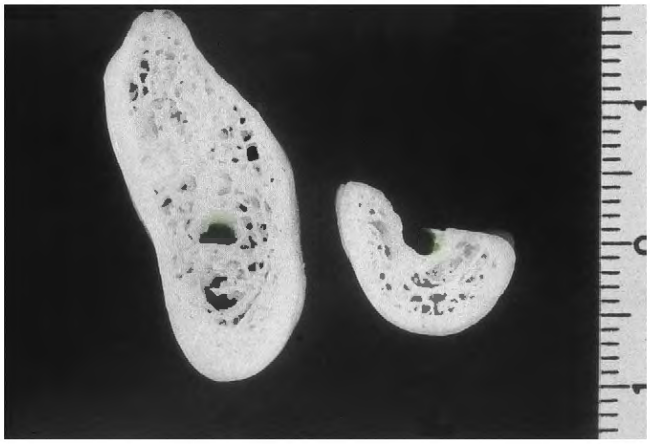


Figure 10-35 The left posterior mandible is Division A, with abundant bone in height and width *(left)*. The residual ridge on the right is a Division D with a dehiscent mandibular canal.

Figure 10-36 A panoramic radiograph of a patient with a Division D mandible wearing a complete denture.

WWW.HIGHDENT.IR

Box 10-7 Division D Bone

- Severe atrophy
  - Basal bone loss
    - Flat maxilla
    - Pencil-thin mandible
- >20 mm crown height

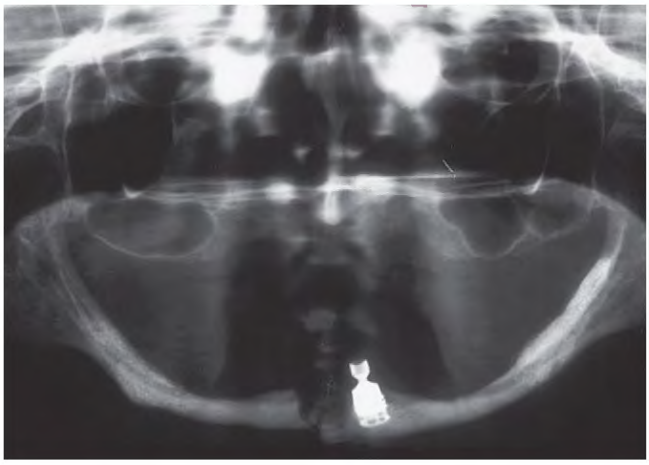


Figure 10-37 Anterior root form implants were placed in a Division D mandible. As a result of the failure of one implant, the mandible has fractured and has a continuity defect.

Completely implant-supported overdentures are indicated whenever possible to decrease the soft tissue and nerve complications, but require anterior and posterior implant support, which almost always require bone augmentation before implant placement. Bone augmentation for Division D is difficult to improve the CHS enough to warrant a fixed restoration. An RP-5 restoration is not suggested, because bone loss will continue in the soft tissue–supported region of the overdenture.

The completely edentulous Division D patient is the most difficult to treat in implant dentistry. Benefits must be carefully weighed against the risks. Although the practitioner and patient often regard this condition as the most desperate, these patients do not usually have oral antral fistulae or deviated facial features before treatment. If implant failure occurs, the patient may become a dental cripple—unable to wear any prosthesis (Figure 10-37).

Autogenous iliac crest bone grafts to improve the Division D are strongly recommended before any implant treatment is attempted.[74] After autogenous grafts are in place and allowed to heal for 5 or more months, the bone division is usually Division C–h or A and endosteal implants may be inserted (Figures 10-38 to 10-46).

The autogenous bone grafts are not intended for improved denture support. If soft tissue–borne prostheses are fabricated on autogenous grafts, 90% of the grafted bone resorbs within 5 years as a result of accelerated

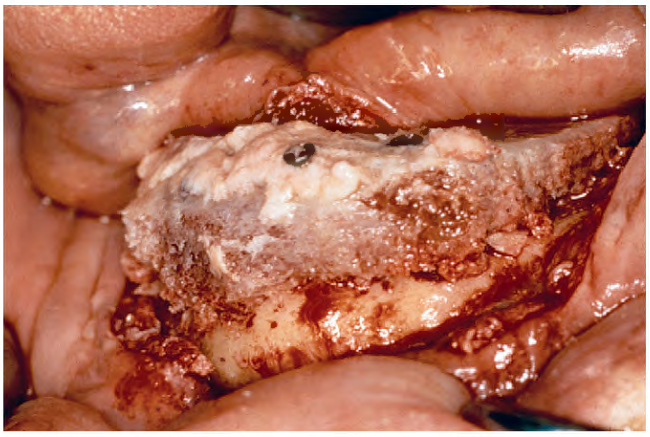


Figure 10-38 An autologous iliac crest bone graft in situ. The block graft is fixated with screws between the foramina and projects over the posterior canals.

Figure 10-39 Lateral cephalogram of the block graft in Figure 10-38. The residual anterior mandible was angled more than 30 degrees lingually, so the bone graft is contoured facially toward the incisal edge of the final restoration.

resorption.[75] Additional augmentation to compensate for this resorption is not indicated. Repeated relines, highly mobile tissue, sore spots, and patient frustration are all consequences. On the other hand, autogenous bone grafts are maintained long term in conjunction with implant placement. The completely flat Division D maxilla should not be augmented with only hydroxyapatite to improve denture support. Inadequate ridge form exists to guide the placement of the material. As a result, migration of the graft at the time of surgery or in the future after soft tissue loading is a frequent sequel.

The partially or completely edentulous patient with a posterior Division D maxilla and healthy anterior teeth or implants may undergo sinus graft procedures

WWW.HIGHDENT.IR

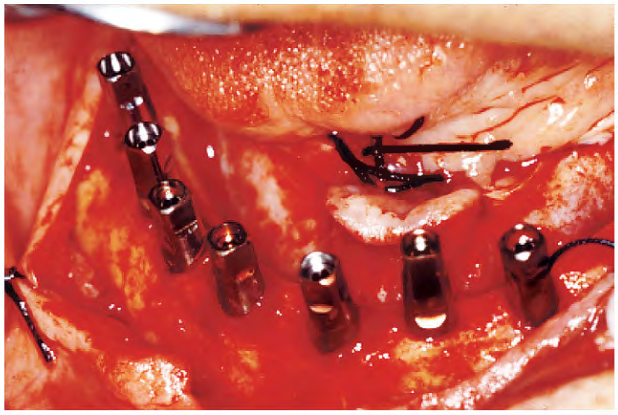


Figure 10-40 Reentry into the iliac crest block graft and placement of seven implants (4 mm in diameter and 12 mm long). The residual ridge was restored to Division A dimensions in the anterior and posterior regions.

Figure 10-41 Panoramic radiograph of the mandible restored with seven mandibular implants. The iliac crest bone graft has not remineralized completely, especially in the posterior regions where the blood supply was minimal in the premaxilla. A dense hydroxyapatite graft was performed to improve the contour for a maxillary denture and delay the continued resorption.

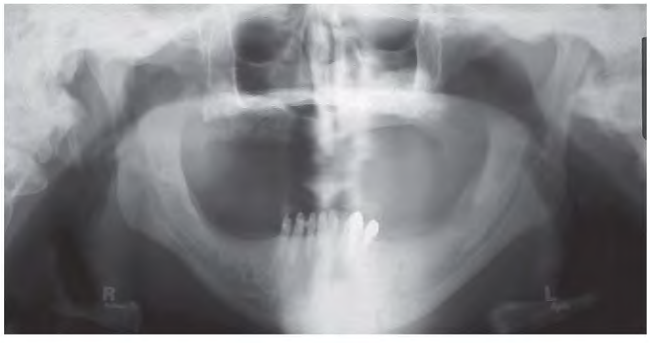


Figure 10-42 A panoramic radiograph of a Division D maxilla opposing unsalvageable mandibular teeth.

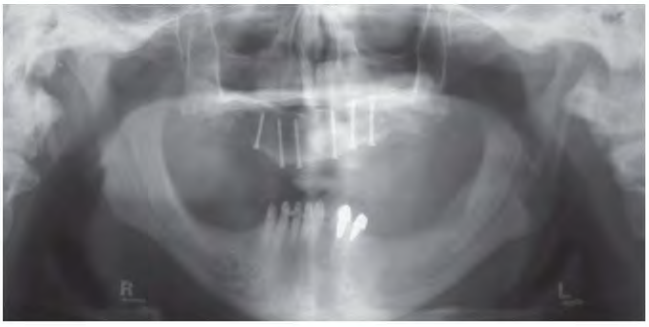


Figure 10-43 An iliac crest bone graft is fixated to the edentulous maxilla.

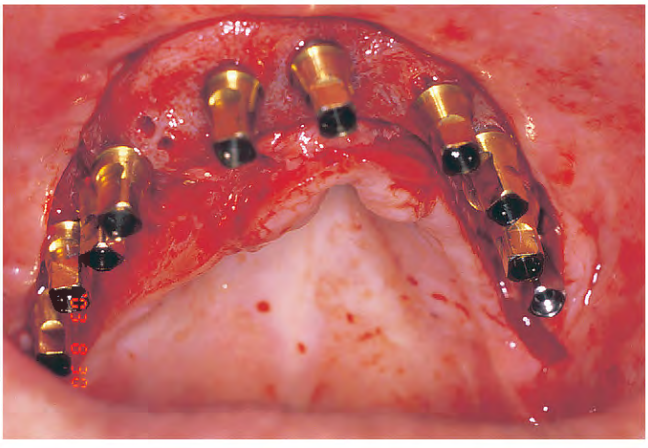


Figure 10-44 Ten endosteal implants are inserted into the illiac crest bone graft 6 months later.

Figure 10-45 A fixed maxillary and mandibular implant prosthesis are fabricated after healing.

with a combination of local autogenous bone, demineralized freeze-dried bone, and calcium phosphate bone substitutes.[65] The CHS may be insufficient for onlay grafts in the posterior maxilla, despite a lack of available bone height, because the sinus expands faster than the crest of the ridge resorbs. Endosteal implants of adequate height can rarely be positioned without a sinus graft. After 6 to 8 months after sinus graft, the Division D posterior maxilla is restored to Division A or C–h, and root form implants may be inserted for posterior prosthodontic support.

Past beliefs regarding subperiosteal implants indicated their use on atrophied bone over any other type of implant support, and the less bone available, the more ideal the indication for a subperiosteal implant. On the contrary, adequate bone should also be present

WWW.HIGHDENT.IR

Figure 10-46 Panoramic radiograph of the final restoration and implants.

for this implant modality. The maxilla rarely provides sufficient support in the Division D ridge for implants of any design. If adequate mandibular anterior bone is present with Division D posterior bone, root form implants, tripodal subperiosteal implants, mandibular staple implants, or ramus frame implants may be used cautiously in the anterior edentulous mandible. However, idiopathic fracture during surgery or from implant failure or removal is a more likely complication than in other bone division. Therefore practitioners treating Division D mandibles should be able to manage future complications, which may be extensive.

Endosteal root form implants without autogenous grafts may be used on rare occasions in the anterior Division D mandible when the remaining bone is dense and the opposing arch is edentulous. Care must be taken during placement, because mandibular fracture at insertion or during postoperative healing is a possible complication.[76,77] Under these conditions, the crown height space is very great and the number of implants often four or fewer. Implant failure after loading is a greater risk. Implant failure results with circumferential bone loss, which may be associated with mandibular fracture through the implant site. An RP-5 removable restoration is usually indicated for Division D with only anterior implants. However, the RP-5 restoration allows continued bone resorption and atrophy to continue in the posterior regions. Therefore the prudent therapy is to educate the patient as to the risks of the situation and offer an autologous bone graft and implants to support an RP-4 restoration. The choice to render treatment is the doctor's, not the patient's. The implant support should not be compromised when implant failure may result in significantly greater risks.

The Division D arch requires greater doctor training and results in more frequent complications related to grafting, early implant failure, and soft tissue management; therefore treatment options include a more guarded prognosis. It should be the goal of every dentist to educate and treat the patient before a Division D bone condition develops. The profession treats periodontal diseases before pain in the region occurs, and carious lesions are removed before abscess formation. The profession monitors bone loss around teeth in fractions of a millimeter and offers continued care to reduce the risks of future tooth and bone loss. Likewise, the prudent practitioner monitors bone loss in edentulous sites and offers education and treatment before deleterious effects.

SUMMARY

In implant dentistry, the prosthesis is designed at the onset of treatment to satisfy the patient's needs and desires and obtain optimal results. This may range from a completely fixed prosthesis to one with primarily soft tissue support. After the final prosthesis type has been established, the key implant positions, patient force factors, bone density in the implant sites, and implant number, size, and design are determined. The primary criterion for proper implant support is the amount of available bone. Four divisions of available bone, based on the width, height, length, angulation, and crown height space in the edentulous site, have been presented. Consistent implant treatment plan procedures elaborated for each category of bone may be followed.

The Division A edentulous ridge offers abundant bone in all dimensions. Division A root form implants are optimally used and most often as independent support for the prosthesis. Division B bone may provide adequate width for narrower, small-diameter root from endosteal implants. The decreased width and surface area usually require additional implants to be included in the final prosthesis design. Division B may be changed in condition to a Division A by augmentation or osteoplasty. The treatment options may be selected in light of the area to be treated. For example, in the anterior maxilla, augmentation is most often selected because of esthetics. In the anterior mandible, osteoplasty is common because of the available bone height and low esthetic concerns. In the posterior mandible, multiple Division B implants may be used, because the bone density is good, the available bone height is limited, and esthetics are not a primary factor. When stress factors are greater, bone augmentation precedes Division A root form implants, regardless of the anatomical location.

The Division C edentulous ridge exhibits moderate resorption and presents more limiting factors for predictable endosteal implants. The decision to restore with endosteal implants or to upgrade the bone division by augmentation before implant placement is influenced by the prosthesis, patient force factors, and patient's desires.

The Division D edentulous ridge corresponds to basal bone loss and severe atrophy, resulting in dehiscent mandibular canals or a completely flat maxilla. The patient often requires augmentation with

WWW.HIGHDENT.IR

autogenous bone before implant and prosthodontic reconstruction.

If the existing conditions do not qualify for a predictable end result, the patient's mind or mouth must be modified. For example, the expectations of the patient must be reduced so the prosthesis may be changed from FP-1 to RP-4, or the bone must be augmented to improve the height and width and change the division so that long-term implant support and prosthetic design will be compatible.

References

1. Misch CE, Judy KWM: Patient dental-medical implant evaluation form, *Int Cong Oral Implant*, 1987.
2. Greenfield EJ: Implantation of artificial crown and bridge abutments, *Dent Cosmos* 55:364-369, 1913.
3. Misch J: *Lehrbuch der Grenzgebiete der Medizin und Zahnheilkunde,* vol 1, ed 2, Leipzig, Germany, 1922, FCW Vogal.
4. Atwood DA: Postextraction changes in the adult mandible as illustrated by microradiographs of midsagittal sections and serial cephalometric roentgenograms, *J Prosthet Dent* 13:810-824, 1963.
5. Atwood DA: Reduction of residual ridges: a major oral disease entity, *J Prosthet Dent* 26:266-279, 1971.
6. Atwood DA, Coy WA: Clinical, cephalometric and densitometric study of reduction of residual ridges, *J Prosthet Dent* 26:280-295, 1971.
7. Tallgren A: The continuing reduction of the residual alveolar ridges in complete denture wearers. A mixed longitudinal study covering 25 years, *J Prosthet Dent* 27:120-132, 1972.
8. Atwood DA: Some clinical factors related to the rate of resorption of residual ridges, *J Prosthet Dent* 12:441-450, 1962.
9. Karkazis HC, Lambadakis J, Tsichlakis K: Cephalometric evaluation of the changes in mandibular symphysis after 7 years of denture wearing, *Gerodontology* 14:10-15, 1997.
10. Karagaclioglu L, Ozkan P: Changes in mandibular ridge height in relation to aging and length of edentulism period, *Int J Prosthodont* 7:368-371, 1994.
11. Kovacic I, Celebic A, Knezovic Zlataric D et al: Influence of body mass index and the time of edentulousness on the residual alveolar ridge resorption in complete denture wearers, *Coll Antropol* Suppl 2:69-74, 2003.
12. Lam RV: Contour changes of the alveolar process following extraction, *J Prosthet Dent* 10:25-32, 1960.
13. Berg H, Carlsson GE, Helkimo M: Changes in shape of posterior parts of upper jaws after extraction of teeth and prosthetic treatment, *J Prosthet Dent* 34:262-268, 1975.
14. Gazabatt C, Parra N, Meissner C: A comparison of bone resorption following intraseptal alveolectomy and labial alveolectomy, *J Prosthet Dent* 15:435-443, 1965.
15. Pietrokovski J, Sorin S, Hirschfeld Z: The residual ridge in partially edentulous patients, *J Prosthet Dent* 36:150-157, 1976.
16. Pietrokovski J, Massler M: Alveolar ridge resorption following tooth extraction, *J Prosthet Dent* 17:21-27, 1967.
17. Pietrokowski J: The bony residual ridge in man, *J Prosthet Dent* 34:456-462, 1975.
18. Parkinson CF: Similarities in resorption patterns of maxillary and mandibular ridges, *J Prosthet Dent* 39:598-602, 1978.
19. Wical KE, Swoope CC: Studies of residual ridge resorption. Part I: use of panoramic radiographs for evaluation and classification of mandibular resorption, *J Prosthet Dent* 32:7-12, 1974.
20. Tallgren A, Lang BR, Miller RL: Longitudinal study of soft-tissue profile changes in patients receiving immediate complete dentures, *Int J Prosthodont* 4:9-16, 1991.
21. Weiss CM, Judy KWM: Severe mandibular atrophy: biological considerations of routine treatment with complete subperiosteal implants, *J Oral Implant* 4:431-469, 1974.
22. Kent JN: Correction of alveolar ridge deficiencies with non-resorbable hydroxyapatite, *J Am Dent Assoc* 105:99-100, 1982.
23. Lekholm U, Zarb G: Patient selection and preparation. In Brånemark PI, editor: *Tissue integrated prostheses: osseointegration in clinical dentistry,* Chicago, 1985, Quintessence.
24. Fallschüssel GKH: Untersuchungen zur Anatomie des zahnlosen Oberkiefers, *Z Zahnarztl Implantol* 2:64-72, 1986.
25. Gruber H, Solar P, Ulm C: Maxillomandibular anatomy and patterns of resorption during atrophy. In Watzek G, editor: *Endosseous implants: scientific and clinical aspects,* Chicago, 1996, Quintessence.
26. Cawood JJ, Howell RA: A classification of the edentulous jaws classes I to VI, *Int J Oral Maxillofac Surg* 17:232-279, 1988.
27. Misch CE: *Treatment planning and implant dentistry* [abstract], Dearborn, Mich, *Misch Implant Institute Manual,* 1985.
28. Misch CE, Judy KWM: Classification of partially edentulous arches for implant dentistry, *Int J Oral Implant* 4:7-12, 1987.
29. Misch CE: Available bone influences prosthodontic treatment, *Dent Today* 44-75, Feb 1988.
30. Misch CE: Bone classification, training keys to implant success, *Dent Today* 39-44, May 1989.
31. Misch CE: Classifications and treatment options of the completely edentulous arch in implant dentistry, *Dent Today* 26-30, Oct 1990.
32. Misch CE: Divisions of available bone in implant dentistry, *Int J Oral Implant* 7:9-17, 1990.
33. Misch CE: Classification de l'os disponible en implantologie [in French], *Implantodontie* 6/7:6-11, 1992.
34. Razavi R, Zena RB, Khan Z et al: Anatomic site evaluation of edentulous maxillae for dental implant placement, *J Prosthet Dent* 4:90-94, 1995.
35. Oikarinen K, Raustia AM, Hartikainen M: General and local contraindications for endosseal implants—an epidemiological panoramic radiographic study in 65 year old subjects, *Community Dent Oral Epidemiol* 23:114-118, 1995.
36. Brånemark PI: Osseointegration and its experimental background, *J Prosthet Dent* 50:399-410, 1983.
37. Minsk L, Polson A, Weisgold A et al: Outcome failures of endosseous implants from a clinical training center, *Compend Contin Educ Dent* 17:848-859, 1996.
38. Stultz RE, Lofland R, Sendax VI et al: A multicenter 5-year retrospective survival analysis of 6,200 integral implants, *Compend Contin Educ Dent* 14:478-486, 1993.
39. Saadoun A, LeGall MG: An 8-year compilation of clinical results obtained with Steri-Oss Endosseous implants, *Compend Contin Educ Dent* 17:669-688, 1996.
40. van Steenberghe D, DeMars G, Quirynen M et al: A prospective split mouth comparative study of two screw-

WWW.HIGHDENT.IR

shaped self-tapping pure titanium implant system, *Clin Oral Impl Res* 11:202-209, 2000.
41. Naert I, Koutsikakis G, Duyck J et al: Biologic outcome of implant-supported restorations in the treatment of partial edentulism, part I: a longitudinal clinical evaluation, *Clin Oral Implants Res* 13:381-389, 2002.
42. Pylant T, Triplett RG, Key MC et al: A retrospective evaluation of endosseous titanium implants in the partially edentulous patient, *Int J Oral Maxillofac Implants* 7:195-202, 1992.
43. Naert I, Quirynen M, van Steenberghe D et al: A six-year prosthodontic study of 509 consecutively inserted implants for the treatment of partial edentulism, *J Prosthet Dent* 67:236-245, 1992.
44. Jemt T, Lekholm U: Oral implant treatment in posterior partially edentulous jaws: a 5-year follow-up report, *Int J Oral Maxillofac Implants* 8:635-640, 1993.
45. Lekholm U, van Steenberghe D, Herrmann I et al: Osseointegrated implants in the treatment of partially edentulous jaws: a prospective 5-year multicenter study, *Int J Oral Maxillofac Implants* 9:627-635, 1994.
46. Higuchi KW, Folmer T, Kultje C: Implant survival rates in partially edentulous patients: a 3-year prospective multicenter study, *J Oral Maxillofac Surg* 53:264-268, 1995.
47. Gunne J, Jemt T, Linden B: Implant treatment in partially edentulous patients: a report on prostheses after 3 years, *Int J Prosthodont* 7:142-146, 1994.
48. Friberg B, Jemt T, Lekholm U: Early failures in 4,641 consecutively placed Brånemark dental implants: a study from stage 1 surgery to the connection of completed prostheses, *Int J Oral Maxillofac Implants* 6:142-146, 1991.
49. Jemt T, Lekholm U: Implant treatment in edentulous maxillae: a 5-year follow-up report on patients with different degrees of jaw resorption, *Int J Oral Maxillofac Implants* 10:303-311, 1995.
50. Testori T, Younan R: Clinical evaluation of short, machined-surface implants followed for 12 to 92 months, *Int J Oral Maxillofac Implants* 18:894-901, 2003.
51. Testori T, Wisemen L, Wolfe S et al: A prospective multicenter clinical study of the Osseotite implant: four-year interim report, *Int J Oral Maxillofac Implants* 16: 193-200, 2001.
52. Weng D, Jacobson Z, Tarnow D et al: A prospective multicenter clinical trial of 3i machined-surface implants: results after 6 years of follow-up, *Int J Oral Maxillofac Implants* 18:417-423, 2003.
53. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing and progressive bone loading, *Int J Oral Implant* 6:23-31, 1990.
54. Misch CE: Short dental implants: a literature review and rationale for use, *Dent Today* 24:64-68, 2005.
55. Misch CE, Steigenga K. Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 5 year case study, *J Periodontol* 77:1470-1477, 2006.
56. Tarnow DP, Cho SC, Wallace SS: The effect of interimplant distance on the height of inter-implant bone crest, *Periodontology* 71:546-549, 2000.
57. Hebel KS, Gajjar R: Achieving superior aesthetic results: parameters for implant and abutment selection, *Int J Dent Symp* 4:42-47, 1997.
58. Rangert B, Krogh P, Langer B et al: Bending overload and implant fracture: a retrospective clinical analysis, *Int J Oral Maxillofac Impl* 10:326-334, 1995.
59. Misch CE, Bidez MW: Occlusion and crestal bone resorption: etiology and treatment planning strategies for implants. In McNeill C, editor: *Science and practice of occlusion*, Chicago, 1997, Quintessence.
60. Misch CE, Wang HL: The procedures, limitations and indications for small diameter implants and a case report, *Oral Health* 94:16-26, August 2004.
61. Misch CE, Bidez MW: Implant protected occlusion, a biomechanical rationale, *Compend Contin Educ Dent* 15:1330-1342, 1994.
62. Misch CE: Maxillary anterior single tooth implant health esthetic compromise, *Int J Dent Symp* 3:4-9, 1995.
63. Misch CE, D'Alessio R, Misch-Dietsh F: Maxillary partial anodontia and implant dentistry—maxillary anterior partial anodontia in 255 adolescent patients: a 15 year retrospective study of 276 implant site replacements, *Oral Health* 95:45-57, 2005.
64. Misch CE, Dietsh F: Bone grafting materials in implant dentistry, *Impl Dent* 2:158-167, 1993.
65. Tatum HO: Maxillary and sinus implant reconstructions, *Dent Clin North Am* 30:207-229, 1980.
66. Misch CM, Misch CE, Resnik RR et al: Reconstruction of maxillary alveolar defects with mandibular symphysis grafts for dental implants: a preliminary procedural report, *Int J Oral Maxillofac Impl* 3:360-366, 1992.
67. Misch CM, Misch CE: The repair of localized severe ridge defects for implant placement using mandibular bone grafts, *Impl Dent* 4:261-267, 1995.
68. Misch CM: Ridge augmentation using mandibular ramus bone grafts for the placement of dental implants: presentation of a technique, *Pract Perio Aesth Dent* 8:127-135, 1996.
69. Ivanoff CJ, Grondahl K, Sennerby L et al.: Influence of variations in implant diameters: a 3-to 5-year retrospective clinical report, *Int J Oral Maxillofac Implants* 14:173-180, 1999.
70. Scurria MS, Morgan ZV, Guckes AD et al: Prognostic variables associated with implant failure: a retrospective effectiveness study, *Int J Oral Maxillofac Implants* 13: 400-406, 1998.
71. Scortecci GM: Immediate function of cortically anchored disk design implants without bone augmentation in moderately to severely resorbed completely edentulous maxillae, *J Oral Implant* 25:70-79, 1999.
72. Judy KW, Misch CE: Evolution of the mandibular subperiosteal implant, *N Y Dent J* 53:9-11, 1983.
73. Misch CE, Dietsh F: The unilateral mandibular subperiosteal implant: indications and technique, *Int J Oral Implant* 8:21-29, 1992.
74. Misch CE, Dietsh F: Endosteal implants and iliac crest grafts to restore severely resorbed totally edentulous maxillae: a retrospective study, *J Oral Implantol* 20:110, 1994.
75. Curtis TA, Ware WH, Beirne OR et al: Autogenous bone grafts for atrophic edentulous mandibles: a final report, *J Prosthet Dent* 57:73-78, 1987.
76. Albrektsson T: A multicenter report on osseointegrated oral implants, *J Prosthet Dent* 60:75-84, 1988.
77. Mason ME, Triplett RG, van Sickels JE et al: Mandibular fractures through endosseous cylinder implants: report of cases and review, *J Oral Maxillofac Surg* 48:311-317, 1990.

WWW.HIGHDENT.IR

Chapter 11

Scientific Rationale for Dental Implant Design

Carl E. Misch, J. Todd Strong, Martha Warren Bidez

The treatment planning sequence for implant dentistry begins with the design of the final restoration.[1] After the position and number of teeth replaced and type of prosthesis are determined,[2] the patient force factors are evaluated.[3] The greater the force factors, the greater the implant support required. The bone density in the region of the implant abutments is then considered, with poorer bone densities requiring a greater amount of implant support.[4] The key implant positions and additional implant number are then determined, followed by the ideal implant size.[5] The available bone in the edentulous sites is then evaluated.[6] When the bone available is present for the size, number, and position of the implants for the planned prosthesis, the treatment proceeds with little compromise. When the bone is not present, a modification of the treatment is necessary. These modifications include: (1) bone augmentation to fulfill the ideal treatment plan; (2) consideration of optional implant locations, usually with additional implants, or an increase in implant size; or (3) optimization of implant design. A favorable implant design may compensate for risk for occlusal loads in excess of normal, poor bone densities, less than ideal implant position or number, or less than an ideal implant size.[7]

There are many different implant body designs available in implant dentistry. They may be categorized as a cylinder type, screw type, press fit, or a combination of features. Dental implants are often designed to answer a primary focus or belief that implant failure may stem from (1) implant surgery, (2) bacterial plaque complications, or (3) loading conditions. For example, in the past, implant body design was driven by the surgical ease of placement. A surgically driven implant design will tend to have a tapered, short implant body or a press-fit insertion. These features permit the implant site and implant to be surgically placed most easily.

A cylinder or press-fit implant has a friction-fit insertion and may have less risk of pressure necrosis from too tight an insertion pressure, has no need to bone tap (even in dense bone), and may have the cover screw already in place because no rotational force is required to insert the implant. As a result, cylinder or press-fit implants are the easiest to insert, were very popular in the 1980s, and were reported to have high initial success rates.[8,9] However, after 5 years of loading, reports of the cylinder implant include loss of crestal bone and implant failure more often observed (Figure 11-1).[10] Most likely, this was related to a fatigue overload condition and harmful shear loads on the bone causing large bone turnover rates, and ultimately less bone-implant contact percent and higher risk of overload failure.

The most predictable aspect of implant dentistry appears to be the surgical success. After many years of clinical studies and evaluations, the surgical success rate from implant insertion to uncovery of the implant is usually higher than 98%, regardless of the implant design or size.[11-14] As such, designing an implant for surgical ease does not appear to be the most important aspect of the overall implant-prosthetic–related process to reduce the incidence of complications.

Another focus of several implant designs is to reduce the plaque-related complications of treatment. With this concept in mind, one consistent implant body design features smooth metal surfaces at the crestal portion of the implant. A smooth crest module (the area between the implant body and prosthetic platform) of the implant is easier to clean related to oral hygiene methods and collects less plaque than rougher surfaces.[15] Therefore the rationale is that if bone loss occurs at the marginal regions of the implant, the smooth implant surface will harbor less plaque and be easier to clean.

The problem with this philosophy is the smooth crest module is initially placed below the crest of the bone and is a design that encourages marginal bone loss from the extension of a biological width after implant uncovery and from shear forces after occlusal loading.[16,17] As a result, this plaque-reducing design feature increases the peri-implant sulcus depth. Paradoxically, the feature designed to decrease bacteria complications actually increases the risks. Therefore an implant body designed for ease of surgery or to reduce peri-implantitis complications most often does not address the most common complications observed in implant prostheses.

WWW.HIGHDENT.IR

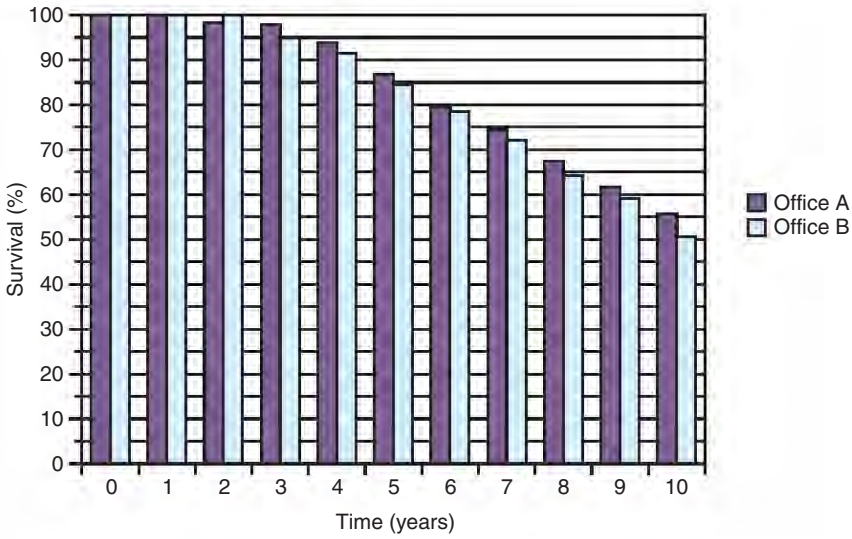


Figure 11-1 The hydroxylapatite-coated cylinder implant has almost 100% success in two offices for the first 3 years. After 5 years, 20% failure rates are observed, and at 10 years almost 50% failure rates are reported.

Table 11-1 Success Rates Using 5 mm as Borderline Compared to Clinical Survival

IMPLANT TYPE	NUMBER OF IMPLANTS PLACED	SUCCESS	CLINICAL SUCCESS*
Hollow screw	112	83 (74%)	101 (90.2%†)
Hollow cylinder	49	31 (63%)	37 (76%)
Angulated hollow cylinder‡	18	11 (61%)	16 (89%)
All implants	179	125 (69.8%)	154 (86%)

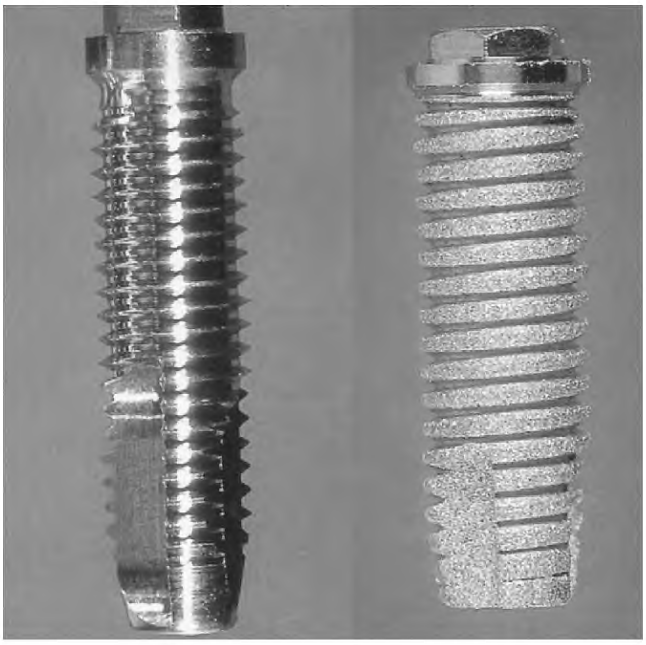
Data from Karoussis IK, Bragger U, Salvi GE et al: Effect of implant design on survival and success rates of titanium oral implants: a 10-year prospective cohort study of the ITI Dental Implant System, *Clin Oral Implants Res* 15:8-17, 2004.
* Clinical success meaning that the implant fulfilled the clinical criteria.
† Represents statistically significant difference compared with HC (Fisher's exact test, $P < 0.025$).
‡ ITI dental implant system.

Most implant body complications in the literature are related to early implant failure after loading, marginal bone loss before loading but after exposure of the implant, and marginal bone loss after the loading of the implant-bone interface.[18] Implant failures are most often observed as early loading failures in softer bone types or shorter implant lengths. A review of the literature by Goodacre et al. between 1981 and 2003 found that, after the implant was loaded, failure rates increased to an average of 16% in soft bone.[18] Misch reviewed the literature between 1981 and 2004 and found an overall 18% failure rate in implants shorter than 10 mm.[19] For example, Weng et al. reported in a multicenter study of 6 years, implant initial healing survival higher than 98%, but 7-mm implants failed 25% of the time within 18 months of loading and short implants in the posterior maxilla had the highest early loading failure of any intraoral region.[20] Therefore implant body designs should attempt to primarily address the primary causes of complications (i.e., the factors that address the loading conditions of the implant after the implants are placed in function).

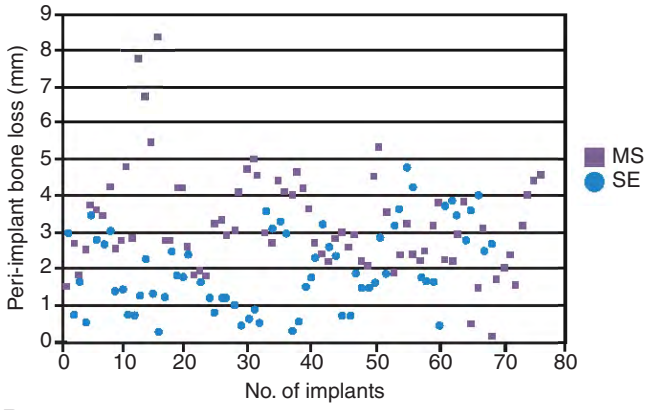
Different implant survival rates and different marginal bone loss after loading have been reported for different implant body designs. A report by Zechner et al. evaluated the peri-implant bone over a 3- to 7-year period around functionally loaded, screw-type implants with a machined surfaced V-thread and sandblasted, acid-etched, square-thread design.[21] Both these implants had a similar crest module and external hex connection. The range of bone loss in the study was 0.1 to 8.5 mm for the machined V-thread and 0.2 to 4.8 mm for the rough surface, square-threaded implant. There were 22 V-threaded implants that lost more than 4 mm of bone compared with three square-threaded implants. Bone loss of less than 1 mm was reported for 16 rough surface implants compared with only two machined surface implants (Figure 11-2). The range of bone loss and the incidence of bone loss both indicate implant design or surface condition made a difference in this report.

A prospective report by Karoussis et al. also indicated that different implant designs yield different incidences of crestal bone loss.[22] Three different implant designs from the same manufacturer (Straumann ITI Dental Implant Systems, Basel, Switzerland) were evaluated with a prospective study over 10 years (Table 11-1). The survival rate of each design over this time was 95.4% versus 85.7% versus 91.2%. The implant with the

WWW.HIGHDENT.IR



A



B

Figure 11-2 **A,** Two different implant designs were evaluated over a 3- to 7-year period in the anterior mandible. The crest module and microgap position were similar. The implant on the left was a machined, V-shaped thread. The implant on the right was a roughened, square-threaded design. **B,** A 22–V-thread design implant (MS) lost more than 4 mm of bone over a 3- to 7-year period. In the square-threaded implant (SE) design group, 16 implants lost less than 1 mm of bone during this time frame.

highest survival rate (hollow screw design) lost more than 5 mm of bone in 26% of the implants, whereas the two other designs reported 37% and 39% incidence of greater than 5-mm bone loss. More than 6 mm of marginal bone loss occurred in 22% of implants with the first design and in 35% and 33% for the other two designs. Therefore implant survival and marginal bone loss was related to implant design.

In a 3-year clinical study, a different amount of marginal bone loss was reported between the Nobel Biocare Brånemark implant and the Straumann ITI implant, with ITI having a greater range of bone loss after loading (Figure 11-3).[23] The microgap and machined surface of

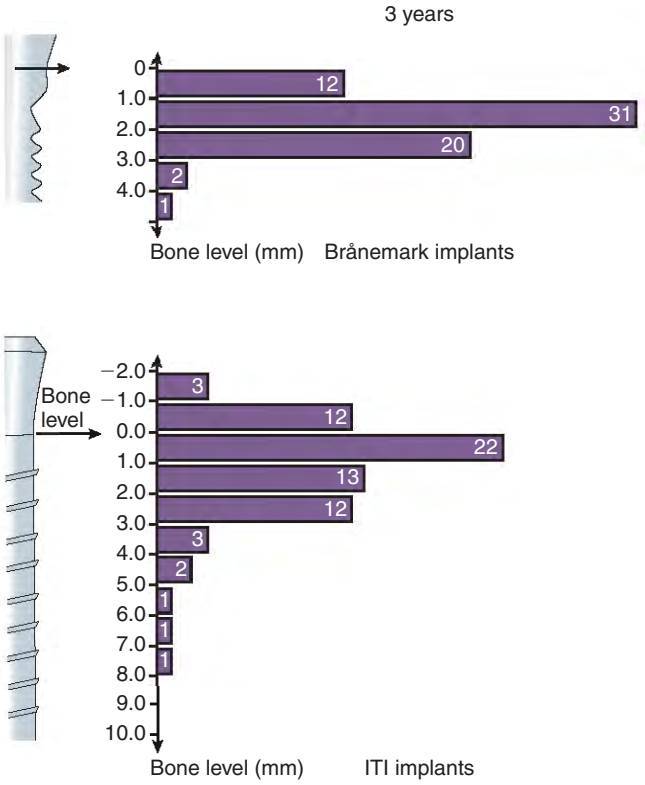


Figure 11-3 Brånemark implants and ITI implants were compared in the partially edentulous maxilla. The ITI implant *(bottom)* has a wider range of marginal bone loss.

the Nobel implant caused less bone loss than the rough cylinder shape at the crest after loading with ITI. In other words, several clinical reports found different implant designs influence not only the implant survival, but also the amount of early crestal bone loss after loading. This chapter will build on and apply basic biomechanics and demonstrate how these principles are related to implant design in order to decrease the more common complications observed in implant dentistry.

IMPLANT DESIGN RELATED TO OCCLUSAL FORCES

Dental implants function to transfer loads to surrounding biological tissues. Thus the primary functional design objective is to manage (dissipate and distribute) biomechanical loads to optimize the implant-supported prosthesis function. Biomechanical load management is dependent on two factors: the character of the applied force and the functional surface area over which the load is dissipated. There are more than 90 dental implant body designs available. A biomechanical rationale of dental implant design may evaluate these designs as to their efficacy to manage biomechanical loads. Fundamental scientific principles related to force and surface area may then be combined with engineering principles to pursue the desired clinical goals.

WWW.HIGHDENT.IR

Force Type and Influence on Implant Body Design

Three types of forces may be imposed on dental implants within the oral environment: compression, tension, and shear. Bone is strongest when loaded in compression, 30% weaker when subjected to tensile forces, and 65% weaker when loaded in shear.[24] An attempt should be made to limit shear forces on bone, because it is least resistant to fracture under these loading conditions. This is most important in regions of decreased bone density, because the strength of bone is also directly related to its density.[25]

An implant has a macroscopic body design and a microscopic component to implant design. Both design features (although independent) are relevant for the clinical behavior. The microscopic features are most important during initial implant healing and the initial loading period. The macroscopic implant body design is most important during early loading and mature loading periods. This chapter will focus on the macroscopic aspect of implant body design.

Smooth-sided, cylindrical implants provide ease in surgical placement; however, the bone-implant interface is subject to significantly larger shear conditions. In contrast, a smooth-sided, cylindrical, tapered implant provides for a component of compressive load to be delivered to the bone-implant interface, depending on the degree of taper.[26] The greater the taper, the greater the component of compressive load delivered to the interface. Unfortunately, the amount of taper cannot be greater than 30 degrees or the implant body length is significantly reduced, along with the immediate fixation required for the initial healing. As a negative feature, the greater the taper of a smooth-sided implant, the less the overall surface area of the implant body under load and the less initial stability provided by that implant at an immediate extraction and implant insertion.

Unlike a cylinder implant, a tapered threaded implant serves no functional surface area advantage, because the threads of a screw bear the compressive loads to the bone. The tapered, threaded implant provides some surgical advantage during initial insertion, because it inserts down within the osteotomy halfway before engaging bone. However, the lesser surface area of a tapered implant increases the amount of stress at the crestal portion, as demonstrated in three-dimensional finite element studies.[27] In addition, in a tapered threaded implant, threads at the apical half are often less deep, because the outer diameter continues to decrease. This limits the initial fixation of the implant.

A smooth-cylinder implant body results in essentially a shear load at the implant-bone interface. Bone grows to a cylinder-shape implant during initial healing. However, this type of body geometry must rely on a microscopic retention system such as roughening or coating (acid etch, mechanical etch, or coatings such as titanium plasma spray or hydroxylapatite [HA]) for the initial loading period.[28] The integrity of the implant interface during initial loading is therefore dependent on the shear strength of the implant surface-to-bone bond. The quality of the coating (e.g., HA) is absolutely paramount in such applications.[29] If the HA is altered from friction during implant surgical insertion, infection, mechanically removed during treatment of peri-implantitis or from bone remodeling over years of function, the remaining smooth-sided cylinder is severely compromised for healthy load transfer to the surrounding tissues (Figure 11-4).[30]

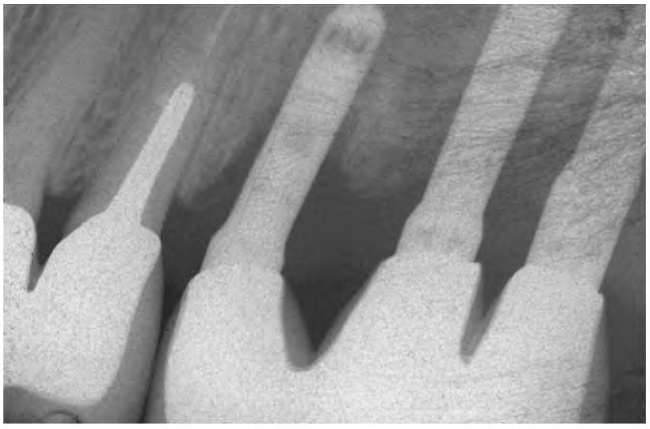


Figure 11-4 The microdesign of a cylinder implant places shear loads to the bone interface. As such, the surface conditions must be rough to compensate for the implant body design. In situations that compromise the integrity of the surface coating, the load bearing capabilities of this design are greatly compromised.

The surface conditions of an implant may enhance bone-implant contact (BIC) and adhesion qualities to the bone-implant interface at initial healing. However, the surface coatings on cylinders do not permit compressive forces to be effectively transmitted to the bone cells, because the microfeatures of the coating are too small for the cells to be loaded in compression.[31] Therefore the surface area–bone contact percentage is greater during initial healing, but the functional surface area over which loads are effectively dissipated during long-term loading to the surrounding bone is most dependent on the macroscopic design of the implant body. For example, Watzek et al. evaluated screw shape and cylinder implants with histologic and histomorphometric analysis after 18 months of occlusal loading in baboons.[32] There was a significant difference in BIC, with screw-type implants having higher values in both the maxilla and mandible (Figure 11-5).[32] In addition, the trabecular bone pattern was irregular around the cylinder implants, but the bone was organized perpendicular to the threads around the screw implants (Figure 11-6).[32] Therefore the bone trabeculae pattern and the higher BIC resulted in a superior support system for threaded implants.

An implant retrieval clinical report by Bolind et al. evaluated cylinder implants compared with threaded

WWW.HIGHDENT.IR

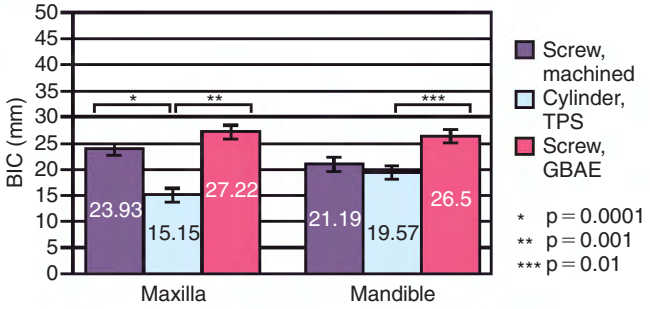


Figure 11-5 Comparison of a cylinder and two screw-type implant designs used in baboons. The bone-implant contact (BIC) is greater in the maxilla and mandible for screw-type designs.

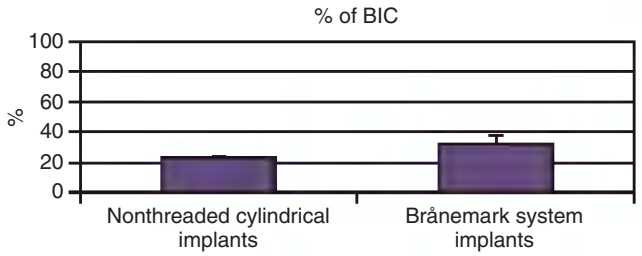


Figure 11-7 Evaluation of human retrievals of 85 cylinder implants and 85 threaded (machined) implants. The bone-implant contact (BIC) is greater in threaded implants, and greater marginal bone loss is observed in the cylinder implant group.

implants from functioning prostheses.[33] Consecutively retrieved oral implants from 117 patients, with 85 cylinder implants and 85 threaded implant designs, were evaluated. Greater BIC was found around threaded implants, and greater marginal bone loss was observed around cylinder implants (Figure 11-7).[33] It should be noted the cylinder implants in this report had a roughened surface condition, whereas the threaded implants had a machined surface. Numerous reports demonstrate roughened surfaces have higher BIC compared with machined surfaces. In this human retrieval report, therefore, implant body design was more important than the surface condition of the implant for crestal bone loss and overall BIC after loading. In Figure 11-8, an HA-coated cylinder is splinted to two HA-coated threaded implants. More crestal bone loss is observed on the cylinder implants.

Any smooth shear surface on an implant body increases the risk of bone loss because of inadequate load transfer.[34] Figure 11-9 depicts one such example characterized by extensive crestal resorption adjacent to a long, smooth shear surface on the two implant bodies (Core-Vent/Paragon implant). The crestal bone loss contributed to an increase in crown height (which further magnifies stress from bending moments) and the fracture of two abutments. The implant body next to these cylinder-type implants was loaded in the same prosthesis, yet the plateau implant body design (which is a compressive load design) maintained bone height over the years of loading.

Force Direction and Influence on Implant Body Design

Bone is weaker when loaded under an angled force.[35] The greater the angle of load, the greater the stresses to the implant-bone interface. The noxious effect of angled loads to bone is further exacerbated because of the anisotropy of bone.[36] Anisotropy refers to how the character of bone's mechanical properties, including

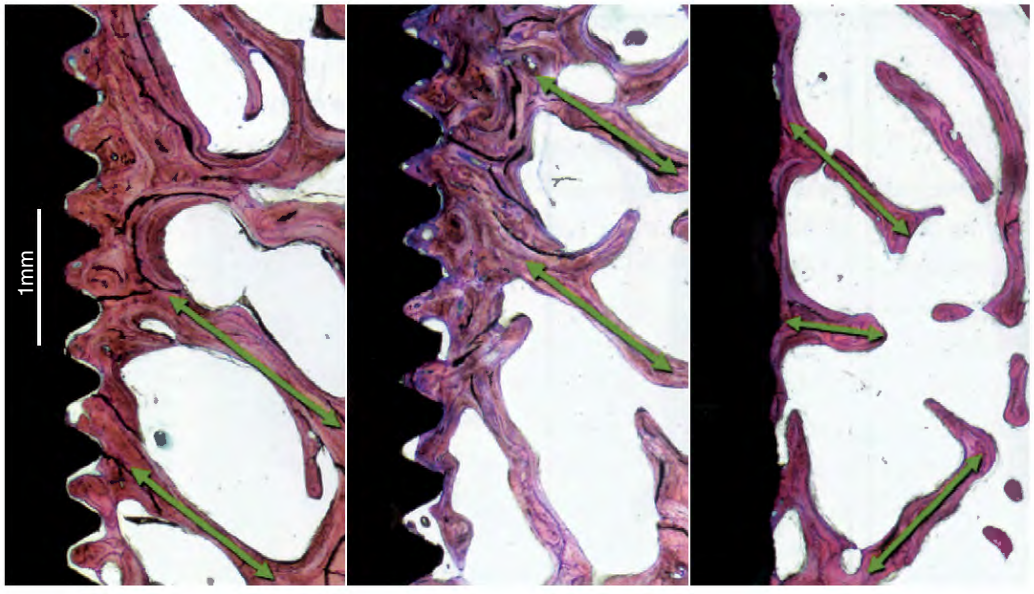


Figure 11-6 Trabecular bone has a more organized form to resist occlusal stresses for screw-type implants compared with cylinder implants. *(From Watzek G, Zechner W, Ulm C et al: Histologic and histomorphometric analysis of three types of dental implants following 18 months of occlusal loading: a preliminary study in baboons,* Clin Oral Implants Res *16:408-416, 2005.)*

WWW.HIGHDENT.IR

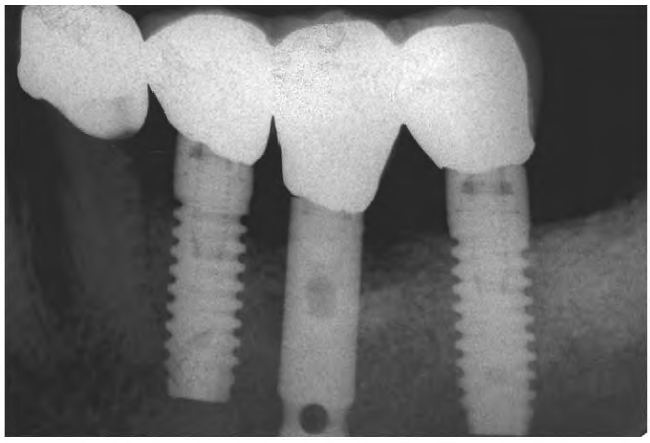


Figure 11-8 A hydroxylapatite (HA)-coated cylinder implant is splinted to two HA-coated screw type implants. Greater crestal bone loss is observed around the cylinder implant.

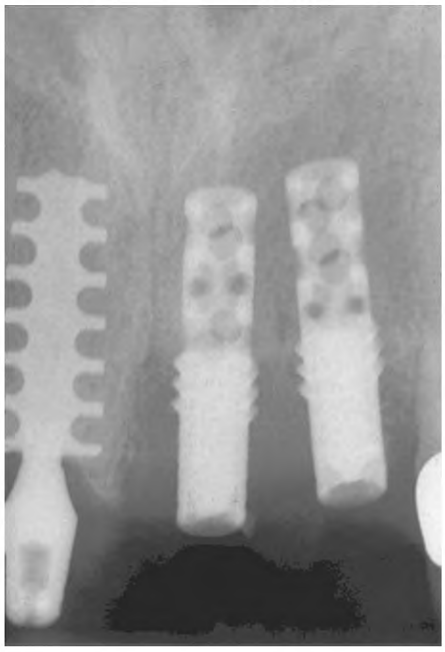


Figure 11-9 Two cylinder-neck design implants were splinted to a plateau implant design. After several years of loading, the crestal bone loss on the cylinder-neck design led to implant abutment fracture.

ultimate strength, depends on the direction in which the bone is loaded. A 30-degree angled load will increase the overall stress by 50% compared with a long axis load, especially around the crestal portion of the implant.[37] Therefore, under ideal conditions, the implant body long axis should be perpendicular to the curve of Wilson and curve of Spee to apply a long axis load to the implant during occlusal load in centric occlusion (where the occlusal forces are usually the greatest). As the angle of load to the implant-bone interface increases, the stresses around the implant increase. As a result, virtually all implants are designed for placement perpendicular to the occlusal plane. Additionally, axial alignment places less shear stress on the overall implant system (i.e., porcelain, cement, abutment and abutment screw components, crestal bone, implant body, and implant-bone interface) and decreases the risk of complications, as screw loosening and fatigue fractures.

Implant body designs with threaded features have the ability to convert occlusal loads into more favorable compressive loads at the bone interface; therefore thread shape is particularly important when considering long-term load transfer to the surrounding bone interface (Figure 11-10). Under axial loads to an implant-bone interface, a buttress or square-shaped thread (typical of BioHorizons, BioLok, and Ankylosis) would transmit compressive forces to the bone. Under axial loads to a dental implant, a V-thread face angle (typical of implants from Zimmer, LifeCore, 3i, and some Nobel Biocare designs) is comparable to the reverse buttress thread (typical of some Noble BioCare designs) because of the similarity in the inferior portion of the thread face angle.

A reduction in shear load and subsequent shear stresses at the thread-bone interface reduces the risk of bone failure and possible reduced bone-implant contact percent of the implant if all the other factors are equal, which is particularly important in compromised

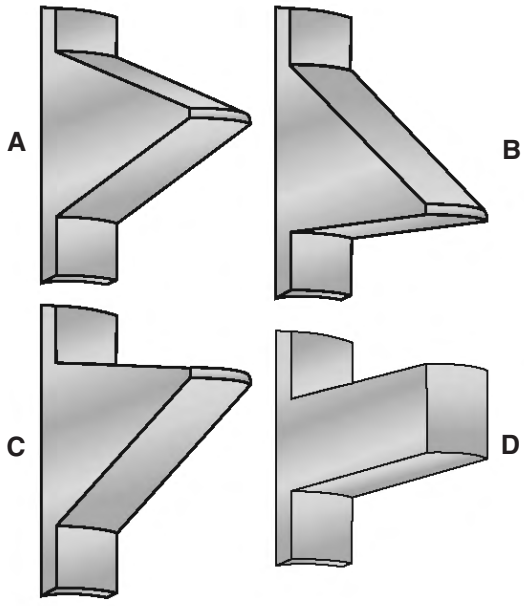


Figure 11-10 The four basic thread shapes for implant design include: **(A)** V-thread, **(B)** buttress thread, **(C)** reverse buttress thread, and **(D)** square thread.

bone densities or shorter implant lengths.[38] The thread shape (macroscopic design) is independent from the surface coating (microscopic design). For example, any threaded implant surface may also be textured with HA coating or other roughened surface conditions to enhance osteointegration and increase the bone-implant contact percent during initial healing.

WWW.HIGHDENT.IR

IMPLANT BODY: FUNCTIONAL VERSUS THEORETICAL SURFACE AREA

Forces applied to the implant may be evaluated in magnitude, duration, type, and direction. The surface area over which the forces are applied is also relevant and is inversely proportional to the stress observed within the implant system *(stress = force ÷ surface area)*. It can be clearly seen from this basic engineering equation that, to reduce stress, the force must decrease or the surface area must increase.

For a given bone volume, implant surface area should be optimized for functional loads. Thus an important distinction is made between theoretical total surface area and functional surface area of an implant. Because bone is 65% weaker to shear forces and 35% weaker to tensile forces, functional surface area is defined as the area that actively serves to dissipate compressive loads to the implant-bone interface.[38] Functional thread surface area, therefore, is that portion of the thread that participates in compressive load transmission under the action of an axial (or near-axial) occlusal load.

In contrast to functional surface area is total theoretical surface area, which may include a "passive" area on the implant that does not participate in load transfer, or has a feature so small bone cannot adapt to load transfer. For example, plasma spray coatings are often reported to provide up to 600% more total surface area for the potential bone-implant contact.[26] However, the size of each plasma spray particle is less than 8 µm, and the 120-µm bone cell does not receive a transfer of mechanical stress from this feature. The amount of actual BIC that can be used for compressive loading may be less than 30% of the total theoretical surface area (Figure 11-11).

An improved functional surface area per *unit length of the implant* (in contrast to *total* surface area) is beneficial to reduce the mechanical stress to bone. Most stress to the implant-bone interface in D1 to D3 bone is in the crestal 5 to 9 mm of the implant; therefore the design of the implant body in the coronal 9 mm is most important to appropriately distribute occlusal stresses to the bone.[27,39,40] For example, a conventional 20-mm V-shaped or reverse buttress-threaded implant of a constant thread depth may have more *total* surface area compared with a 13-mm square threaded or plateau design implant. The *functional* area, however, that is available to resist compressive biomechanical loads in the zones of greatest stress to the BIC may be significantly higher in the 13-mm implant because of the thread geometry.

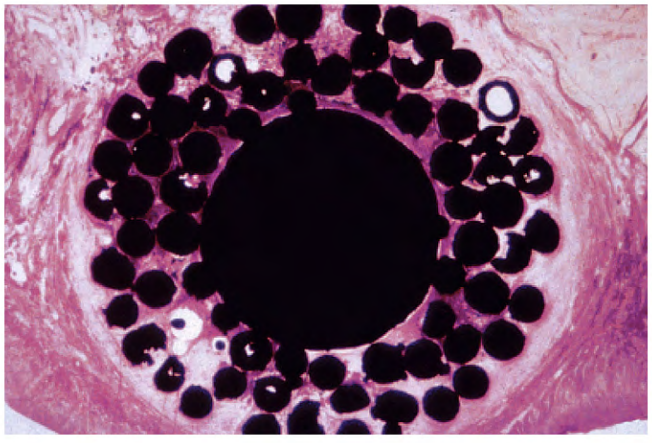


Figure 11-11 Functional surface area of an implant design does not include spaces too small for bone to grow into and act as a stress transfer medium. *(From Steflik D, Corpe RS, Young TR et al: Light microscopic and scanning electron microscopy retrieval analysis of implants biomaterials retrieved from humans and experimental animals,* J Oral Implantol *27:5-15, 2001.).*

Functional surface area also plays a major role in addressing the variable initial BIC zones related to bone density upon initial loading. D1 bone, the densest bone found in the jaws, is also the strongest, has the stiffest modulus of elasticity, and has the highest initial BIC percent, which approximates 80%. D2, D3, and D4 bone have progressively decreasing percentages of bone at the initial implant interface, with D4 bone ranging around 25% interface contact at the initial healing and uncovery of a machined titanium implant.[4,41] As a result, the implant geometric body design, length, and bone density are related to the functional surface area. For example, in more compromised bone sites (i.e., D4 bone), longer implants are required to resist off-axis and moment loads because of cantilevers, improper occlusion, or parafunction.[42] Recall that mechanical stress is equivalent to the applied load divided by the surface area over which the load is dissipated. D4 bone has the weakest biomechanical strength and the lowest BIC area to dissipate the load at the implant-bone interface. The functional surface area requirements would increase from a minimum for an implant in D1 bone to a maximum for implants in the D4 bone (Figure 11-12).[42]

The diameter of an implant may influence the length requirements of the implant body, because the functional surface area is increased. However, the higher bite forces and lower bone density of the posterior regions of the mouth may not always be able to be addressed adequately by only implant diameter. For example, Ivanoff et al. in 1999 found 6-mm-long implants with a 5-mm diameter had a failure rate of 33% in the mandible and 10% in the maxilla.[44] The 8-mm-long, 5-mm-diameter implant yielded 33% and 25% failure rates in the mandible and the maxilla, respectively. On the other hand, the longer 10-mm and 12-mm implants that were 5 mm in diameter yielded no mandibular failures and a 10% failure rate in the maxilla. Therefore the 5-mm implant diameter was not enough to compensate for the shorter implants in this report.

An ideal treatment plan would include implant length of 12 mm or greater with 4-mm diameter for most anterior implant sites and 5 mm or greater in the

WWW.HIGHDENT.IR

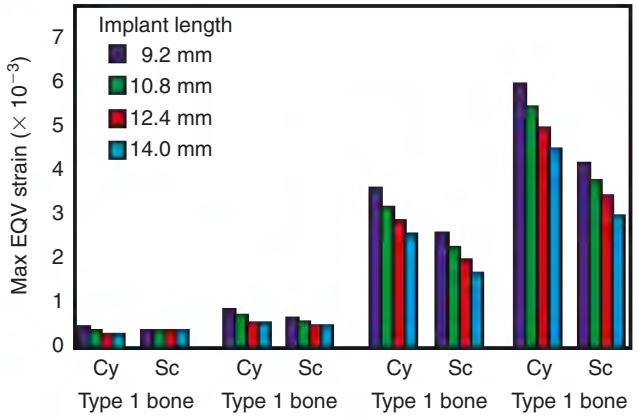


Figure 11-12 The required functional surface area is less for implants in D1 bone *(far left)* and greatest for implants in D4 bone *(far right)*. Screw-type implants have more functional surface area, which is an advantage, especially in softer bone types. *(From Tada S, Stegiaroiu R, Kitamura E et al: Influence of Implant design and bone quality on stress/strain distribution in bone around implants: a 3-dimensional finite element analysis,* Int J Oral Maxillofac Implants *18:357-368, 2003.)*

molar regions.[5] When the ideal implant size cannot be inserted because of inadequate bone, an alternative to bone augmentation may be to increase the surface area of the implant by modifying the implant body design.

IMPLANT BODY GEOMETRY VERSUS OCCLUSAL LOAD

Different implant survival rates and amounts of marginal bone loss may be directly related to different implant body designs. The macrodesign of an implant has an important bearing on the overall surface area to the load of the bone. Protruding elements of the implant surface, such as ridges, crests, teeth, ribs, or the edge of threads may act as stress transfers to the bone when load is applied. Hoshaw et al. tensile loaded titanium V-threaded implants (Nobel Biocare Brånemark) in the cortical bone of canine tibiae (Figure 11-13).[45] The osteons in the tibia are usually oriented parallel to the long axis of the bone. However, around the implants with axial tensile loads, osteons were oriented encircling the implants and secondary osteons appeared oriented around the depth of the implant threads. Therefore the cortical bone remodeling observed around endosteal implants may be an attempt to improve strain orientation of the bone in relation to the functional loads.

In an animal study using a square thread, microscopic observation noted that when the bone did not fully occupy the threads, greater bone volume was observed on the lower aspect compared with the upper aspect of the square threads (Figure 11-14).[46] In addition, a bone bridge was found from one square thread to another. The square thread shape of the tested implants was designed to enhance compression loads and reduce shear loads

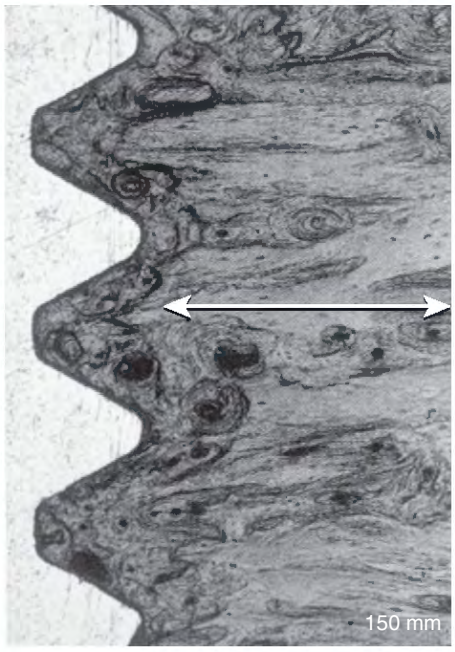


Figure 11-13 Hoshaw tensile loaded implants in dog tibiae. The osteons in the tibia of the dog run parallel to the long axis to resist the weight and axial forces of the animal. When the implants were inserted perpendicular to the long axis and tensile loaded for 5 days, the osteons 4 to 5 mm around the implants reoriented to encircle the implant rather than remain parallel to the long axis of the tibiae to resist the tensile loads. *(From Hoshaw SJ, Brunski JB, Cochran GVB: Mechanical loading of Brånemark fixtures affects interfacial bone modeling and remodeling,* Int J Oral Maxillofac Implants *14:173-180, 1999.)*

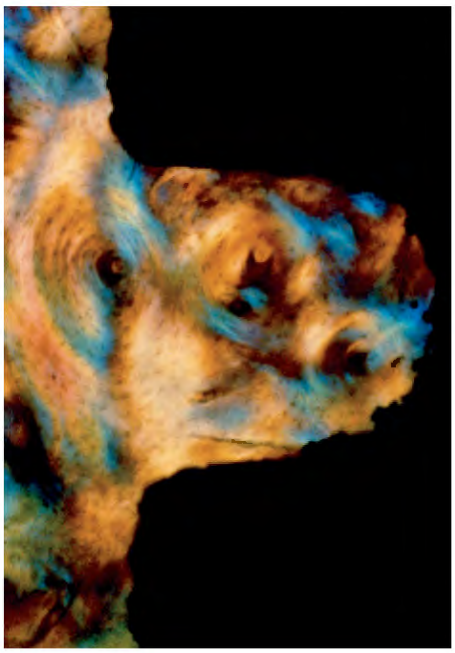


Figure 11-14 Bone bridges forming from one square thread to another and a higher bone contact percent on the bottom of a square thread, which could load the bone in compression. *(From Baumgardner JD, Boring G, Cooper RC et al: Preliminary evaluation of a new dental implant design in canine models,* Implant Dent *9:252-260, 2000.)*

WWW.HIGHDENT.IR

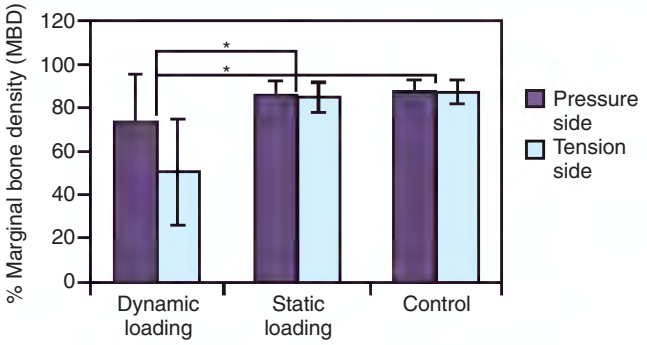


Figure 11-15 There is more bone on the bottom of a V-threaded implant (pressure side) compared with the thread (tensile side) for dynamic-loaded implants. The static loaded and unloaded implants have a similar bone density on the top and bottom of the thread.

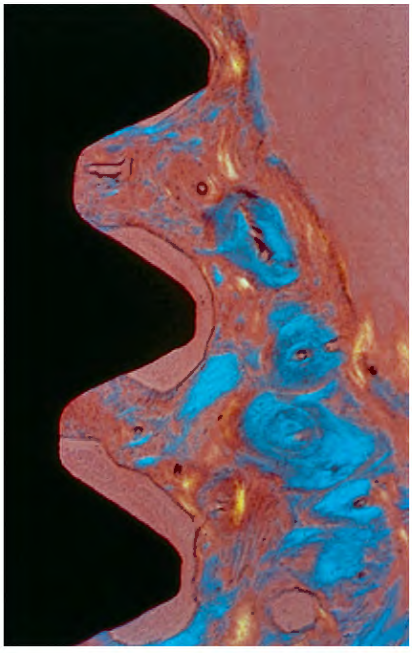


Figure 11-16 Lateral loads are placed to V-shaped implants, and bone bridging is found from the depth of one thread to another. Because the strain would be highest at the peak of a thread and lowest at the depth, the bone contact point is related to lower strain positions. *(From Kohn DH: Overview of factors important in implant design,* J Oral Implantol *18:204-219, 1992.)*

delivered to the implant interface. Duyck et al. also found in an animal model that the bone density was equally distributed above and below a threaded implant after initial bone healing. However, after dynamic loading, the bone implant density was greater on the bottom of the thread face angle and less on top of the thread (Figure 11-15).[47] Kohn and Hollister demonstrated that when the Nobel Biocare Brånemark implant was loaded laterally, a bone bridge formed from the depth of one thread to another (Figure 11-16).[48] The local strain field within the bone-implant interface under this condition is inhomogeneous. During a lateral load, the strain was more concentrated at the tip of the thread and the strain was decreased from the exterior to the interior regions of the thread. Kohn and Hollister speculated that because the strains were highest at the tip of each thread, bone was resorbed, and where strains were reduced at the depth of the thread, bone was maintained.

A retrieval report by Bolind et al. confirmed a consistent pattern of BIC location in humans.[33] The bone contact was least at the tip of each thread (where the highest strain occurs) and was the greatest under the thread face angle (where the bone is loaded more in compression). Therefore the design of the implant not only governs the initial stability of the implant, but as important determines the BIC percent and location of contact available for effective load transfer to the bone after occlusal loading.

SCIENTIFIC RATIONALE FOR IMPLANT DESIGN

Cortical and trabecular bone are modified by modeling or remodeling.[49] Modeling is the result of independent sites of formation and resorption that change the shape or size of bone. Remodeling is a process of resorption and formation at the same site that replaces previously existingbone and is primarily responsible for the change in bone quality. Bone modeling and remodeling are primarily controlled by the mechanical environment of strain.

The histologic description of bone includes lamellar bone, woven bone, composite bone, and bundle bone.[50] The first two of these bone types are often found next to an osteointegrated dental implant. Lamellar bone is the most organized, highly mineralized, and strongest of the bone types. It has been called *load-bearing bone* and is most desired next to an implant. Woven bone also is called *immature bone* because it is unorganized, less mineralized, and has less strength than the other types. These histologic terms may be used to describe the macroscopic bone types of cortical and trabecular bone.

Nicolella et al. found that a 0.15% deformation in a bone specimen may have microstructural level strain values as large as 3.5% at various regions within the cellular microstructure.[51,52] Microstrain levels 100 times less than the ultimate strength of bone may be responsible for remodeling rates within the structure, because the bone cell membranes are able to act as a mechanosensory system in bone.[53] In other words, the cellular behavior of bone cells is largely determined by the mechanical environment of strain or deformation of the bone cells.[54-56] It is speculated that the source of energy to open the ion membrane channels in the bone cell membrane is the microstrain in the cells as a result of the load applied to the bone.[57,58]

Frost described four microstrain zones for compact bone and related each of these categories to the mechanical adaptation to strain.[59] These four zones include the pathologic overload zone, the mild overload

WWW.HIGHDENT.IR

zone, the adapted window, and the acute disuse window. Briefly stated, the pathologic overload zone and the acute disuse window are the two extremes of bone reaction to strain conditions. Each of these conditions, however, may result in a similar condition of less bone. Pathologic overload could lead to microfractures, which require repair and may result in net bone resorption. The disuse zone also increases remodeling, which decreases bone mass (see Figure 7-7).

The remodeling rate, or bone turnover, is the time needed for new bone to replace the existing bone and allows for the adaptation of bone to its environment (e.g., next to a dental implant).[60] The bone remodeling rate (BRR) also has been expressed as a percentage or volume of new bone within a specific time period. Lamellar bone forms at a rate of 1 to 5 µm each day, whereas woven bone can form at rates of more than 60 µm each day.[61,62] Therefore a higher BRR is directly related to an increase in the amount of woven bone formation. The mild overload zone is more likely to have a higher BRR than the adapted window zone and more reactive woven bone formation (less organized, less mineralized, and weaker) to create and maintain bone mass in response to the mechanical challenge.[63] The adapted window zone is most likely to be organized, highly mineralized, lamellar bone. Misch et al. stated the adapted window would be the ideal strain condition next to a dental implant, providing bone that is more mature and more resistant to periodic changes in strain conditions.[64] Therefore the BRR may be directly related to the strength of the implant interface and the degree of risk for the implant-bone interface. The higher risk is related to higher turnover rates, because the bone is less mineralized, less organized, and less strong at the interface.

Interface remodeling allows a viable bone interface to form between the dental implant and the original bone after the implant has been surgically inserted.[62] Before implant insertion, the bone is usually mature, lamellar bone. The surgical trauma causes the bone to repair, with primarily woven bone formation. By the end of 4 months of a maturation phase next to an implant interface, osteoblasts have deposited about 70% of the mineral found in mature vital bone and have reformed lamellar bone. The remaining 30% of mineral deposition occurs during secondary mineralization over the next 8-month period.[50]

After the bone has healed and the implant is then loaded, the interface again remodels, as influenced by its local strain environment.[49] If woven bone forms as a result of mechanical loading, it is called *reactive woven bone* and is very similar in structure and properties to the "repair" woven bone from surgical trauma. The long-term maintenance of the implant involves a continuous remodeling of the interface. In part, this allows new bone to replace bone, which may have sustained microfractures or fatigue as a result of cyclical loading. In vivo microdamage in bone and an elevated remodeling activity to repair those regions have been identified by Frost.[65] To date, the BRR of the bone in the jaws for humans is not well documented; however, it appears to reach 40% each year.[66]

Microdamage in cortical bone surrounding screw-type implants has been reported during both insertion and with pullout forces, and the amount of microdamage was related to the thread design of the implants.[67] Microdamage acts as a key step in signaling increases in remodeling and replacement of skeletal tissue and is similar to the local tissue remodeling response to physical injury in other tissues.[68] Mori and Burr provided evidence of an increased BRR in regions of microfracture from fatigue damage.[69] Verborgt et al. found that in the ulnae of rats, fatigue loading produced a large number of terminal dUTP nick-end labeling to detect apoptotic cells (TUNEL)-positive osteocytes in bone surrounding microcracks.[70] The intracortical resorption was almost 300% greater than the controls. The authors noted a strong association between microdamage, osteocyte apoptosis, and subsequent bone remodeling. Therefore, in addition to the increased BRR at the interface that is related to the trauma induced during the implant surgery, there may be heightened remodeling of bone some distance beyond the surgical interface after loading. Hoshaw et al. found an increase in bone remodeling next to threaded dental implants in dog tibiae when loaded for 5 consecutive days after a 12-month initial healing period.[45] Hoshaw et al. also found titanium threaded implants with axial tensile loading have higher remodeling rates and less mineralized bone than control implants that did not receive a load after healing.[71] The increase in the BRR found in the overload zone of Frost and the increase in BRR from the microfracture are directly related.

Barbier and Schaper investigated implant-supported prostheses under nonaxial and axial loads.[72] A higher cellular response, including osteoblasts and inflammatory cells, was observed next to implants under nonaxial shear loading conditions compared with axial loads (Figure 11-17). These authors stated nonaxially loaded implants exhibited a greater BRR compared with axially loaded implants in animals with the same implant design. It appears the implant design, direction of load, or surface condition may all affect the bone at the implant interface, which affects the bone turnover rate at the interface. Because bone in the jaws usually remodels at 40% per year, this most likely represents the adapted window zone. The higher the BRR, the more likely the bone is in an overload condition.

Thread Geometry

Threads are designed to maximize initial contact, enhance surface area, and facilitate dissipation of loads at the bone-implant interface.[38] Functional surface area per unit length of the implant may be modified by varying

WWW.HIGHDENT.IR

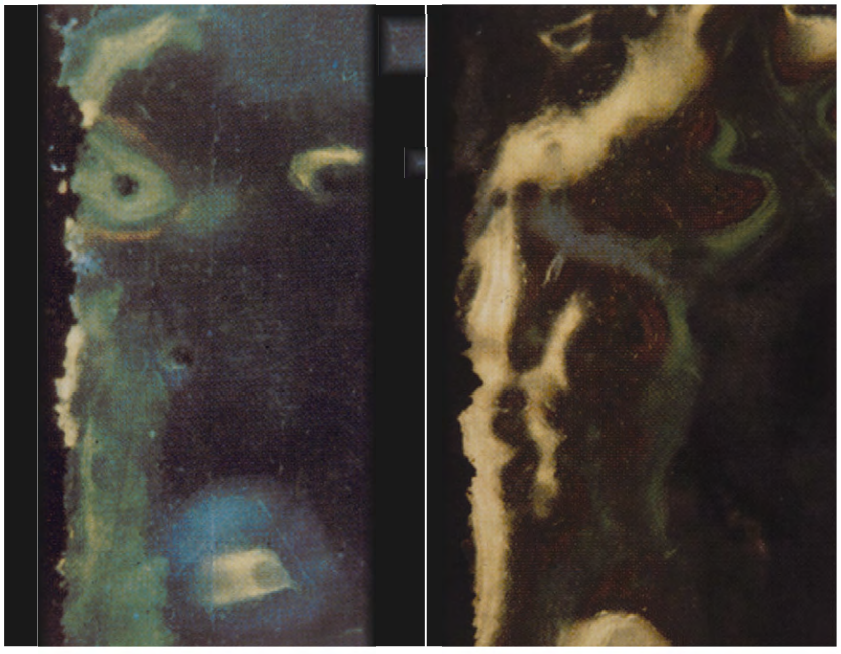


Figure 11-17 **A,** In this study of loaded implants in an animal (dog), axially loaded implants have lamellar bone more often at the interface. **B,** Implants with non-axial loading have primarily woven bone at the interface, a higher bone remodeling rate, and osteoclasts and inflammatory cells. *(From Barbier L, Schepers E: Adaptive bone remodeling around oral implants under axial and nonaxial loading conditions in the dog mandible,* Int J Oral Maxillofac Implants *12:215-223, 1997.)*

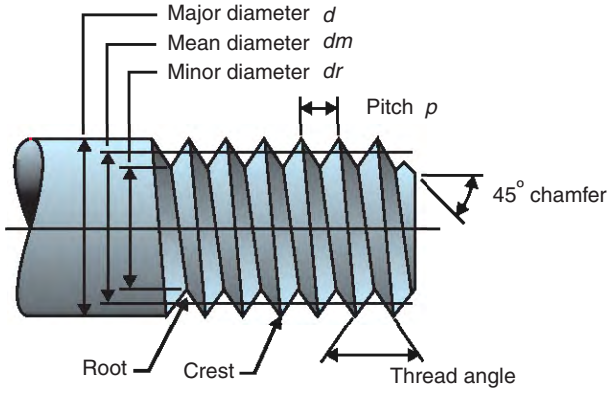


Figure 11-18 There are several parameters of an implant that may alter the functional surface area. Three of these include: thread pitch, thread shape, and thread depth.

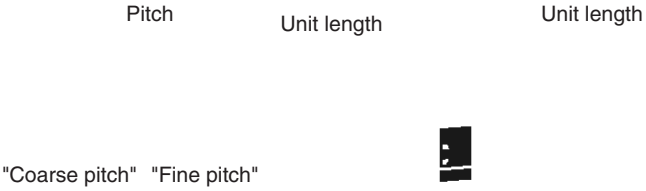


Figure 11-19 The thread pitch describes the number of threads per unit length of an implant. The implant on the right has a smaller thread pitch and greater surface area, whereas the implant on the left has the largest thread pitch and the least overall surface area.

three geometric thread parameters: thread pitch, thread shape, and thread depth (Figure 11-18).[43]

Thread Pitch

Thread pitch is the distance measured parallel between adjacent thread form features of an implant.[38] The height of the threaded portion of the implant body divided by the pitch equals the threads per unit length. The smaller (or finer) the pitch, the more threads on the implant body for a given unit length, and thus the greater surface area per unit length of the implant body if all other factors are equal. Restated, a decrease in the distance between threads will increase the number of threads per unit length. The greater number of threads, the greater the surface area, if all other factors are the same (Figure 11-19). Because stress is directly related to the magnitude of the force and indirectly related to the area over which the force is applied, the implant pitch may be made smaller when the magnitude of the force is greater than usual.[38]

Roberts observed in an animal model that the thread number may affect the bone implant contact percentage.[62] When two different implant designs were placed in the same animal, a higher BIC was observed with the implants with the greater thread number (Figure 11-20).

It is interesting to note that of all the design variables, pitch has the most significant effect on changing the surface area on a threaded implant. This is a major point to consider when looking at the anatomical dimensional limitations presented in the oral environment. For example, when an ideal implant length cannot be planned without advanced bone augmentation, an implant with greater thread numbers may improve the functional surface area for the height dimension compromise.[73]

WWW.HIGHDENT.IR

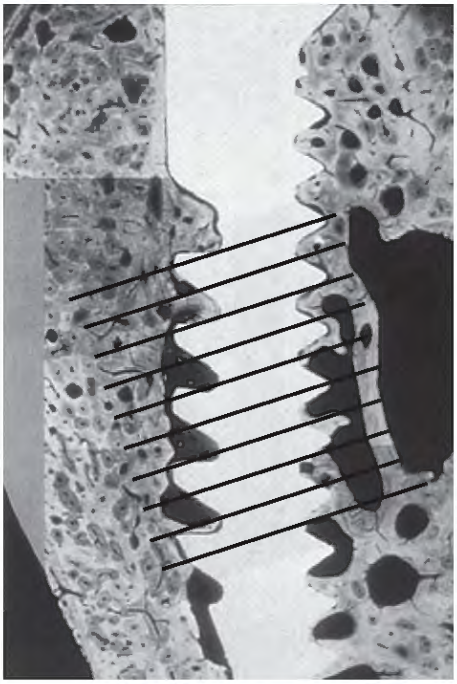


Figure 11-20 There is a higher bone-implant contact (BIC) with implants of greater thread number compared with implants with fewer threads. A composite of a typical result demonstrates a higher BIC on the right side compared with the left side of the implant. *(From Roberts WE, Smith RK, Zilerman Y et al: Osseons adaptation to continuous loading of rigid endosseous implants,* Am J Orthod *86:95-111, 1984.)*

The thread pitch may be used to help resist the forces to bone with poorer quality.[36] Because the softest bone types are 58% weaker than ideal bone quality, the implant thread number may be increased to increase the overall surface area and reduce the amount of stress to the weaker bone trabeculae. Therefore if force magnitude is increased, implant length is decreased, or bone density decreased, the thread pitch may be decreased to increase the thread number and increase the functional surface area.

Most manufacturers provide implant systems with a fixed pitch and fixed surface area per unit length, regardless of the character of forces or the bone density of the anatomical site. However, different popular implant designs often have different thread pitches. For example, the distance between the threads for one implant design is 1.5 mm (Straumann ITI), whereas a thread pitch of 0.6 mm exists for others (Zimmer Screw-Vent, Biomet 3I). Each implant pitch has a different number of threads per unit length and a different amount of functional surface area.

The thread number is most significant for the shorter length implants. For example, the Straumann ITI 6- and 8-mm-long implants may only have three threads to carry the compressive load (Figure 11-21). On the other hand, the thread pitch of other implant designs may feature 7 to 10 threads for a similar length (Figure 11-22). The greater the thread number, the greater the initial fixation and the greater the overall surface area after loading.

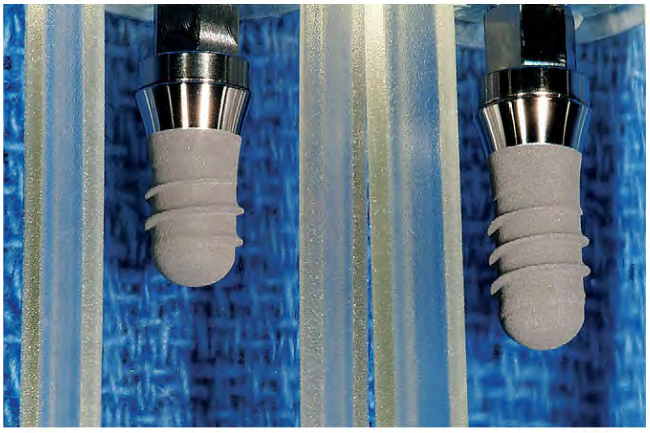


Figure 11-21 The Strauman ITI implant length on the left is 6 mm, and the one on the right is 8 mm. Each implant only has three threads for surgical fixation and load bearing after healing.

Figure 11-22 The BioHorizons Maestro implant on the left (7 mm long) has 7 threads. The implant on the right (9 mm) has 10 threads because of variable thread pitch designs.

The thread number may be affected by the implant crest module design. When the implant body has an extended smooth crest module, the number of the thread to support the occlusal load is reduced. For example, some implant designs have a smooth crest module of 3 mm or more, whereas other designs have only a 0.7-mm area above the first thread. When the thread pitch is 0.5 mm, there are four fewer threads for every 2 mm of smooth crest module. Therefore a 7-mm-long implant may have 28% fewer threads with an extended smooth crest module (Figure 11-23).

Several manufacturers advertise implant bodies with double or triple thread leads (e.g., Zimmer, Nobel Biocare). These terms relate to the manufacturing process and do not increase functional surface area. Rather than machining one thread at a time with one cutting instrument, a double thread uses two cutting blades and a triple thread uses three blades to manufacture the threads. As a result, when a one thread lead implant rotates 1 rpm, the implant inserts a distance of one thread. A double-thread implant at 1 rpm inserts two

WWW.HIGHDENT.IR

Figure 11-23 The internal hex implant design on the left (Zimmer ScrewVent) features an internal hex. The implant on the right (BioHorizons Maestro) has an external hex crest module. The implant on the left is 1 mm longer, but has fewer threads than the implant on the right. The implant on the left is manufactured with triple thread lead; the one on the right has a single thread lead. However, the overall surface area is not affected by the thread lead.

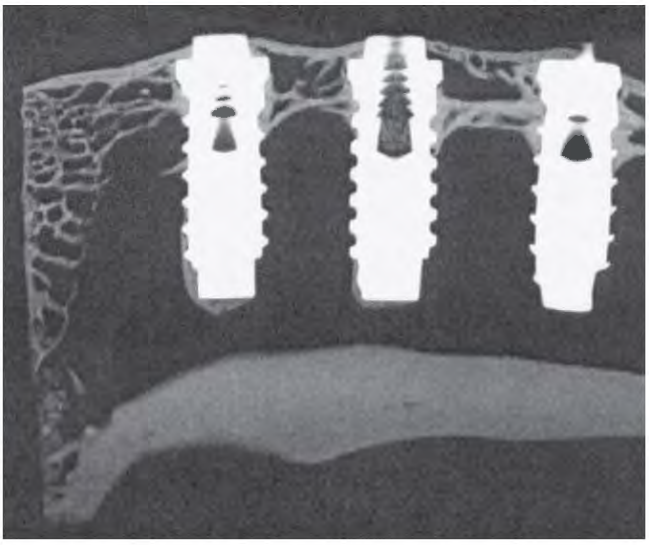


Figure 11-24 In this animal study, three different thread designs were evaluated for initial bone healing. The thread number, depth and surface condition (resorbable blast medium [RBM]) were kept identical. The implant length and diameter were also the same. The crest module designs of the implants were also similar.

threads into the bone. If the revolutions per minute on a single thread lead is doubled (e.g., 30 rpm versus 15 rpm), both implants thread into the bone at the same rate. In any case, no functional surface area change is found between a single- double-, or triple-thread lead.

The surgical ease of implant placement is related to thread number. The fewer the threads, the easier to insert the implant. If fewer threads are used in denser bone, the ease of placement is improved, because hard bone is more difficult to tap and insert a threaded implant.

Thread Shape

The thread shape is another important characteristic of overall thread geometry.[38] As described previously, thread shapes in dental implant designs include: square, V-shape, buttress, and reverse buttress (see Figure 11-10). In conventional engineering applications, the V-thread design is called a *fixture* and is primarily used for fixating metal parts together.[74] The reverse buttress thread shape was initially designed for pullout loads by Krupp.[74] This screw design was used to secure cannons to concrete bunkers so that the discharge forces during firing of the cannon would not pull the screws out of the foundation. The force transfer for occlusal loads to the bone is similar to that of the V-thread design. Dental implant applications dictate the need for a thread shape optimized for long-term function (load transmission) under occlusal, intrusive (the opposite of pullout) load directions. The square or power thread provides an optimized surface area for intrusive, compressive load transmission. Most automobile jacks or engineering designs built to bear a load use some form of a square design. Yet very few implant designs have incorporated a square thread design (BioHorizons, Ankylosis). A buttress thread shape may also load the bone with primarily a compressive load transfer (e.g., BioLok).

The thread shape has primarily design applications for loading conditions, but may also contribute to the initial healing stage for the direct bone interface. An animal study by Steigenga et al. compared three thread shapes with identical implant width, length, thread number, thread depth, and surface condition[75-77] (Figure 11-24).

Table 11-2 Percentage of Bone to Implant Comparing V-Shaped, Reverse Buttress, and Square-Threaded Implant Designs

N = 12 *n* = 69	V-SHAPED THREAD	REVERSE BUTTRESS	SQUARE THREAD	SIGNIFICANCE
Percent bone implant contact	65.46*	63.05†	74.37†	*$P < 0.01$ †$P < 0.001$

N, Number of rabbits; *n*, number of implants evaluated; *P*, significance value.
* V-shaped thread.
† Reverse buttress or square thread.
Comparisons were made using one-way analysis of variance with Bonferroni multiple comparisons test.

WWW.HIGHDENT.IR

Table 11-3 Reverse Torque Removal Values (N-cm) Comparing V-Shaped, Reverse Buttress, and Square-Threaded Implant Designs

N = 12 *n* = 36	V-SHAPED THREAD	REVERSE BUTTRESS	SQUARE THREAD	SIGNIFICANCE
Reverse torque value (N-cm)	15.58*	15.46†	23.17†	*†P < 0.05

N, Number of rabbits; *n*, number of implants evaluated; *P*, significance value.
* V-shaped thread.
† Reverse or square thread.
Comparisons were made using repeated measures analysis of variance with Bonferroni multiple comparisons test. F = 2.6723, P = 0.02 for overall model.

The V-shaped and reverse buttress thread shapes had similar BIC percent and similar reverse torque values to remove the implant after initial healing (Table 11-2). The square thread design had a higher BIC percent and a greater reverse torque test value (Table 11-3). Therefore it appears thread shape may also be a parameter in an implant design for the initial healing phase of osteointegration.

A consistent loading profile approach is warranted in implant dentistry. In general, materials are strongest under compressive loads and weakest to shear loads. This is true for porcelain, dental cements, implant materials, fixation screws, and bone.[36] Therefore a compressive force should be applied to all these components and shear force should be reduced wherever possible.

The face angle of the thread or plateau in an implant body can modify the direction of the occlusal load imposed on the prosthesis and abutment connection to a different direction at the bone interface. The face angle of a V-shaped thread is 30 degrees off the long axis, whereas a square thread may be perpendicular to the long axis. As a result, occlusal loads in the axial direction of an implant body may be compressive at the bone interface when the implant body incorporates square or plateau designs, but can be converted to higher shear loads at the bone interface when the implant body incorporates V-shaped threads (Figure 11-25).[38]

The shear force on a V-thread face that is 30 degrees (typical of Zimmer Screw-Vent and Biomet 3I) is approximately 10 times greater than the shear force on a square thread.[43] The shear component per unit length of a reverse buttress thread design is similar to a V-thread when subjected to an occlusal load. The shear component of a 15-degree face angle (Straumann ITI implant) is five times greater than the shear force on a square thread. The reduction in shear loading at the thread-bone interface provides for more compressive load transfer, which is particularly important in compromised bone density, short implant lengths, or higher force magnitudes.

A 30-degree load to an implant increases the force to the bone-implant interface by 50% compared with a long axis load, and the shear component of force is increased.[24] A 30-degree load to the implant-bone interface loads the bone interface at a 30-degree angle and also decreases the compression and tensile strength of the interface. The 30-degree load to bone decreases the bone strength in compression by 11% and decreases the tensile strength of bone by 26%.[24] Therefore, when a long axis load is delivered to the implant crown, the face angle of the implant body thread can modify the occlusal axial load to an angle bone-implant load. A power thread (square) may load the bone interface in compression when an axial load is delivered to the implant crown.

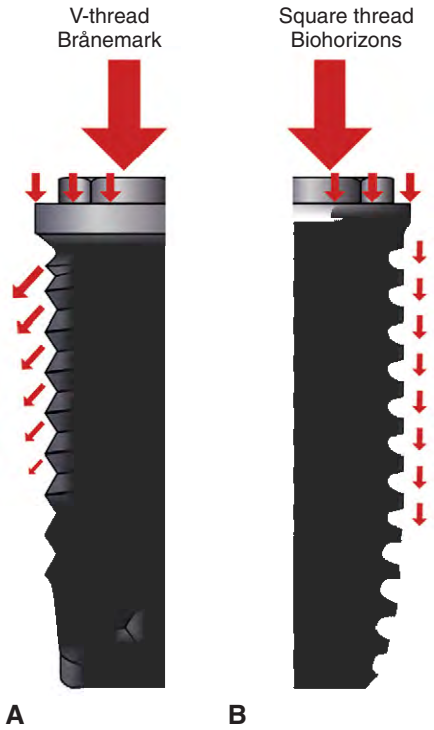


Figure 11-25 **A,** A long axis load to an implant body with V-thread with a 30-degree thread face converts the load direction to a 30-degree angle at the implant interface. An axial load on the prosthesis may result in an axial load to the implant platform. However, the implant body design may convert the primary compressive forces to the prosthesis and results in a 30-degree angled force with more tensile and shear loads to the implant-bone interface. **B,** A plateau or square-thread design can deliver a compressive force to the bone when the implant is loaded in the long axis. The implant body design determines the type of force transmitted to the implant-bone interface.

WWW.HIGHDENT.IR

IMPLANT TYPE	IMPLANT SIZE	SQUARE MILLIMETERS
BioHorizons (internal)	4 × 12 D3	206.5
BioHorizons (external)	4 × 12 D3	214.9
3I	4 × 13	210.4
3I	4 × 11.5	182.6
Replace Select	4 × 12	167.1
ITI Staumann	4 × 12	161.3

Figure 11-26 Evaluation of three thread type implants of similar diameter and length, thread number, and depth with a three-dimensional finite element analysis. The overall stresses and shear stresses are compared. The square-thread implant has less overall stress and less shear stress. The V-shape thread and reverse buttress thread shape are similar. *(From Kim WT, Cha YF, Oh SJ et al: The three dimensional finite element analysis of stress according to implant thread design under the axial load,* Korean J Oral Surg *27:3-8, 2001.)*

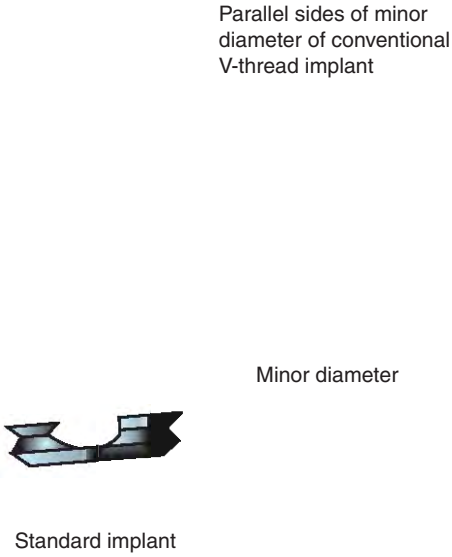


Figure 11-27 The thread depth of an implant refers to the distance between the outer (or major) diameter and the inner (or minor) diameter of the thread. The deeper the thread depth, the greater the functional surface area.

A finite element, three-dimensional study of a thread shape was evaluated by Kim et al.[78] An implant with the same number and depth of threads with different thread shapes (V-shaped, reverse buttress and square) were evaluated (Figure 11-26). The V-shape and reverse buttress had similar values. The square thread had less stress in both compressive and, more important, shear forces. Chun et al. also used finite element analysis to evaluate design parameters of osteointegrated dental implants.[79] They also concluded the square-thread design has a beneficial shape for occlusal loading compared with other thread designs.[14,80,81] Therefore thread shape may alter the functional load conditions and influence the type of force transmitted to bone. A review of the literature suggests that the square thread implant design may provide similar success rates in the maxilla and mandible in a wide range of differences in bone density.[14,71-73]

Thread Depth

The thread depth is the distance between the major and minor diameter of the thread (Figure 11-27).[38] Conventional implants provide a uniform thread depth throughout the length of the implant. A straight minor diameter, which is used in almost every screw-type dental implant, results in uniform cross-sectional area throughout a parallel-walled implant length. A tapered implant often has a similar minor diameter, but the outer diameter decreases in relationship to the taper, so the thread depth decreases toward the apical region. As a result, this implant design has overall less surface area, which is more critical in shorter implant lengths. The tapered, threaded implant may have less ability to fixate the bone in the apical region at initial insertion and has less functional surface area. The implant body taper may result in higher stresses, especially in shorter implant lengths.[27]

The greater the thread depth, the greater the surface area of the implant, if all other factors are equal. Different manufacturers use different thread depths. Some threaded implants have a 0.24-mm thread depth (Nobel Replace), the thread depth of Straumann ITI is 0.3 mm, and the thread depth of many V-shape threads is 0.375 mm (Biomet 3i and Zimmer Screw-Vent). The square thread of the 4-mm-diameter BioHorizons implant body has a 0.42-mm thread depth. Therefore, if all other factors were equal, each type of implant in these examples would have a different functional surface area directly related to the depth of the thread, with BioHorizons having the most surface area and Nobel Replace the least (Table 11-4).

WWW.HIGHDENT.IR

Figure 11-28 The BioHorizons implant on the left is 4 mm in diameter and has a thread depth of 0.42 mm. The center and right implants are 5 and 6 mm in diameter, respectively, and have a thread depth of 0.78 mm. As a result, the surface area of the two implants on the right increase by implant diameter and thread depth and have more than 200% more surface area than the implant on the left.

The more shallow the thread depths, the easier it is to thread the implant in dense bone, and the less likely bone tapping is required prior to implant insertion. Because implant surgeons often decide what implant they will insert based on ease of surgical insertion, it is not unusual that an implant with fewer threads and less deep threads are selected, because both conditions facilitate insertion. However, after the implant is placed into the bone, the conditions that make implant surgical insertion easier create less functional surface area, and increase the risk of occlusal overload to the bone-implant interface.

In the conventional implant design, the implant body design remains identical, regardless of the implant diameter. As a consequence, the implant increases in surface area by 15% to 25% for every 1-mm increase in diameter.[6] However, as the implant becomes wider, the depth of the thread may be deeper without decreasing the body wall thickness between the inner diameter and the abutment screw space within the implant. Therefore the thread depth may be modified relative to the diameter of the implant, and thereby the overall surface area may be increased by 150% for every 1-mm-diameter increase (Figure 11-28). For example, the Biomet 3I 4.0-mm-diameter implant body has 200-mm^2 surface area, and the 5.5-mm implant has 245-mm^2 surface area (when the implant is 12 mm long). The 4.0-mm-diameter BioHorizons implant has a 215-mm^2 surface area and the 5-mm implant has a 356-mm^2 surface area.[27,31]

The overall functional surface area of an implant body is therefore related to the thread pitch, thread shape, and thread depth. Tables 11-4 and 11-5 illustrate the difference in several popular implant designs when the implant body is 12 mm long. Note the difference between the 4- and 5-mm-diameter implants. It is interesting to note the molars of natural teeth have 200% more surface area than the premolars. Natural teeth not only increase their diameters, they also modify their design to cope with the higher bite forces in the posterior regions of the mouth. Yet most implant designs only increase surface area by 10% to 30% with a larger-diameter implant body.

Table 11-5 Surface Area of Implant Designs (5mm in Diameter)

IMPLANT TYPE	IMPLANT SIZE	SQUARE MILLIMETERS
BioHorizons (external)	5 × 12 D3	356.2
3I	5 × 13	267.1
3I	5 × 11.5	231.7
Replace select	5 × 12	193.6
ITI Straumann	5 × 12	207.1

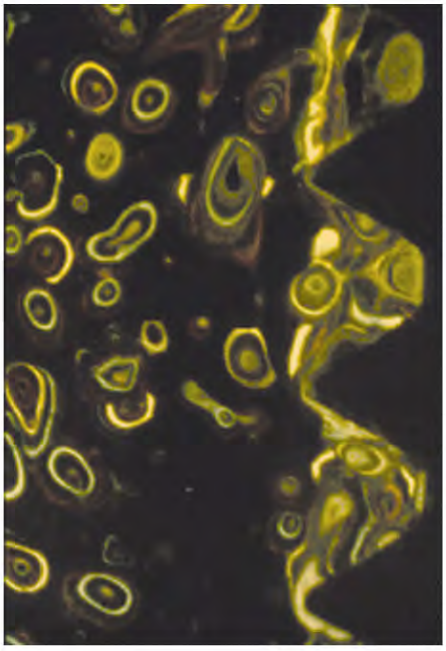


Figure 11-29 In an animal model (dog) the remodeling rate of bone away from the implant was 40% per year (physiologic or adapted window). The bone remodeling rate next to the Brånemark implant (Nobel Biocare) was 500% per year (mild overload zone). *(From Garetto LP, Chen J, Parr JA et al: Remodeling dynamics of bone supporting rigidly fixed titanium implants: a histomorphometric comparison in four species including humans,* Implant Dent *4:235-243, 1995).*

Bioengineering of an Implant Design

Recent studies have shown that when implants act as functionary unit for a prosthesis, an elevated BRR is an ongoing response adjacent to many dental implants.[82] A BRR higher than 500% per year in the bone immediately

WWW.HIGHDENT.IR

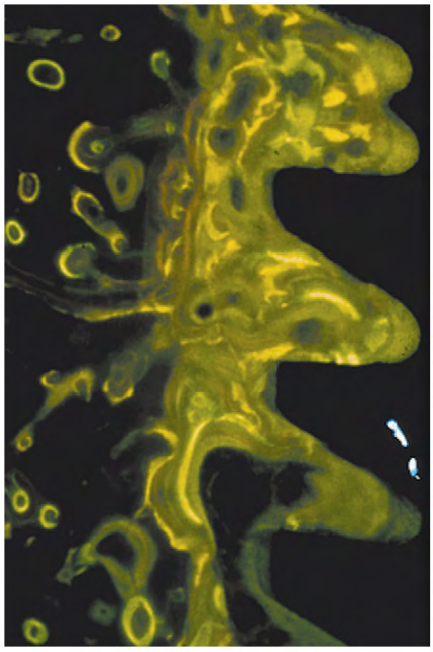


Figure 11-30 Garetto et al. evaluated in an animal model (dog) the bone remodeling rate (BRR) next to a SteriOss dental implant (Nobel Biocare). The bone several millimeters away from the implant had a BRR of 40% per year. The region around the implant had a BRR of 680% per year (mild to pathologic overload). As a result, the SteriOss dental implant had a smaller percentage of bone-implant contact compared with the Brånemark implant design. The higher the BRR, the less lamellar bone and the closer to the pathologic overload zone, which may cause formation of fibrous tissue. *(From Garetto LP, Chen J, Parr JA et al: Remodeling dynamics of bone supporting rigidly fixed titanium implants: a histomorphometric comparison in four species including humans,* Implant Dent *4:235-243, 1995.)*

adjacent (within 5 mm) to a V-shape threaded implant, but approximately 50% in the regions distant from the interface has been observed. These findings suggest the bone at the interface of the implants in their report is likely in the mild overload zone (Figure 11-29). Cooper presented a histologic report of Roberts, in 1999, which evaluated the turnover rate of bone next to a macrosphere surface design (Sybron Corp., Endopore) implant retrieved from a human maxilla in the premolar region.[83] This implant had a BRR that ranged between 400% and 908% per year. It is interesting to note the developers of this implant design report that bone loss is often a consequence of stress shielding as in orthopedic hip prostheses as determined by finite element analysis, rather than overload conditions. Therefore they believe more stress should be applied to the implant interface. The BRR, at least in this one reported patient, appears to indicate a greater strain condition does exist with this design, compared with other implants or conditions that have been reported.

Roberts et al. observed a difference in interface contact percentage between an asymmetrical implant thread design (Nobel Biocare, SteriOss, Yorba Linda, Calif.) and a symmetrical threaded surface (Nobel BioCare, Brånemark, Gothenburg, Sweden).[84] The V-shaped thread of Brånemark had a higher bone contact and reduced bone turnover rate (500%) compared with the reverse buttress thread shape with a reduced thread number (Steri-Oss implant with 680% BRR) (Figure 11-30). Therefore the BRR reported is different in each of these three different designed implants.

If an implant design is bioengineered so that loading will produce a microstrain within the adapted window zone, it should maintain lamellar bone at the interface during loading, as represented by a similar BRR adjacent to and distal to the implant interface. Therefore a prospective goal of an implant design project involved modifying the parameters that affect the response of the surrounding bone tissue next to the implant at the cellular level.[85]

The magnitude of load may also affect the BRR at the implant interface. Roberts evaluated successful implants in humans, used for orthodontic anchorage for 3 years or more.[86] The bone-implant interface of these implants only remodeled at a rate in the range of 30% per year. The duration of an orthodontic force is constant, and the forces used for tooth movement (<5 N) is far inferior to the typical forces of function or parafunction (up to 250 N). As a result, the lower bone turnover rate may signify the small-magnitude tensile loads at the implant interface during orthodontic anchorage. Brunski et al.[87] used a dog model in which overloads were applied to a titanium V-shaped thread screw dental implant. Implants in the mandibles and radii were allowed to heal for 4 to 7 months and then loaded with cyclical axial compression in the mandibles and axial tension on the radii sites. There were no significant differences between the controls and loaded interfaces. Brunski et al. believe this was most likely a result of insufficient levels of loading to observe a difference. The similar result may also be related to the thread shape, which gives similar stress profiles in compression and tension. A follow-up study by Hoshaw et al.[71] using dog tibiae and larger axial tensile loads with a longer healing time before loading found more crestal bone loss around the loaded implants, an elevated BRR in the cortex, and resorptive modeling on the periosteal surface. There are several studies and reports that demonstrate prosthetic loading conditions on the implants can cause implant failure, crestal bone loss, or implant fracture.[88] It is therefore hypothesized the phenomenon of the elevated BRR at the implant interface, compared with that found several millimeters away, may be used as an indication of increased biomechanical risk for the supporting implant-bone interface, as related or created by specific clinical conditions.[64]

To reduce shear loading to bone, a square-thread design to axial loading may be used on the body of the dental implant.[77,78] The turnover rate of bone within the threads of the BioHorizons Maestro D2 and D3 implants in both a dog study and human case report has been reported to be 40% to 50% and was the same

WWW.HIGHDENT.IR

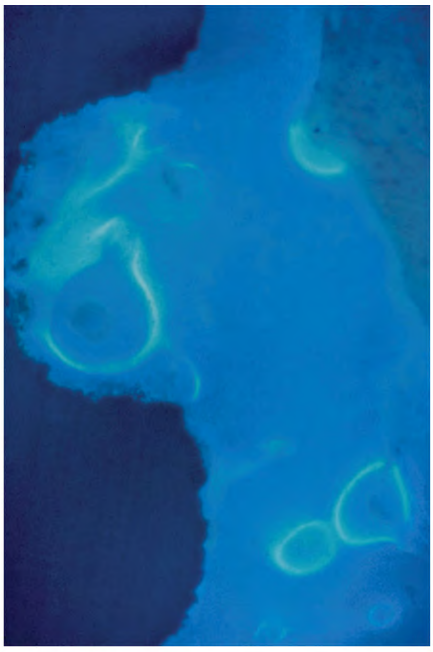


Figure 11-31 A square-threaded implant was loaded in an animal model. A bone remodeling rate of 50% per year was observed, which was similar to the remodeling rate several millimeters from the implant interface.

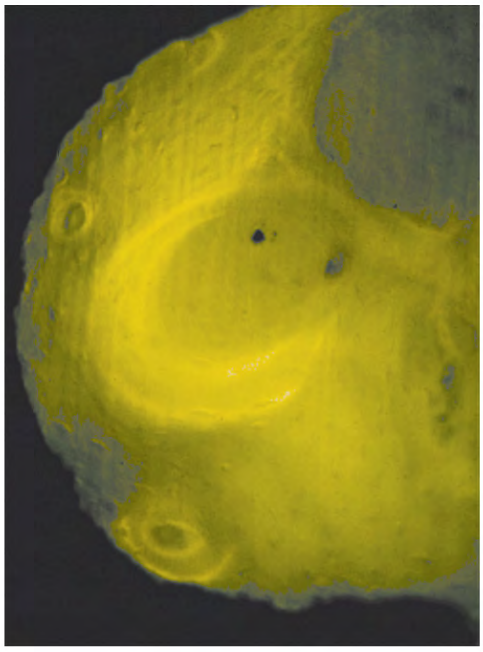


Figure 11-32 The bone remodeling rate of bilateral maxillary first premolar implants with a square thread design was evaluated after 1 year of occlusal loading in a human. The bone remodeling rate was 40% to 50% per year and similar to the bone away from the implant interface.

as observed in the bone away from the implant interface (Figures 11-31 and 11-32).[46,64] This rate corresponds to the rate of lamellar bone remodeling in the adapted window zone. This condition may place the interface at less biomechanical risk, because lamellar bone is more mineralized, more rigid, and stronger compared with the reactive woven bone found in the mild overload zone.

The concept presented in this chapter is not meant to suggest adequate strain levels may only be obtained with one implant design, or even that ideal strain levels are necessary for long-term maintenance of osteointegration. The strain environment around an endosteal implant is very complex, and variables such as bone density, bone volume, and bone shape are most likely influential, but not yet fully understood. Future experimental models should evaluate how the influence of implant design, surface condition, intensity of load, frequency of load, direction of force, and bone density interrelate to the long-term success of dental implants. It is suggested the bone turnover rate may be a method to evaluate these conditions.

Crest Module Considerations

The crest module of an implant body is the transosteal region, which extends from the implant body and often incorporates the antirotation components of the abutment implant connection. The crest module of the implant has a surgical influence, a biological width influence, a loading profile consideration (characterized as a region of highly concentrated mechanical stress), and a prosthetic influence. Therefore this area of the implant body is a determinant for the overall implant body design.

During the surgical phase the crest module design primarily benefits the crestal implant interface. The crest module of an implant should be slightly larger than the outer thread diameter of the implant body. In this way, the crest module seals completely the osteotomy, providing a barrier and deterrent for the ingress of bacteria or fibrous tissue during initial healing.[43] The seal created by the larger crest module also provides for greater initial stability of the implant following placement, especially in softer unprepared bone, because it compresses the crestal bone region.[43]

The larger crest module diameter also increases surface area, which can further decrease stress at the crestal region. A crest module height of 2 mm, which is 4.2 mm compared with 4.0 mm, has 5% greater surface area ($\pi \times$ diameter).[6] Because the stresses are highest in this region, the greater surface area decreases stress to the bone and increases the strength of the implant body.

The increase in crest module diameter increases the platform of the abutment connection with a stress reduction to the abutment screw during lateral loading. In fact, the platform dimension is more critical to reduce the stress applied to the abutment screw than is the height (or depth) of the antirotational hex of the abutment to implant body connection.[89]

The concept of designing an implant crest module with a smooth collar is for a reduction of plaque accumulation and improved hygiene (Figure 11-33). However, the crest module is initially placed below the bone in

WWW.HIGHDENT.IR

Figure 11-33 Many implants have a crest module with a smooth metal collar for reduction of plaque accumulation and improved oral hygiene conditions.

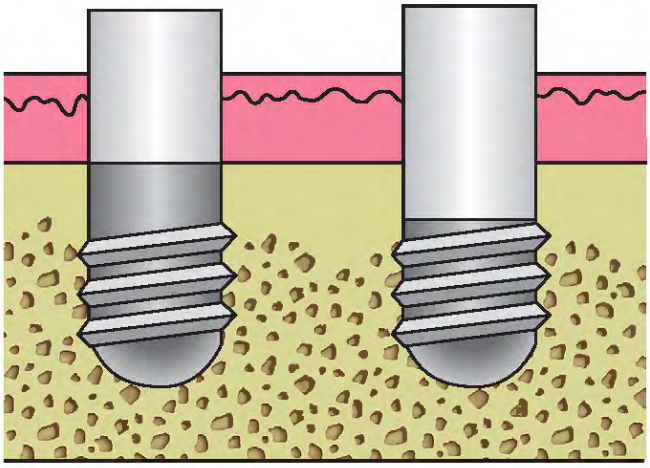


Figure 11-34 Herman evaluated the initial unloaded healing of 12 one-piece transgingival implant designs in dogs. The implant on the left had roughened metal placed at the crestal region. The implant on the right had a smooth metal collar of 1.5 mm placed below the crestal bone.

most two-piece implant designs. Therefore the need for daily hygiene and plaque control is not relevant, unless crestal bone loss occurs. The initial sulcular soft tissue thickness approximates 3 mm above the bone. A toothbrush bristle only extends 0.5 to 1 mm into the sulcus during oral hygiene procedures. Therefore unless the tissue recedes, even when bone loss occurs on the implant crest module, the gingival sulcus depth cannot be accessed by hygiene procedures after crestal bone loss, especially because the gingival sulcus increases in depth after marginal bone loss.

There are at least six causes of marginal bone loss at the crestal bone region of implants, including the formation of a "biological width" and occlusal overload after the implant is in function.[90,91] The biological width of an implant is related to marginal bone loss before, or separate from, occlusal loading influences. Both these entities have increased bone loss risk when smooth metal is placed below the bone. In an animal study, Herrman placed one-piece implants with a 1.5-mm smooth crest module below the bone and compared it with a roughened crest module of similar design at the bone crestal level in dogs (Figures 11-34 and 11-35).[92] The two implant designs were followed for 6 months, with no occlusal load. Within 1 month after the implant insertion and extension through the soft tissue, marginal bone loss occurred to the smooth region 1.5 mm below the crestal region. No bone loss occurred when the implant crest module was rough and placed at the level of the crestal bone. Hammerle et al. inserted similar implant designs in human patients and found a similar

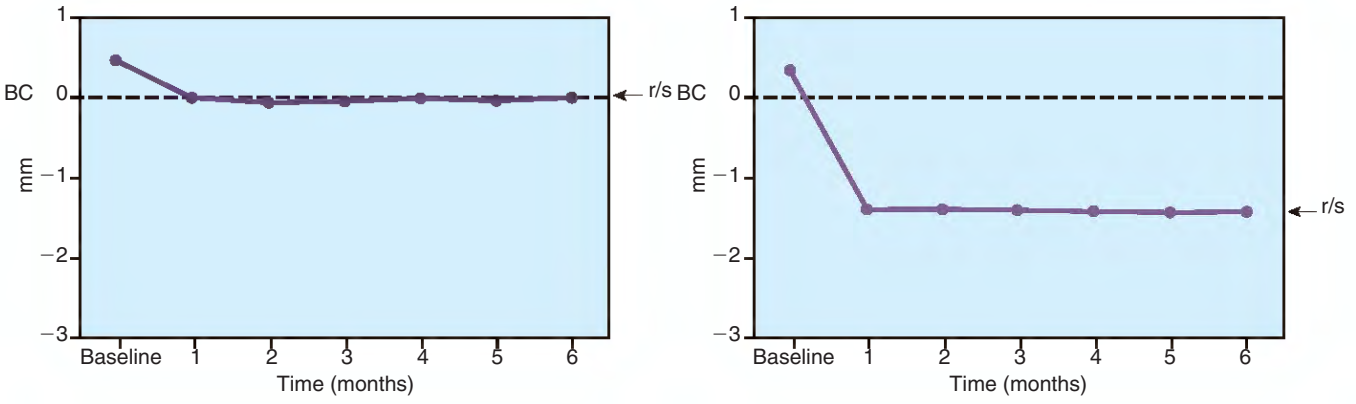


Figure 11-35 **A,** The implants with a 1.5-mm smooth metal collar below the bone lost 1.5 mm of bone to the first month during an unloaded 6 month period. **B,** The implants with a rough surface at the crestal bond lost no bone during an unloaded 6-month period.

WWW.HIGHDENT.IR

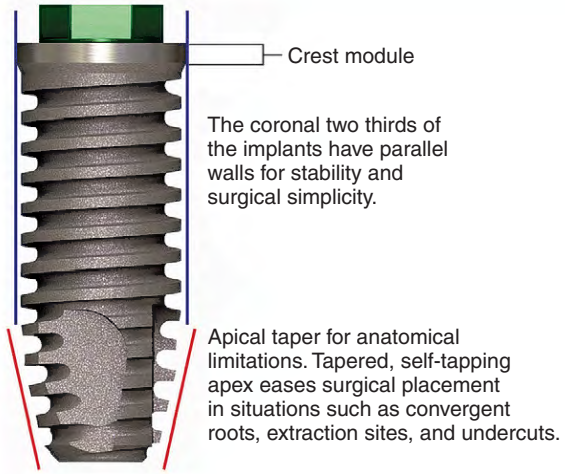


Figure 11-36 A 0.5-mm smooth metal collar may provide for a closer abutment-crest module connection compared with a rough surface connection. The apical end of an implant is often tapered to ease surgical placement, because it fits within the osteotomy prior to engaging the walls of the bone.

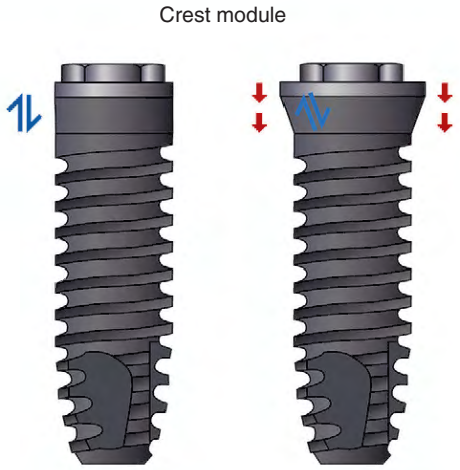


Figure 11-37 The crest module with a cylinder metal collar transfers primarily shear forces to the bone *(far left).*

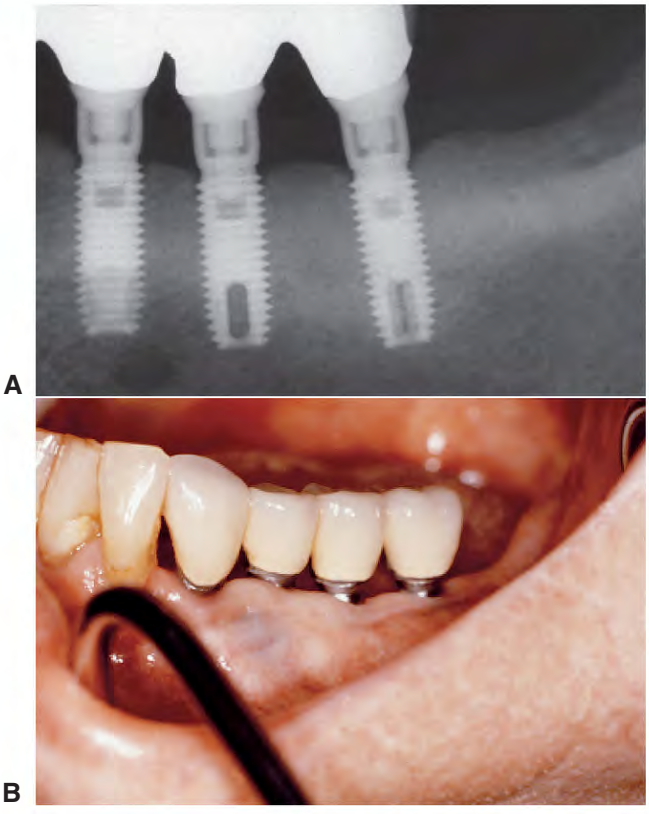


Figure 11-38 **A,** These three implants with an acid-etched collar were placed at the crest of the bone. After 2 years of occlusal loading, the bone has been lost to the first thread on all three implants. **B,** The clinical appearance of 3.5 mm of bone loss in this patient was tissue shrinkage and exposure of the implant crest modules.

crestal relationship to smooth metal below bone.[16] Hanggi et al. also compared two different crest module designs in patients and found the longer smooth metal collar had more crestal bone loss.[93] Therefore the crest module of the implant should not have an extended smooth area placed below the bone.

A biological width of at least 0.5 mm has been reported apical to the abutment-to-implant connection regardless of the implant crest module design. A 0.5-mm collar length may provide for a desirable smooth surface close to the perigingival area, while preserving the biomechanical performance of the remaining portion of the crest module (Figure 11-36). The microgap between the implant crest module and abutment may also be reduced when polished surfaces are approximated.

The next consideration of the crest module is related to occlusal loading.[17,34,91,94] Most of the occlusal stress occurs at the crestal region of an implant design.[91,94] A smooth, parallel-sided crest module will increase the risk of bone loss after loading (Figure 11-37). As previously discussed, smooth metal promotes shear stresses in the adjacent bone interface.[90] Because bone is 65% weaker to shear loads, attempts to limit shear are prudent. In addition, smooth metal does not encourage bone cell contact before loading.[15] When the surface condition of the implant is roughed, the BIC is increased during initial healing. Reverse torque tests observe consistent higher values when roughened surfaces are compared with machined or polished surface conditions.[95] Significant loss of crestal bone has been reported for implants with machined (smooth) coronal regions, and the amount of the bone loss is directly related to the length of the smooth crest module (Figure 11-38).[96] This bone loss is attributed to the biological width before loading and the lack of effective mechanical loading between the machined coronal region of the implant and the surrounding bone after occlusal loading. This clinical problem is reduced by using implants that incorporate a roughened surface and a crest module designed to reduce shear loading.

WWW.HIGHDENT.IR

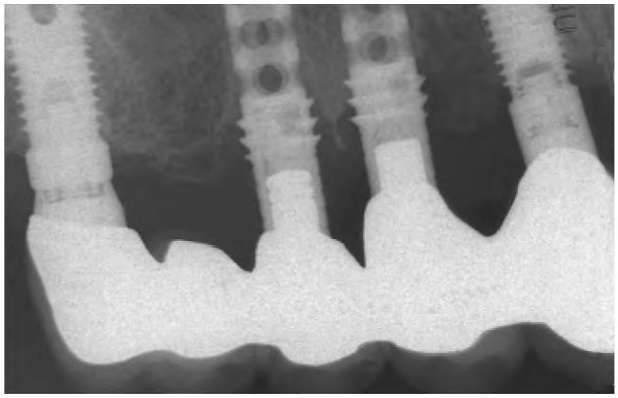


Figure 11-39 The amount of crestal bone loss is often related to the implant design. In this five-unit fixed partial denture, there are three different implant designs. The implant on the far left has a 2-mm smooth collar and a hydroxylapatite-coated thread. The two center implants have a sharp buttress threaded acid-etched cylinder smooth metal collar. The implant on the far right has a 4.5-mm smooth collar above the first thread.

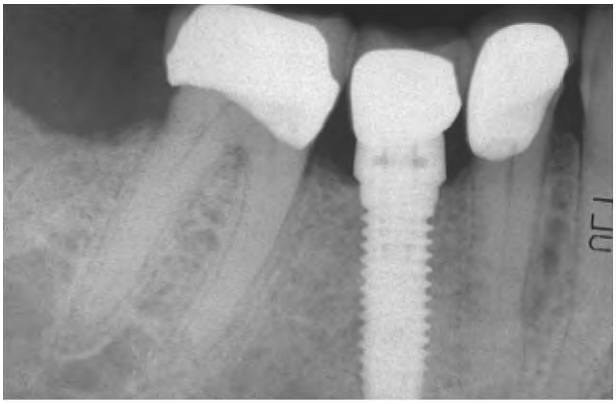


Figure 11-40 Implants with a biomechanical design to reduce crestal stresses often have less crestal bone loss in clinical reports (i.e., BioHorizons Maestro Dental Implant).

A roughened surface (but a shear load design crest module) maintains the bone through the biological width cycle, but may lose the marginal bone during the occlusal load conditions. For example Straumann ITI has a roughened crest module, which is a cylinder below the bone. The initial bone loss before loading is minimal. However, a clinical report on human patients demonstrated 26% of the implants[5] lost more than 5 mm of bone and 22% lost more than 6 mm during a 10-year prospective study[22] (see Table 1-1). Astrand et al. compared ITI and Brånemark implants in partially edentulous maxillae. The roughened surface ITI had more bone loss than the machined metal Brånemark implants (see Figure 11-3).[23] DeBruyn et al. followed clinical outcomes of screw-vent implants (with an internal hex and long rough cylinder crest module). Crestal bone loss was consistently found at least to the first thread, which accounted for more than 2 mm of bone loss.[97] In other words, a rough crest module may not be sufficient to stop crestal bone loss once the implant is loaded.

It has been a common clinical observation that bone is often lost to the first thread after loading, regardless of the manufacturer type or design. Bone often grows above the threads during healing, but after prosthesis loading, bone loss is often observed. For example, the first thread is 1.2 mm below the platform of the Nobel Biocare Mark IV implant, 2 mm below the platform on the Nobel Replace design, and 3.5 mm on internal hex Zimmer implant designs. The magnitude of the crestal bone loss is often directly related to the distance between the crest module and the first thread distance, with the most bone loss on the internal hex crest module design with an extended cylinder crest module (regardless of whether it is smooth or rough) (Figure 11-39). Therefore the bone loss cannot be related to a specific biological width, but may be in part caused by crest module design. The bone loss often stops at the first thread because the first thread changes the shear load created by the crest module to a component of compressive loading. Instead of designing the crest module for shear, an improved design can reduce the crestal bone loss risk.

Any crest module design that incorporates an angled geometry or grooves to the crest module, coupled with a surface texture that increases bone contact, will impose a beneficial compressive component to the contiguous bone and decrease the risk of bone loss. By way of example, implants designed with stress transfer features (i.e., Astra, BioHorizons) often exhibit less bone loss in clinical reports (Figure 11-40).[14,80] This is not a coincidence, but rather is a design feature that influences the end result.

The prosthetic features of the crest module may affect the implant design. For example, in an internal hex implant, the antirotational feature of the abutment is designed within the implant body. As a result, the implant body is lower in profile and easier to cover with soft tissue during surgery. In addition, the antirotational feature is often deeper within the body compared with external hex implants. However, because the internal antirotation feature is wider than an abutment screw, the wider body diameter at the crest module is reduced. As a result, the threads on the outside of the implant body can not be designed at or above the antirotational feature of the implants. Therefore greater smooth metal and shear forces are observed above the first implant body thread compared with an implant with an external hex (Figure 11-41). The threads may progress more crestally with the external hex, because the abutment screw diameter is narrower and the outer body wall is thicker; therefore the threads may approach the crestal region of the implant.

Apical Design Considerations

The apical portion of a root form implant is most often tapered to permit the implant to seat within the

WWW.HIGHDENT.IR

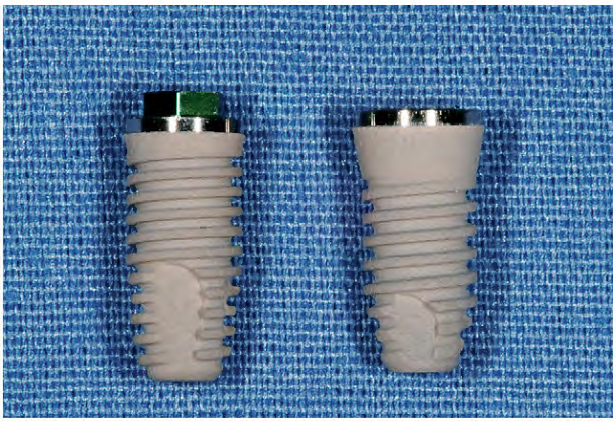


Figure 11-41 The implant on the left has an external hex; the implant on the right has an internal hex (BioHorizons Dental Implants). The external hex designs allow the fabrication of threads closer to the crestal region of the implant.

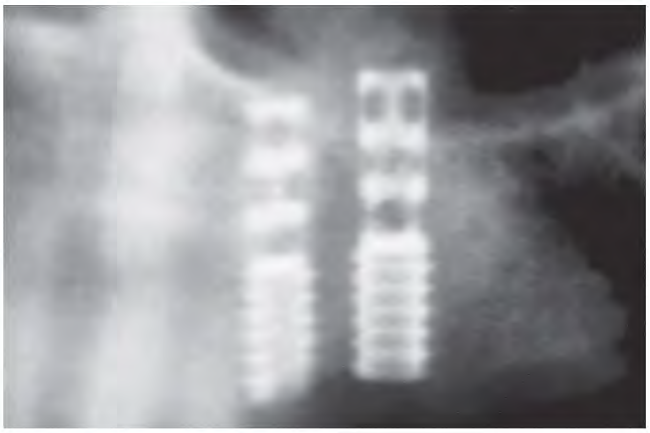


Figure 11-42 The apical hole or open cylinder may fill with mucus when placed within a sinus and result in a retrograde infection around the implant.

osteotomy before the implant body engages the crestal bone region (see Figure 11-36, *B*). As a result, the patient does not need to open the mouth as wide, which is especially of benefit in the posterior regions of dentate patients. This apical feature favors the initial step of implant insertion.

Most root form implants are circular in cross section. This permits a round drill to prepare a round hole, precisely corresponding to the implant body. Round cross sections, however, do not resist torsion/shear forces when abutment screws are tightened or when freestanding, single-tooth implants receive a rotational (torsional) force. As a result, an antirotational feature is incorporated into the implant body, usually in the apical region. The most common design is a hole or vent. In theory, bone can grow through the apical hole and resist torsional loads applied to the implant. The apical hole region may also increase the surface area available to transmit compressive loads to the bone.

A disadvantage of the apical hole occurs when the implant is placed through the sinus floor or becomes exposed through a cortical plate. The apical hole may fill with mucus and become a source of retrograde contamination (Figure 11-42) or will likely fill with fibrous tissue. If the apical hole is several millimeters in height, the region filled with fibrous tissue decreases bony contact in the apical region of the implant. This concern is greatest with an open basket body design, less with a vertical hole of 4 mm, and even less with a round 1-mm hole. The apical aspect of a solid implant (without apical hole) may slightly perforate any opposing cortical plate and act as a wedge to seal the opening.

Another antirotational feature of an implant body may be flat sides or grooves along the body or apical region of the implant body. Bone grows against the flat or grooved regions and helps resist torsional loading. In addition, the grooves or recessed areas of the apical portion of the implant help to enhance the "self-tapping" aspect of an implant design. This occurs in several ways. First, the recess areas of the apical portion allow bone fillings from the cutting threads to fill the area. Otherwise, these bone chips may fall to the apical floor and prevent the implant from completely seating or compress into the trabecular bone and contribute to a pressure or resistance to rotational insertion of the implant. Second, the recess area may be designed to decrease the angle of the cutting thread along the apical portion of the implant. As a result, less torque is required to thread the implant into the bone.

The apical end of each implant should be flat rather than pointed. Pointed geometry has less surface area, thereby raising the stress level in that region of bone. Additionally, if an opposing cortical plate is perforated, a sharp, V-shaped apex may irritate or inflame the soft tissues if any movement occurs (e.g., the inferior border of the mandible).

IMPLANT BODY BIOMATERIAL RELATED TO FRACTURE

Implant bodies and components have a risk of fracture, most often during the intermediate to long-term loading condition. According to Goodacre et al., the risk of implant body fracture in the early to intermediate period for implants 3.75 mm in diameter is 1%, the abutment screw fracture risk is 2%, and the prosthetic screw risk is 4%.[18] However, the incidence of fracture dramatically increases when force conditions are greater. Cantilevers, angled loads, and parafunction increase the risk of fracture. The risk of fracture also increases over time. Rangert reported that in the long-term loading condition, 80% of all failures may be related to implant body fracture.[98] The elements of the implant body that influence the fracture risk include the biomaterial, the size, and the design.

WWW.HIGHDENT.IR

Force Magnitude and Implant Body Design

Normal physiology imposes constraints on the magnitude of forces that must be withstood by engineering designs in the oral environment. The magnitude of bite force varies as a function of anatomical region and state of the dentition. Average bite forces can range from 10 to 350 lb.[99] The magnitude of force is greater in the molar region (200 lb), less in the canine area (100 lb), and least in the anterior incisor region (25 to 35 lb). These average bite forces increase with parafunction to magnitudes that may approach 1000 lb in the posterior regions.[100]

Many biocompatible materials are unable to withstand the type and magnitude of parafunctional loads that may be imposed on dental implants. As an example, ceramic, which has excellent biocompatibility, is very susceptible to tension and bending loads. Such loads are commonly applied to dental implants and render this material unsuitable in many implant body applications. In addition, materials such as HA are quite biocompatible with biological tissues, but lack the mechanical material properties to withstand the loads imparted on implants. In contemporary applications, many of these materials are considered for use as a coating when applied to a stronger substrate material.

Titanium and titanium alloys have a long history of successful use in dental and orthopedic applications. The excellent biocompatibility of titanium and its alloy has been well documented.[101] There are four grades of CP titanium used in dentistry (grades I to IV), and one grade of titanium alloy (grade V). Regardless of the grade of titanium or alloy, the surface condition to the bone is similar (i.e., titanium oxide). With its highly active titanium oxide layer, these materials are extremely well tolerated by local tissues. Titanium-aluminum-vanadium alloy (Ti-6Al-4V) has been shown to exhibit the most attractive combination of mechanical and physical properties, corrosion resistance, and general biocompatibility of all metallic biomaterials.[101,102] The primary advantage of titanium alloy as compared with other grades of titanium is its strength. As shown in Table 11-6, the mechanical properties of titanium alloy are superior to those of commercially pure titanium.[103-105] Titanium alloy is four times stronger than grade 1 CP-titanium and almost twice as strong as grade 4. Ultimate strength and fatigue strength is a primary consideration given the ramifications of the loading profiles to which dental implant bodies are subjected and the possible component fractures incurred due to inferior materials or designs.

The modulus of elasticity (stiffness or rigidity) of the four different grades of titanium is similar (103 GPa), and titanium alloy is only slightly higher (113 GPa). Although there is a significant difference in strength between grades of CP titanium and its alloy, the elastic modulus is similar for all of these materials. Titanium and its alloy represent the closest approximation to the stiffness of bone of any surgical grade metal used as an artificial replacement for skeletal tissue, even though it is almost 6 times stiffer than dense cortical bone. However, because of the geometry and functional requirements of dental implants, the elastic modulus is not nearly as important as the biocompatibility and strength offered by titanium alloy. Thus titanium alloy represents the best compromise (given current biomaterials technology) between biomechanical strength, biocompatibility, and the potential for relative motion (from modulus mismatch) at the bone-implant interface.

Implant Design Failures Related to Biomaterial and Force Magnitude

Two examples of implant body failures related to biomaterial choice have appeared in the historical implant literature. The vitreous carbon implants optimized the modulus of elasticity (stiffness) of the biomaterial (carbon) without appropriate attention to ultimate strength considerations. Conversely, Al_2O_3 ceramic implants optimized ultimate strength without adequate attention to modulus of elasticity or type of loading conditions placed on implants in parafunction.

The vitreous carbon implant design was composed of a smooth, tapered-step carbon body with an internal 316-L stainless steel post. The stiffness of the carbon was compatible with the surrounding bone; however, the carbon body was incapable of withstanding the physiologic loads within the oral environment.[106,107] The geometric design of the implant is equally, if not more important to resist occlusal loads. Microcracks in the body developed, and a pathway of biological fluids was thus introduced to the internal stainless steel post. The post was then subjected to dramatic corrosion with the subsequent release of metallic ions

Table 11-6 Mechanical Properties for Different Grades of Titanium

PROPERTY	GRADE 1	GRADE 2	GRADE 3	GRADE 4	TI-6A1-4V
Tensile strength, min (MPa)	240	345	450	550	930
Yield strength, 0.2% offset, min (MPa)	170	275	380	483	860
Modulus of elasticity (GPa)	103	103	103	103	113

Grades 1 to 4, CP titanium; *Ti-6Al-4V,* titanium-aluminum-vanadium alloy.

WWW.HIGHDENT.IR

into the interfacial tissues. Gross tissue inflammation with ultimate implant failure and removal resulted. A close match of biomaterial and bone material stiffness alone cannot, in isolation, provide clinical success.

The ceramic implants, as a class, were antithetical to the carbon implants. Ultimate compressive strength was optimized at the sacrifice of matching biomaterial and bone stiffness. In addition, the brittle nature of ceramic and susceptibility of failure in tension and shear require geometric designs of the implant that may not be compatible with the anatomical dimensional limitations (i.e., available bone width and height) of the jaw. The modulus of elasticity for ceramics is approximately 33 times stiffer than cortical bone.[108] The result was apparent stress shielding of the interfacial bone. Bone must receive greater than 50 microstrains to function in a physiologic load window.[109] The very stiff ceramic implants carried a disproportionate amount of the load, and the interfacial bone was moved into disuse atrophy.

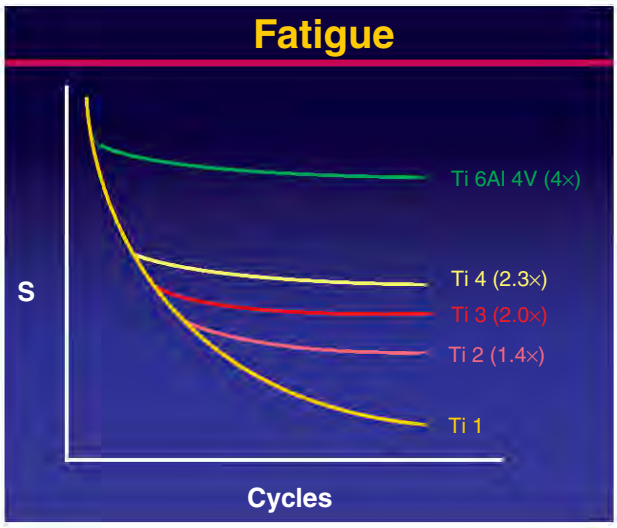


Figure 11-43 Implant bodies and components made of titanium alloy have a lower risk factor related to complication of fatigue fracture. The bone-implant contact percent is similar because all grades of titanium and titanium alloy form titanium oxide on the surface. Therefore the bone cannot distinguish between these materials. S, stress.

FORCE DURATION AND IMPLANT BODY DESIGN

The duration of bite forces on the dentition has a wide range. Under ideal conditions, the teeth come together during swallowing and eating for only brief contacts. The total time of those brief episodes is less than 30 minutes per day.[110] Patients who exhibit bruxism, clenching, or other parafunctional habits, however, may have their teeth in contact several hours each day. This force duration essentially is able to create a fatigue load on the implant.

Influence on Implant Body Design

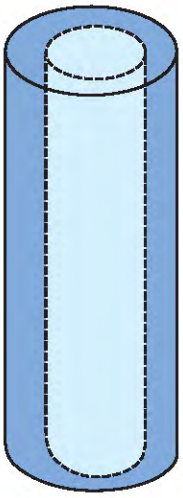
Implant bodies and components are prone to fatigue fractures, with incidence reported between 1% to 4% after 5 to 10 years of loading.[18,111] The most common cause of long-term failure occurred when a grade 1 titanium implant body was used, coupled with a less than ideal treatment plan that has higher stresses, as was demonstrated with 80% of failures from fracture of the Brånemark implant body.[98] An increase in fracture risk was also observed with parafunctional forces when a cantilever load was applied to the implant body.[35] Several factors influence the risk of fracture because of fatigue, including the material, force type, the direction of the applied load, and the overall geometric design.

Typical mechanical failures are due to either static loads or fatigue loads. Static load (i.e., one load cycle) failures cause the stress in the material to exceed its ultimate strength after one load application. Fatigue load failures happen if the material is subjected to lower loads but repeated cycles of that load. The endurance limit or fatigue strength is the level of highest stress a material may be repetitively cycled without failure. The endurance limit of a material is often less than one half its ultimate tensile strength. Therefore fatigue and ultimate strength values are related, but fatigue is a more critical factor, especially for patients with parafunction because they impose higher stress magnitude and greater cycles of load. Different materials have varying degrees of resistance to repeated loading and subsequent fatigue-related failures. The fatigue strength of titanium alloy (Ti-6Al-4V) is four times greater (and safer) than grade 1 titanium, and almost two times greater than grade 4 titanium (Figure 11-43).[105] Therefore long-term fracture of implant bodies and components may be dramatically reduced with the use of titanium alloy rather than any grade of commercially pure titanium. Therefore grades 1 to 4 titanium is at greater risk of implant body fracture under any condition when compared with the alloy (grade 5).

IMPLANT BODY SIZE AND DESIGN RELATED TO FRACTURE

Implant body designs used in the posterior areas of the mouth with higher forces should incorporate specific design features that make them less susceptible to the higher load profiles; therefore, further decreasing the risk of fracture. The ability of implants and components (e.g., abutment screws) to resist fracture from bending loads is directly related to the component's moment of inertia (or bending fracture resistance factor). This parameter is a function of the size of the cross-sectional

WWW.HIGHDENT.IR



$$I_{hollowcylinder} = \frac{1}{4} \times \pi \times (r_{outerdiameter}^4 - r_{innerdiameter}^4)$$

$$I_{hollowcylinder} = \frac{1}{4} \times \pi \times (1.475^4 - 1.25^4)$$

$$\text{or } I = 1.799 \text{ mm}^4$$

Figure 11-44 The equation for determining resistance to fatigue fracture is largely controlled by wall thickness in a dental implant.

geometry of the component. A solid cylinder fracture resistance is equal to the radius to the fourth power.

$$\text{I (moment of inertia)}_{\text{solid cylinder}} = \frac{1}{4} \times \pi \times \text{radius}^4$$

Therefore an implant or component 2 times as wide is 16 times more resistant to fracture. As a result, wider diameter implants may be used when offset loads (cantilevers) or greater stress conditions (i.e., parafunction, molar regions) exist.

The relationship of moment of inertia to overall fracture resistance can be demonstrated by this engineering mechanics equation:

$$\sigma = \frac{My}{I}$$

Where σ represents the stress, M is the moment load caused by eccentric loading and cantilevers, y is a point in the center of the part, and I is the moment of inertia. Therefore it can be shown that by increasing I, which is dependent on the part geometry, stress is decreased. Restated, by optimizing the implant body geometry within the anatomical dimensional limitation, it is possible to reduce the overall stress in the implant.

Considering the same equations, it can be shown that an abutment screw, which has a smaller cross-sectional area than an implant (typically about 2 mm), is more susceptible to fracture. This is particularly true when the abutment screw comes loose and bears a large, disproportionate component of a transverse load to the occlusal surface.

Some investigators have suggested the phenomenon of screw breakage to be a long-term advantage for the implant. Restated, it is better for the screw to break than the implant because the screw is easily retrievable—the implant body is not. Although this concept has some value, it is also a faulty safety factor. Most implant prostheses have more than one implant abutment. As soon as one screw loosens or breaks, the stresses are increased to the remaining implants, components, and bone interfaces. The additional off-axis loads resulting from the screw fracture increases the stresses in the overall system and may contribute to bone loss or implant component fracture of the remaining fixated units. Therefore, rather than depending upon a safety factor as a broken screw when the stresses are too great, transverse load contacts should be identified and corrected by designing reduced cantilevers or occlusal adjustments before screw breakage is imminent.

Implant body design may also increase the risk of long-term fatigue fracture. The abutment screw length is shorter than the receptor site within the implant. This permits the receptor site within the implant to be machined and allows the abutment screw to tighten the abutment, without the risk of "bottoming out" before the screw is completely tightened. The cross section of this portion of the implant body can be modeled as an annulus or hollow cylinder, similar to the cross section of a pipe. The wall thickness of the implant body in the region below the abutment screw controls the resistance to fatigue fracture (Figure 11-44).

Morgan et al. reported on fatigue failures of Brånemark implants subjected to bending loads.[111] Scanning electron micrographs of the fracture surfaces, coupled with the known position of the implants within the oral environment, enabled the authors to determine the load direction was buccolingual bending. Implant fracture occurred, as predicted, in the region of the implant that was characterized by a reduced annular cross section (Figure 11-45). When the abutment screw is only 6 mm long, and crestal bone loss of 6 mm occurs, the annulus (and weakest portion of the implant

WWW.HIGHDENT.IR

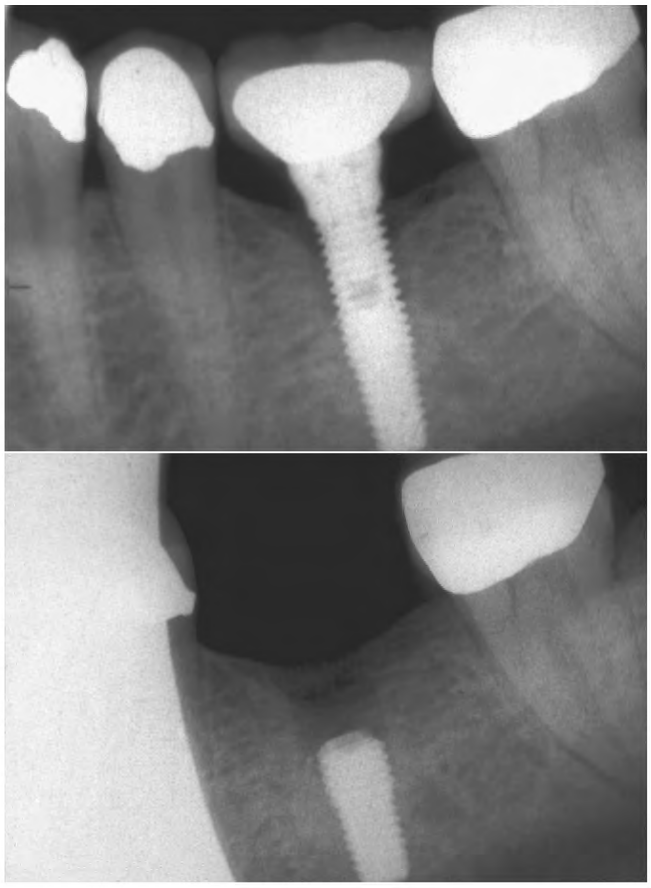


Figure 11-45 **A,** The annulus is the space within the implant body, below the abutment screw. **B,** When bone loss occurs to the annulus, the hollow cylinder implant design is susceptible to fracture.

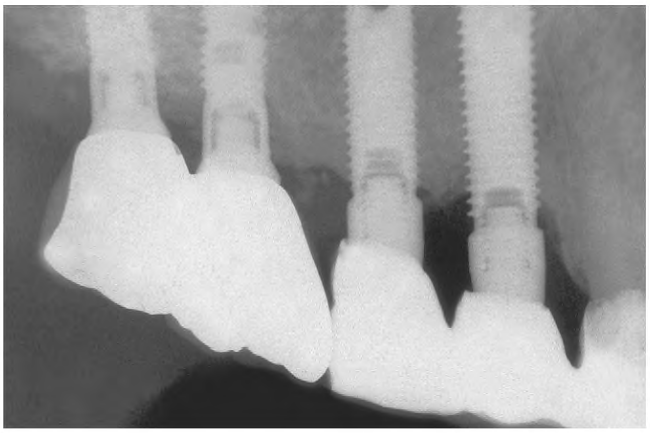


Figure 11-46 The anterior two implants are splinted together, and the abutment screw stops within the implant body near the crestal bone level. The annulus (open pipe design) of the implant body is weakest at this position.

body) is positioned in the highest stress locations from the increased bending moment at that location. As a result, implant body fracture is imminent. Therefore the abutment screw length is an important implant design issue and should be as long as possible.

The formula for the bending fracture resistance in an annulus condition is related to the outer diameter radius to the fourth power, minus the inner diameter radius to the fourth power. For example, if a 3.75-mm-diameter implant has a 0.4-mm thread depth, the outer diameter related to body fracture is actually 2.95 mm (3.75 to 0.4 mm on each side or 2.95 mm with a radius of 1.475 mm). If this implant has a 2.5-mm abutment screw hole (inner radius of 1.25 mm), its moment of inertia or bending fracture resistance at the end of the screw can by shown as:

$$I_{\text{hollow cylinder}} = \frac{1}{4} \times \pi \times (1.475^4 - 1.25^4)$$

Even a small dimensional change in wall thickness can result in a significant change in bending fracture resistance, because the dimension is multiplied to a power of four. When the outer diameter increases 0.1 mm and the inner diameter remains unchanged, the moment of inertia increases to 2.329 mm^4 or a 30% increase in strength. When the outer diameter remains unchanged and the inner diameter decreases 0.1 mm, the increase is 2.087 mm^4, or a 16% increase. Therefore an increase in outer diameter has a more significant effect on body wall strength, even though the metal thickness is similar in both scenarios. Therefore a 4-mm-diameter implant body has a significant increase in strength, compared with a 3.75-mm implant body, especially at the annulus position. The overall bending stress (and likelihood of fracture) decreases exponentially as the moment of inertia (bending fracture resistance factor) increases (Figures 11-46 and 11-47).

An interesting analysis compares a solid, one-piece implant to a two-piece, traditional root form implant. A solid implant with a 1.23-mm-diameter has the same resistance to bending fracture as the annulus region of a 3.75-mm traditional design. Moreover, a solid 3-mm implant has an approximately 340% increase in moment of inertia over the 3.75-mm traditional two-piece root form at the annulus position.

The crest module of an implant body may be designed to have a space around the abutment screw. This space increases the risk of fracture at this location. An external hex implant has the space above the implant body, whereas an internal hex system has the space within the implant body. Therefore the external hex has a slightly higher risk of fracture within the abutment, and an internal hex implant body has an increased risk of fracture at the crest module of the implant. The reduced strength of the implant body is not as great as the annulus at the end of the abutment screw. However, the lack of metal continuity is at the crest of the ridge, where the greatest forces occur. As a result, conditions as a decrease in implant diameter or angled forces increase the risk of fracture to the crest module at this site (Figure 11-48).

WWW.HIGHDENT.IR

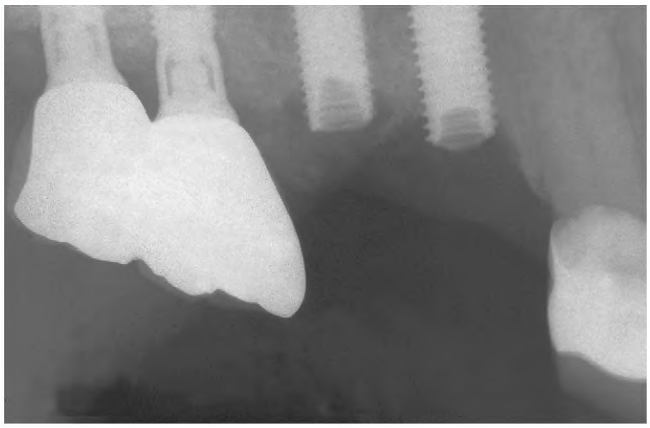


Figure 11-47 The two implants in Figure 11-46 fractured at the annulus position as a result of fatigue.

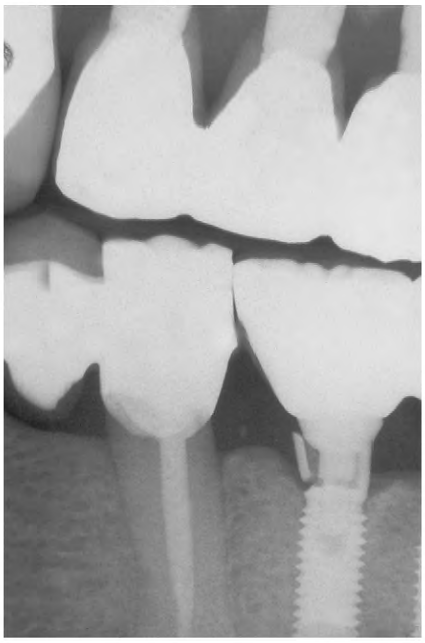


Figure 11-48 The internal hexagonal implant shown (Screw Vent, Zimmer, Encino, Calif.) has fractured at the crest module as a result of fatigue and implant design.

SUMMARY

The implant body design is responsible for transmitting the occlusal stress of the prosthesis to the supporting bone. The product used by the implant team may increase or decrease the risk of screw loosening, crestal bone loss, implant body bone loss, peri-implantitis, esthetics of the soft tissue drape, implant failure, and implant body fracture. Therefore it is prudent to make a selection based on a scientific approach, rather than on advertising or marketing opinion. This decision is even more important when force factors are greater than usual, bone density is poorer than usual, or implant body size is reduced.

References

1. Misch CE: Considerations of biomechanical stress in treatment with dental implants, *Dent Today* 25:80-85, 2006.
2. Misch CE: Prosthetic options for implant dentistry, *Dent Today* 8:39-44, 1989.
3. Misch CE, Palattella A: Bruxism and its effect on implant treatment plans, *Int Mag Oral Implantol* 2:6-16, 2002.
4. Misch CE: Bone density: effect on treatment planning, surgical approach and progressive loading, *Int J Oral Implant* 6:23-31, 1990.
5. Misch CE: Wide-diameter implant: surgical, loading, and prosthetic considerations, *Dent Today* 25:66-71, 2005.
6. Misch CE: Divisions of available bone. In Misch CE, editor: *Contemporary implant dentistry*, St Louis, 1993, Mosby.
7. Misch CE, Bidez MW: A scientific rationale for dental implant design. In Misch CE, editor: *Contemporary implant dentistry*, ed 2, St Louis, 1999, Mosby.
8. Golec TS, Krauser JT: Long-term retrospective studies on hydroxyapatite coated endosteal and subperiosteal implants, *Dent Clin North Am* 36:39-65, 1992.
9. McGlumphy EA, Peterson LJ, Larsen PE et al: Prospective study of 429 hydroxyapatite-coated cylindric omniloc implants placed in 121 patients, *Int J Oral Maxillofac Implants* 18:82-92, 2003.
10. Scortecci GM, Bert M, Leclercq P: Complications, prevention, correction and maintenance. In Scortecci GM, Misch CE, Brenner KU, editors: *Implant and restorative dentistry*, New York, 2000, Martin Dunitz.
11. Winkler S, Morris HF, Ochi S: Implant survival to 36 months as related to length and diameter, *Am Periodontol* 5:22-31, 2000.
12. Lekholm U, Gunne J, Henry P, et al: Survival of the Brånemark implant in partially edentulous jaws: a 10-year prospective multicenter study, *Int J Oral Maxollofac Implants* 14:639-645, 1999.
13. Buser D, Mericske-Stern R, Bernard JP et al: Long-term evaluation of non-submerged ITI implants. Part 1: 8-year life table analysis of a prospective multi-center study with 2359 implants, *Clin Oral Implants Res* 8:161-72, 1997.
14. Misch CE, Hoar JE, Beck G et al: A bone quality-based implant system: a preliminary report of Stage I and Stage II, *Implant Dent* 7:35-41, 1998.
15. Yukna RA: Optimizing clinical success with implants: maintenance and care, *Compend Cont Educ Dent* 15(Suppl):554-561, 1993.
16. Hammerle CH, Bragger U, Burgin W et al: The effect of subcrestal placement of the polished surface of ITI implants on marginal soft and hard tissues, *Clin Oral Implants Res* 7:111-119, 1996.
17. Hansson S: The implant neck: smooth or provided with retention elements—a biomechanical approach, *Clin Oral Implants Res* 10:394-405, 1999.
18. Goodacre CJ, Bernal G, Runcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121, 2003.
19. Misch CE: Short dental implants: a literature review and rationale for use, *Dent Today* 24:64-68, 2005.
20. Weng D, Jacobson Z, Tarnow D et al: A prospective multicenter clinical trial of 3i machined-surface implants: results after 6 years of follow-up, *Int J Oral Maxillofac Implants* 18:417-423, 2003.
21. Zechner W, Trinkl N, Watzek G et al: Radiologic follow-up of peri-implant bone loss around machine-surfaced and

WWW.HIGHDENT.IR

rough-surfaced interforaminal implants in the mandible functionally loading for 3 to 7 years, *Int J Oral Maxillofac Implants* 19:216-221, 2004.
22. Karoussis IK, Bragger U, Salvi GE et al: Effect of implant design on survival and success rates of titanium oral implants: a 10-year prospective cohort study of the ITI Dental Implant System, *Clin Oral Implants Res* 15:8-17, 2004.
23. Astrand P, Engquist B, Anzen B et al: A three-year follow-up report of a comparative study of ITI Dental Implants and Brånemark System implants in the treatment of partially edentulous maxilla, *Clin Implant Dent Related Res* 6:130-141, 2004.
24. Reilly DT, Burstein AH: The elastic and ultimate properties of compact bone tissue, *J Biomech* 8:393, 1975.
25. Misch CE, Qu Z, Bidez MW: Mechanical properties of trabecular bone in the human mandible: implications for dental implant treatment planning and surgical placement, *J Oral Maxillofac Surg* 57:700-706, 1999.
26. Lemons J: Biomaterials in implant dentistry. In Misch CE, editor: *Contemporary implant dentistry*, St Louis, 1993, Mosby.
27. Rieger MR, Mayberry M, Brose MO: Finite element analysis of six endosseous implants, *J Prosthet Dent* 63:671-676, 1990.
28. Burr DB, Mori S, Boyd RD et al: Histomorphometric assessment of the mechanics for rapid ingrowth of bone to HA/TCP coated implants, *J Biomed Mater Res* 27: 645-653, 1993.
29. Thomas KA, Kay JF, Cook SD et al: The effect of surface macrotexture and hydroxylapatite coating on the mechanical strengths and histologic profiles of titanium implant materials, *J Biomed Mater Res* 21L:1395-1414, 1987.
30. Baltag I, Watanabe K, Kusakari H et al: Long term changes in hydroxylapatite coated dental implants, *J Biomed Mater Res* 53:76-85, 2000.
31. Cook SD, Kay JF, Thomas KA et al: Interface mechanics and histology of titanium and HA coated titanium for dental implant applications, *Int J Oral Maxillofac Impl* 2:15-22, 1987.
32. Watzek G, Zechner W, Ulm C et al: Histologic and histomorphometric analysis of three types of dental implants following 18 months of occlusal loading: a preliminary study in baboons, *Clin Oral Implants Res* 16:408-416, 2005.
33. Bolind PK, Johansson CB, Becker W et al: A descriptive study on retrieved non-threaded and threaded implant designs, *Clin Oral Implants Res* 18:447-455, 2005.
34. Wiskott HW, Bleser UC: Lack of integration of smooth titanium surfaces: a working hypothesis based on strains generated in the surrounding bone, *Clin Oral Implants Res* 10:429-444, 1999.
35. Bidez MW, Misch CE: Issues in bone mechanics related to oral implants, *Implant Dent* 1:289-294, 1992.
36. Cowin SC: *Bone mechanics*, Boca Raton, Fla, 1989, CRC Press.
37. Misch CE, Bidez MW: Implant-protected occlusion: a biomechanical rationale, *Compend Cont Educ Dent* 15:1330-1343, 1994.
38. Strong JT, Misch CE, Bidez MW et al: Functional surface area: thread form parameter optimization for implant body design, *Compend Contin Educ Dent* 19:4-9, 1998.
39. Vaillancourt H, Pillar RM, McCammond D: Finite element analysis of crestal bone loss around porous-coated dental implants, *J Applied Biomaterials* 6:267-282, 1995.
40. Rieger MR, Adams WK, Kinzel GL et al: Finite element analysis of bone-adapted and bone-bonded endosseous implants, *J Prosthet Dent* 62:436-440, 1989.
41. Misch CE: Bone character: second vital implant criterion, *Dent Today* 7:39-40, 1988.
42. Tada S, Stegaroiu R, Kitamura E et al: Influence of implant design and bone quality on stress/strain distribution in bone around implants: a 3 dimensional finite element analysis, *Int J Oral Maxillofac Implants* 18:357-368, 2003.
43. Misch CE, Bidez MW: A scientific rationale for dental implant design. In Misch CE, editor: *Contemporary implant dentistry*, ed 2, St Louis, 1999, Mosby.
44. Ivanoff CJ, Gronhahl K, Sennerby L et al: Influence of variations in implant diameters: a 3- to 5-year retrospective clinical report, *Int J Oral Maxillofac Implants* 14:173-180, 1999.
45. Hoshaw SJ, Brunski JB, Cochran GVB: Mechanical loading of Brånemark fixtures affects interfacial bone modeling and remodeling, *Int J Oral Maxillofac Implants* 9:345-360, 1994.
46. Baumgardner JD, Boring G, Cooper RC et al: Preliminary evaluation of a new dental implant design in canine models, *Implant Dent* 9:252-260, 2000.
47. Duyck J, Rønold HJ, Van Oosterwyck et al: The influence of static and dynamic loading in marginal bone reactions around osseointegrated implants: an animal study, *Clin Oral Implant Res* 12:207-218, 2001.
48. Kohn DH: Overview of factors important in implant design, *J Oral Implantol* 18:204-219, 1992.
49. Currey JD: *The mechanical adaptations of bones*, Princeton, NJ, 1984, Princeton University Press.
50. Roberts WE, Turley PK, Brezniak N et al: Bone physiology and metabolism, *Calif Dent Assoc J* 15:54-61, 1987.
51. Nicolella DP, Lankford J, Jepsen K et al: Correlation of physical damage development with micro-structure and strain localization in bone. In Chandrin KB, Vanderby R Jr, Hefzy MS, editors: *Bioengineering conference,* vol 35, New York, 1997, American Society of Mechanical Engineers.
52. Nicolella DP, Nicholls AE, Lankford J: Micromechanics of creep in cortical bone. In *Transactions of the 44th Orthopedic Research Society, New Orleans, La, March 16-19, 1998*, Chicago, 1998, Orthopedic Research Society.
53. Cowin SC, Moss-Salentyin L, Moss MC: Candidates for mechanosensory system in bone, *J Biomech Eng* 113: 191-197, 1991.
54. Rubin CT, Hausman MR: The cellular basis of Wolff's law: transduction of physical stimuli to skeletal adaption, *Rheum Dis Clin North Am* 14:503-517, 1988.
55. Brighton CT, Strafford B, Gross SB et al: The proliferative and synthetic response of isolated calvarial bone cells of rats to cyclic biaxial mechanical strain, *J Bone Joint Surg* 73:320-331, 1991.
56. Jones DB, Nolte H, Scholubbers JG et al: Biomechanical signal transduction of mechanical strain in osteoblast like cells, *Biomaterials* 12:101-110, 1991.
57. Sachs F: Mechanical transduction in biological systems, *Crit Rev Biomed Eng* 16:141-169, 1988.
58. Sachs F: Mechanical transduction by membrane ion channels: a mini review, *Mol Cell Biochem* 104:57-60, 1991.
59. Frost HM: Mechanical adaptation: Frost's mechanostat theory. In Martin RB, Burr DB, editors: *Structure, function and adaption of compact bone,* New York, 1989, Raven Press.
60. Parfitt AM: The physiological and clinical significance of bone histomorphometric data. In Reck RR, editor:

WWW.HIGHDENT.IR

Bone histomorphometry: techniques and interpretation, Boca Raton, Fla, 1983, CRC Press.
61. Roberts WE, Garetto LP, De Castro RA: Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants with threaded or smooth surfaces: indications for provisional loading and axially directed occlusion, *Indiana Dent J* 68:19-24, 1989.
62. Roberts WE, Smith RK, Zilerman Y et al: Osseous adaptation to continuous loading of rigid endosseous implants, *Am J Orthod* 86:95-111, 1984.
63. Martin RB, Burr DB: *Structure, function, and adaption of compact bone*, New York, 1989, Raven Press.
64. Misch CE, Bidez MW, Sharawy M: A bioengineered implant for a predetermined bone cellular response to loading forces—a literature review and case report, *J Periodontol* 72:1276-1286, 2001.
65. Frost HM: Presence of microscopic cracks in vivo in bone, *Henry Ford Hosp Med Bull* 8:25-35, 1960.
66. Tricker ND, Garetto, LP: Cortical bone turnover and mineral apposition in dentate dog mandible [abstract], *J Dent Res* 76 SI:201, 1977.
67. Hoshaw SJ, Schaffler MB, Fyhrie DP: Effect of thread design on microdamage creation in cortical bone, *Trans Orthopaed Res Soc* 19:537, 1994.
68. Qiui SJ, Hoshaw SJ, Gibson GJ et al: *Osteocyte apoptosis after acute matrix injury in compact bone* [abstract], 43rd Annual Meeting, Orthopedic Research Society, Feb 9-13, 1997, San Francisco, Calif.
69. Mori S, Burr DB: Increased intracortical remodeling following fatigue damage, *Bone* 14:103-109, 1993.
70. Verborgt O, Gibson GJ, Schaffler MB: Loss of osteocyte integrity in association with microdamage and bone remodeling after fatigue in vivo, *J Bone Mineral Res* 15:60-67, 2000.
71. Hoshaw SJ: *Investigation of bone modeling and remodeling at a loaded bone-implant interface* [dissertation], Troy, NY, 1992, Rensselaer Polytechnic Institute.
72. Barbier L, Schepers E: Adaptive bone remodeling around oral implants under axial and non axial loading conditions in the dog mandible, *Int J Oral Maxillofac Implants* 12:215-223, 1997.
73. Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study, *J Periodontol* 77:1340-1347, 2006.
74. Singley JE, Mischke CR: *Mechanical engineering design*, ed 5, New York, 1989, McGraw-Hill.
75. Steigenga JT: *The effect of implant thread geometry on strength of osseointegration and the bone implant contact* [master's thesis], Ann Arbor, Mich, 2003, University of Michigan.
76. Steigenga JT, Al-Shammari KF, Nociti FH et al: Dental implant design and its relationship to long term implant success, *Implant Dent* 12:306-317, 2003.
77. Steigenga J, Al-Shammari K, Misch CE et al: Effects of implant thread geometry on percentage of osseointegration and resistance to reverse torque in the tibia of rabbits, *J Periodontol* 75:1233-1241, 2004.
78. Kim WT, Cha YF, Oh SJ et al: The three dimensional finite element analysis of stress according to implant thread design under the axial load, *Korean J Oral Surg* 27:3-8, 2001.
79. Chun HS, Cheong SY, Han JH et al: Evaluation of design parameters of osseintegrated dental implants using finite element analysis, *J Oral Rehabil* 29:565-574, 2002.
80. Misch CE, Dietsh-Misch F, Hoar J et al: A bone quality-based implant system (BioHorizons maestro dental implants): a prospective study of the first year of prosthetic loading, *J Oral Implant* 25:185-197, 1999.
81. Hoar JE, Beck GH, Crawford EA et al: Prospective evaluation of crestal bone remodeling of a bone density-based dental implant system, *Compend Cont in Educ Dent* 19:17-24, 1998.
82. Garetto LP, Chen J, Parr JA et al: Remodeling dynamics of bone supporting rigidly fixed titanium implants: a histomorphometric comparison in four species including humans, *Implant Dent* 4:235-243, 1995.
83. Cooper C: Advantages of a short implant design, *Am Acad Implant Dent*, San Diego, Calif, 1998.
84. Roberts WE: Bone dynamics of osseointegration, ankylosis, and tooth movement, *J Indiana Dent Assoc* 78:24-32, 1997.
85. Misch CE, Bidez MW, Strong JT: *Design process for skeletal implants to optimize cellular response*, patent 5,628,630, issued May 13, 1997.
86. Roberts WE, Marshall KJ, Mosary PG: Rigid endosseous implants utilized as anchorage to protract molars and close an atrophic extraction site, *Angle Orthod* 60: 135-152, 1990.
87. Brunski JD, Hipp JA, Cochran GVB: The influence of biomechanical factors at the tissue-biomaterial interface. In Hanker JS, Girnmara BC, editors: *Biomechanical materials and devices*, Pittsburgh, 1989, Materials Research Society.
88. Scurria MS, Morgan V, Gucher AD et al: Prognostic variables associates with implant failure: a retrospective effectiveness study, *Int J Oral Maxollofac Implants* 13: 400-406, 1998.
89. Boggan S, Strong JT, Misch CE et al: Influence of hex geometry and prosthetic table width on static and fatigue strength of dental implants, *J Prosth Dent* 82:436-440, 1999.
90. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, Dental Learning Systems Co, Inc, *Postgrad Dent* 3:3-17, 1995.
91. Misch CE, Suzuki JB, Misch-Dietsh FM et al: A positive correlation between occlusal trauma and peri-implant bone loss: literature support, *Implant Dent* 14:108-116, 2005.
92. Hermann JS, Buser D, Schenk RK et al: Biologic width around one and two-piece titanium implants, *Clin Oral Implants Res* 12:559-571, 2001.
93. Hanggi MP, Hanggi DC, Schoolfield JD et al: Crestal bone changes around titanium implants. Part I: a retrospective radiographic evaluation in humans comparing two non-submerged implant designs with different machined collar lengths, *J Periodontol* 76:791-802, 2005.
94. Misch CE: *A three-dimensional finite element analysis of two blade implant neck designs* [master's thesis], Pittsburgh, 1989, University of Pittsburgh.
95. Rasmusson L, Kahnberg KE, Tan A: Effects of implant design and surface on bone regeneration and implant stability: an experimental study in the dog mandible, *Clin Implant Dent Relat Res* 3:2-8, 2001.
96. Jung YC, Han CH, Lee KW: A 1-year radiographic evaluation of marginal bone around dental implants, *Int J Oral Maxillofac Impl* 11:811-818, 1996.
97. DeBruyn H, Collaert B, Linden U et al: Clinical outcome of screw vent implants. A 7-year prospective follow-up study, *Clin Oral Impl Res* 10:139-148, 1999.

WWW.HIGHDENT.IR

98. Rangert B, Krogh PH, Langer B et al: Bending overload and implant fracture: a retrospective clinical analysis, *Int J Oral Maxillofac Implants* 10:326-334, 1995.
99. Gibbs CH, Mahan PE, Mauderli A et al: Limits of human bite strength, *J Prosthet Dent* 56:226-229, 1986.
100. Scott I, Ash MM Jr: A six channel intra-oral transmitter for measuring occlusal forces, *J Prosthet Dent* 16:56, 1966.
101. Williams DF: *Biocompatibility of clinical implant materials,* vol 1, Boca Raton, Fla, 1981, CRC Press.
102. Luckey HA, Kubli F Jr: *Titanium alloys in surgical implants,* ASTM STP 796, 1983, Philadelphia.
103. ASTM F 67-95: *Standard specification for unalloyed titanium for surgical implant applications,* Philadelphia, 1995, American Society for Testing and Materials.
104. ASTM F 1472-93: *Standard specification for wrought Ti-6Al-4V alloy for surgical implant applications,* Philadelphia, 1993, American Society for Testing and Materials.
105. ASTM F 136-96: *Standard specification for wrought titanium-6Al-4V ELI alloy for surgical implant applications,* Philadelphia, 1996, American Society for Testing and Materials.
106. Brown SA, Lemons JE: *Medical applications of titanium and its alloys,* ASTM STP 1272, 103, Philadelphia, 1996.
107. Schnitman PA, Schulman LB, editors: Dental implants: benefit and risk. In *Proceedings of NIH—Harvard Consensus Development Conference, June 1978*, Washington, DC, 1978, US Department of Health and Human Services.
108. Lemons J: Biomaterials in implant dentistry. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.
109. Frost HM: The mechanostat: a proposed pathogenetic mechanism of osteoporosis and bone mass effects of mechanical and nonmechanical agents, *Bone Miner* 2: 73-85, 1988.
110. Graf H: Bruxism, *Dent Clin North Am* 13:659-665, 1969.
111. Morgan MJ, James DF, Pilliar RM: Fractures of the fixture component of an osseointegrated implant, *Int J Oral Maxillofac Impl* 8:409-413, 1993.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 12

Preimplant Prosthodontics: Overall Evaluation, Specific Criteria, and Pretreatment Prostheses

Carl E. Misch, Francine Misch-Dietsh

Implants serve as a foundation for the prosthetic support of missing teeth. However, in the partially edentulous patient, the existing teeth may often require restorations or treatment. The existing conditions of the stomatognathic system should be evaluated and treated, when necessary. As such, preimplant prosthodontic considerations are a vital phase of the overall treatment before implant surgery. For example, the surgical decision to augment or perform osteoplasty before implant surgery is primarily dependent on the desired prosthetic result. Most all conventional forms of construction, from buildings to art form, require a clear vision of the end result before the project is begun.

OVERALL EVALUATION

The preimplant prosthodontic evaluation of the patient's overall condition closely resembles traditional dentistry. When a restoring dentist first evaluates the prosthetic needs of a patient, an orderly process is required, regardless of the current state of the dentition. In other words, regardless of whether the patient has all teeth or is missing all teeth, after the dentist accepts the responsibility of long-term professional guidance and treatment as necessary, a consistent approach to care is beneficial. There are five initial elements that should be assessed in sequence and treated when indicated. These elements include: maxillary anterior tooth position, the existing occlusal vertical dimension, the mandibular incisor edge position, the maxillary occlusal plane, and the mandibular occlusal plane (Box 12-1). These elements are evaluated in a partially edentulous patient during the initial clinical examination and on mounted diagnostic casts (which may also serve for diagnostic wax-up procedures).

MAXILLARY ANTERIOR TOOTH POSITION

The position of the existing maxillary anterior teeth is first assessed. Most often these natural teeth are adequate in location and incisal edge position. However, if their position is undesirable for any reason, orthodontics or restoration may be indicated. At this point, the evaluation is not for the cosmetic aspects of tooth color or shape,

Box 12-1 Sequence of Treatment

Overall evaluation

Maxillary anterior tooth position
Occlusal vertical dimension
Mandibular incisal edge
Maxillary posterior plane
Mandibular posterior plane

Specific criteria

Lip lines
Maxillomandibular arch relationship
Existing occlusion
Crown height space
Temporomandibular joint status
Extraction of hopeless or guarded-prognosis existing teeth
Existing prostheses
Arch form (ovoid, tapering, square)
Natural tooth adjacent to implant site
Soft tissue evaluation of edentulous sites

WWW.HIGHDENT.IR

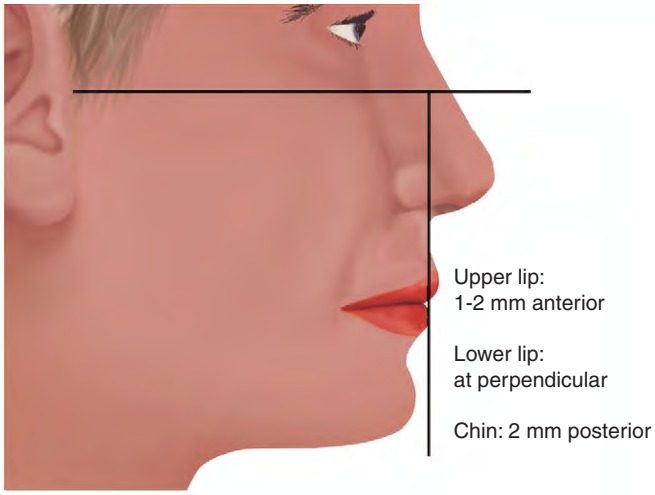


Figure 12-1 The labial position of the teeth is first evaluated relative to the support of the maxillary lip. A vertical line drawn through the subnasal point and perpendicular to the Frankfort plane is a baseline. The maxillary lip should be 1-2 mm anterior to this line, the lower lip even with the line, and the chin 2 mm behind the line.

but tooth position is scrutinized. If the maxillary incisor edge is modified in either the horizontal or vertical plane, all other four elements of the stomatognathic system may also need to be changed.

The labial position of the maxillary anterior teeth is first determined with the lip in repose. This is primarily evaluated by overall support of the maxillary lip and its relationship to the balance of the face, especially in relation to the nose and presence or absence of a philtrum in the midline (Figure 12-1).[1-7] When the teeth are positioned more labial, the vertical position of the lip is elevated. Likewise, a more palatal position of the maxillary anterior teeth results in a more inferior or extended position of the lip. If the labial or horizontal position is going to be altered, orthodontic therapy is the treatment of choice. On occasion, a prosthetic or surgical approach may be indicated with or without orthodontic treatment.

The next step in the evaluation process (when the labial position is acceptable) is the vertical position of the maxillary anterior teeth related to the lip in repose. The maxillary canine is the key for this position. Misch has suggested the canine tip be located approximately 1 mm level with the lip in repose, regardless of the age or sex of the patient (Figure 12-2).[8] A horizontal line drawn from one canine tip to the other should be level to the horizon. The central incisors are 1 to 2 mm longer in a horizontal plane to the canines.

If the patient is wearing a maxillary complete denture, the maxillary anterior tooth position is often incorrect. As a result of resorption of the premaxilla, the denture shifts apically and posteriorly after the bone loss pattern. No other region of the mouth should be restored until this position is corrected, because it negatively influences the proper position of every other segment (e.g., occlusal vertical dimension, mandibular anterior tooth position, posterior planes of occlusion).

Figure 12-2 The vertical position of the maxillary anterior teeth is assessed. The ideal position is determined by the canine to lip in repose position: a horizontal line is drawn from canine tip to canine tip, and the central incisors are 1 to 2 mm longer. This position is consistent (within 1 mm) regardless of the age or sex of the patient.

The maxillary anterior horizontal and vertical tooth positions are evaluated prior to any other segment of the arches, including the occlusal vertical dimension (OVD). If the maxillary anterior teeth are significantly malpositioned, the clinician should obtain further diagnostic studies, such as a cephalometric radiograph, to determine the relationship of the maxilla to the cranial base. The patient may have unfavorable skeletal relationships (vertical maxillary excess or deficiency). If the position of the natural maxillary anterior teeth is undesirable for any reason, orthodontics, orthognathic surgery, or restoration may be indicated. After the position of the maxillary anterior teeth is acceptable, the next prosthetic step is the evaluation of the OVD.

EXISTING OCCLUSAL VERTICAL DIMENSION

In order to determine the crown height space (CHS), the overall issue of OVD must be addressed. The patient's existing OVD should be evaluated early in an implant prosthetic treatment plan, because any modification

WWW.HIGHDENT.IR

will significantly modify the overall treatment. Not only will a change in OVD require at least one full arch to be reconstructed, it will also affect the CHS and therefore the potential number, size, position, and angulation requirements of the implants. The OVD is defined as the distance between two points (one in the maxilla and the other directly below in the mandible) when the occluding members are in contact.[9] This dimension requires clinical evaluation of the patient and cannot be evaluated solely on the diagnostic casts.

The determination of the OVD is not a precise process, because a range of dimensions is possible without clinical symptoms.[10-23] At one time, it was believed OVD was very specific and remained stable throughout a patient's life. However, this position is not necessarily stable when the teeth are present or after the teeth are lost. Long-term studies have shown this is not a constant dimension and often decreases over time without clinical consequence in the dentate or partially or completely edentulous patient. A completely edentulous patient often wears the same denture for more than 10 years, during which time the OVD is reduced 10 mm or more without symptoms or patient awareness.

The OVD may be altered permanently without the symptoms of pain or dysfunction. However, this is not to say that altering the OVD has no consequence. A change in OVD affects the CHS. As such, it may affect the biomechanics of the support system of a prosthesis. In addition, any change in the OVD will also modify the horizontal dimensional relationship of the maxilla to the mandible. Therefore a change in OVD will modify the anterior guidance, range of function, and esthetics.

The most important effect of OVD on tooth (implant) loading may be the effect on the biomechanics of anterior guidance. The more closed the OVD, the farther forward the mandible rotates and the more Class III the chin appears (Figures 12-3 and 12-4). In a Class II, division 2 patient, the more closed the OVD, the steeper the anterior guidance and the greater the vertical overlap of the anterior teeth. Anterior guidance is necessary to maintain incisal guidance during mandibular excursions to decrease the risk of posterior interferences. These conditions will increase the forces to the anterior teeth. Increasing the OVD has the opposite effect. In general, for the dentate patient it may be more precarious to close an OVD than to open it, because the resulting anterior rotation of the mandible will position mandibular incisor teeth facially in a closer relationship to the maxillary teeth in centric occlusion. In completely edentulous patients restored with fixed implant prosthodontics, a change in OVD in either direction affects biomechanics. Opening the OVD and decreasing the incisal guidance with a resulting bilaterally balanced occlusion may increase forces placed on posterior implants during mandibular excursion. Closing the OVD may increase the forces to anterior implants during any excursion. On occasion, a change in the OVD may also affect the sibilant sounds by altering the horizontal position of the mandible.

The OVD is almost never naturally too large and, unless some manufactured interference has been created, it is within clinical guidelines or collapsed. Therefore the restoring dentist most often should determine whether the OVD needs to be increased. In other words, the existing OVD is a position to start the evaluation, not one that necessarily must be maintained.

According to Kois and Phillips, three situations primarily mandate the modification of the OVD: (1) esthetics, (2) function, and (3) structural needs of the dentition.[12] Esthetics is related to OVD for incisal edge positions, facial measurements, and the occlusal plane. Function is related to the canine positions, incisal guidance, and angle of load to teeth or implants. Structural requirements are related to dimensions of teeth for restoration while maintaining a biological width.

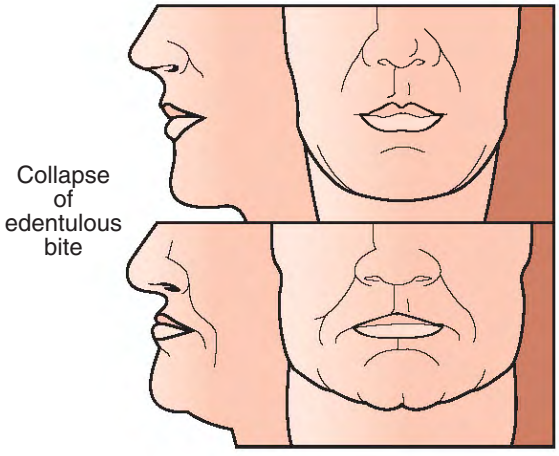


Figure 12-3 The closed occlusal vertical dimension (OVD) rotates the chin farther forward.

Figure 12-4 The more closed the occlusal vertical dimension, the farther forward the mandibular teeth occlude.

WWW.HIGHDENT.IR

Methods to Evaluate Occlusal Vertical Dimension

In traditional prosthodontics, a range of techniques has been described to establish the OVD. Objective methods use facial dimension measurements, whereas subjective methods rely on esthetics, resting arch position, and closest speaking space. There is no consensus on the ideal method to obtain the OVD. Therefore this dimension is part art form and part science. And yet it is critical enough that a final treatment plan should not be rendered until a determination has been made relative to this dimension.

The subjective methods to determine OVD include the use of resting interocclusal distance and speech-based techniques using sibilant sounds. Niswonger proposed the use of the interocclusal distance ("freeway space"), which assumes that the patient relaxes the mandible into the same constant physiologic rest position.[13] The practitioner then subtracts 3 mm from the measurement to determine the OVD. Two observations conflict with this approach. First, the amount of freeway space is highly variable in the same patient, depending on factors such as head posture, emotional state, presence or absence of teeth, parafunction, and time of recording (greater in the morning). Second, interocclusal distance at rest varies 3 to 10 mm from one patient to another. As a result, the distance to subtract from the freeway space is unknown for a specific patient. Therefore the physiologic rest position should not be the primary method to evaluate OVD. However, it should be evaluated once the OVD is established to ensure a freeway space exists when the mandible is at rest.

Silverman stated that approximately 1 mm should exist between the teeth when making an *S* sound.[14] Pound further developed this concept for the establishment of centric and vertical jaw relationship records for complete dentures.[15,16] Although this concept is acceptable, it does not correlate to the original OVD of the patient. Denture patients often wear the same prosthesis for more than 14 years and during this time lose 10 mm or more of their original OVD. Yet all of these patients are able to say "Mississippi" with their existing prosthesis. If speech were related to the original OVD, these patients would not be able to pronounce the *S* sound because their teeth would be more than 11 mm apart. Therefore the speaking space should not be used as the only method to establish OVD. After the OVD has been determined, the speaking space should be observed, and the teeth should not touch during sibilant sounds. On occasion, a short adjustment period of a few weeks may be required to establish this criterion. Therefore a transitional prosthesis should be used to evaluate this position in case it must be modified before the final restoration.

Kois[12] has noted the subjective method of esthetics to establish an OVD is the most difficult to teach inexperienced dental students and therefore is least likely to be initially addressed when teaching the concepts of determining OVD. However, experienced clinicians often value this method more than any other to assess OVD.

After the position of the maxillary incisor edge is determined, the OVD influences esthetics of the face in general. Facial dimensions are directly related to the ideal facial esthetics of an individual and can be easily assessed regardless of the clinician's experience.[17-25] This objective evaluation is usually the method of choice to evaluate the existing OVD or establish a different OVD during prosthetic reconstruction. In addition, it may be performed without the need of additional diagnostic tests.

Facial measurements can be traced back to antiquity, when sculptors and mathematicians followed the *golden ratio* for body and facial proportions as described by Pythagorus. The golden ratio relates to the length and width of a golden rectangle as 1 to 0.618. Many human body proportions follow the golden ratio because it is considered the most esthetically appealing to the human eye.[18-20,25] Leonardo da Vinci later contributed several observations and drawings on facial proportions, which he called *divine proportions*.[21] He observed the distance between the chin and the bottom of the nose (i.e., OVD) was a similar dimension as (1) the hairline to the eyebrows, (2) the height of the ear, and (3) the eyebrows to the bottom of the nose—and each of these dimensions equaled one third of the face.

Many professionals, including plastic surgeons, oral surgeons, artists, orthodontists, and morticians, use facial measurements to determine OVD. A review of the literature found that many different sources reveal many correlations of features that correspond to the OVD:

1. The horizontal distance between the pupils
2. The horizontal distance from the outer canthus of one eye to the inner canthus of the other eye
3. Twice the horizontal length of one eye
4. Twice the horizontal distance from the inner canthus of one eye to the inner canthus of the other eye
5. The horizontal distance from the outer canthus of the eye to the ear
6. The horizontal distance from one corner of the lip to the other, following the curvature of the mouth (cheilion to cheilion)
7. The vertical distance from the external corner of the eye (outer canthus) to the corner of the mouth
8. The vertical height of the eyebrow to the ala of the nose
9. The vertical length of the nose at the midline (from the nasal spine [subnasion] to the glabella point)
10. The vertical distance from the hair line to the eyebrow line
11. The vertical height of the ear
12. The distance between the tip of the thumb and the tip of the index finger when the hand lays flat, with the fingers next to each other (Figure 12-5)

WWW.HIGHDENT.IR

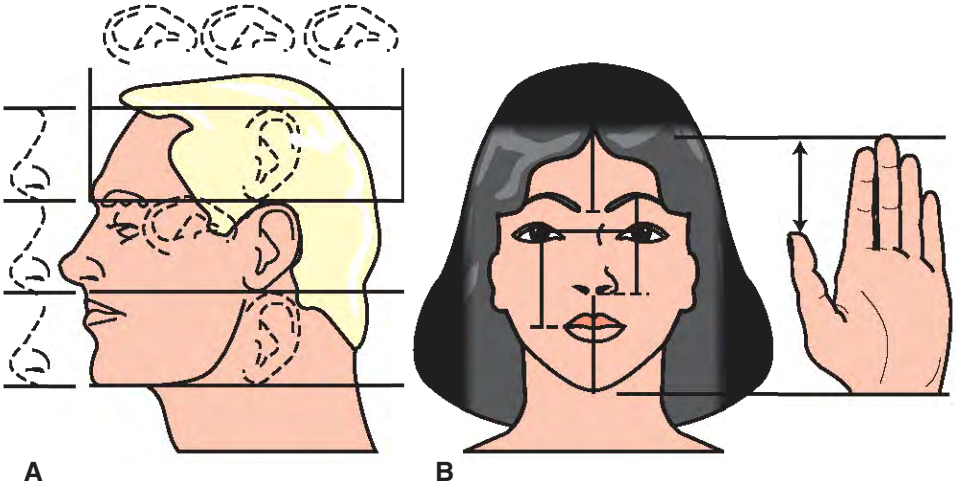


Figure 12-5 The occlusal vertical dimension (OVD) may initially be evaluated by objective measurements, comparing facial dimensions to the existing OVD. Leonardo da Vinci described divine proportions in the following way: **(A)** "The distance between the chin and the nose and the hairline and the eyebrows are equal to the height of the ear and a third of the face. From the outer canthus of the eye to the ear, the distance is equal to the height of the ear and to one-third of the face height." **B,** In addition, facial height (from chin to hairline) is equal to the height of the hand, and the nose is the same length as the distance between the tip of the thumb and the tip of the index finger.

All these measurements do not correspond exactly to each other, but usually do not vary by more than a few millimeters (with the exception of the vertical height of the ear) when facial features appear in balance. An average of several of these measurements may be used to assess the existing OVD. In a clinical study by Misch, the OVD was often slightly larger than the facial measurements listed (more in men than women), but was rarely a smaller dimension.[22] The subjective criteria of pleasing esthetics may then be considered after the facial dimensions are within balance to each other.

Radiographic methods to determine an objective OVD are also documented in the literature. Tracings on a cephalometric radiograph is suggested when gross jaw excess or deficiency is noted. Such conditions may stem from vertical maxillary excess, vertical maxillary deficiency, vertical mandibular excess (long chin), vertical mandibular deficiency (short chin), apertognathia or Class II, division 2 (deep bite) situations. Orthodontic treatment planning of a dentate patient often includes a lateral cephalogram and may be used to evaluate OVD (glabella-subnasale, subnasale-menton). The same measurements may be performed on the edentulous patient.[26,27]

Esthetics are influenced by OVD, because of the relationship to the maxillomandibular positions. The smaller the OVD, the more Class III the jaw relationship becomes; the greater the OVD, the more Class II the relationship becomes. The maxillary anterior tooth position is determined first and is most important for the esthetic criteria of the reconstruction. Alteration of the OVD for esthetics rarely includes the maxillary tooth position. For example, the OVD position may be influenced by the need to soften the chin for a patient with a large mental protuberance.

After the OVD satisfies the esthetic requirement of the prosthetic reconstruction, it may still be slightly refined. For example, the OVD may be modified to improve the direction of force on the anterior implants. In addition, anterior mandibular implants on occasion are too facial to the incisal edge position, and increasing the OVD makes them much easier to restore. Therefore, because the OVD is not an exact measurement, the ability to alter this dimension within limits may often be beneficial.

The evaluation of the pretreatment OVD is also very important for the patient wearing a complete maxillary denture opposing a partially edentulous mandible, especially in the case of edentulous posterior segments that are not compensated by a removable partial denture (Kennedy-Applegate Class I). Under these conditions, a combination (Kelly) syndrome may be present and is especially noteworthy if the OVD is within normal limits.[28] The clinical symptoms include: (1) maxillary incisor denture position up and rotated back from ideal, (2) lower natural anterior teeth overerupted and beyond the mandibular occlusal plane, (3) horizontal occlusal plane tilted apically in the anterior and occlusally in the posterior regions, (4) enlarged tuberosities encroaching on the mandibular interarch space, (5) maxillary palatal hyperplasia, and (6) highly mobile tissue in the premaxilla. In addition, because the mandibular posterior teeth have been missing many years to develop this syndrome, there is a lack of posterior bone in the mandible to place endosteal implants (Figure 12-6).

The proper maxillary incisal edge position and OVD are especially critical for these patients, because of the incidence of mandibular incisor extrusion beyond

WWW.HIGHDENT.IR

Figure 12-6 **A,** A combination syndrome describes the clinical conditions occurring when a maxillary denture opposes mandibular anterior teeth and no partial denture. The mandibular teeth overerupt as the maxillary denture seats up in the anterior and down in the posterior. **B,** The premaxillary bone is lost and the tuberosities enlarged.

the maxillary occlusal plane. The extrusion is usually accompanied by the alveolar process. To position the maxillary incisors properly, the mandibular anterior teeth must be repositioned at the proper incisal plane. Endodontic therapy and crown lengthening procedures usually precede the restorations on the lower arch to obtain a retentive and esthetic restoration.

On occasion, the remaining roots of the mandibular anterior teeth are too short to consider for long-term prognosis after the crown lengthening is performed. Under these conditions, extraction of the mandibular anterior teeth, alveoloplasty, and implant placement may be indicated. When the arch shape is ovoid to tapered, five anterior implants may be adequate to serve as support for a full arch implant–supported restoration. Therefore the implants replace the teeth extracted from overeruption, and they can also replace the posterior missing teeth. This is usually very helpful, because long-term edentulous posterior segments are usually deficient in bone volume. Thus this approach eliminates the need for posterior bone grafts to restore the lower arch with a fixed implant–supported restoration.

MANDIBULAR INCISOR EDGE POSITION

After the maxillary incisal edge and the OVD are deemed clinically acceptable, the position of the lower anterior teeth is evaluated. When natural teeth are present, or when a fixed prosthesis is planned in the anterior region, the mandibular teeth incisal edge should contact the lingual aspect of the maxillary anterior natural teeth at the desired OVD position. A vertical overlap with the maxillary anterior teeth is usually in the range of 3 to 5 mm. The incisal guidance is defined as the influence of contacting surfaces of the mandibular and maxillary anterior teeth on mandibular movements.[9] The incisal guide angle is formed by the intersection of the plane of occlusion and a line within the sagittal plane determined by the incisal edge of the maxillary and mandibular central incisors when in maximal intercuspation. It is responsible for the amount of posterior tooth separation during mandibular excursions and to do so, it should be steeper than the condylar disc assembly (Christensen's phenomenon). Therefore any planned prosthesis and associated compensating curves should be developed within these confines. If not, the maxillomandibular arch position may be improper (i.e., in the skeletal Class II patient) and the posterior teeth may exhibit lateral contacts during mandibular excursions. Under these conditions, the masseter and temporalis muscles do not reduce their contraction force during these movements (as they do when only anterior teeth occlude in excursions), and the strong muscles of mastication continue to contract and place an increased force on the entire stomatognathic system.

The incisal guidance is evaluated on the mounted diagnostic models. A steep incisal guidance helps in avoiding posterior interferences in protrusive movement. However, the steeper the incisal guide angle, the greater force applied to anterior crowns. This may present a significant problem for an anterior single-tooth implant replacement. On occasion, the tooth is lost as a result of severe parafunction on a tooth with a steep incisal guidance (usually from fracture after endodontic therapy). On the other hand, if the existing incisal guidance is shallow, it may be necessary to plan recontouring or prosthetic restoration of posterior teeth that exhibit contact during excursions. For example, a mesially tipped mandibular third molar is often in this situation and may greatly compromise the implant placed in a maxillary second molar region.

EXISTING OCCLUSAL PLANES (POSTERIOR MAXILLARY AND MANDIBULAR PLANES OF OCCLUSION)

After the maxillary anterior teeth position, OVD, and mandibular anterior teeth position are deemed acceptable, the horizontal occlusal planes are evaluated in

WWW.HIGHDENT.IR

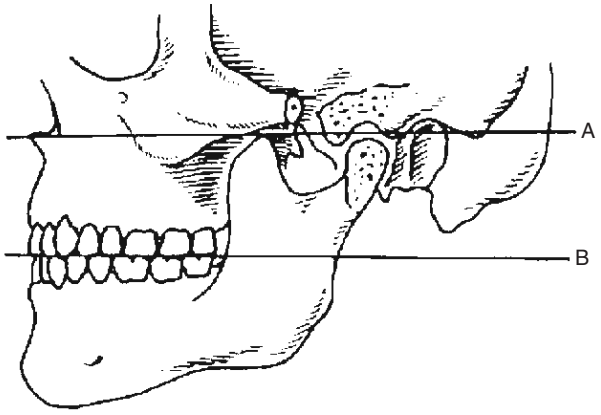


Figure 12-7 The ala–tragus line (Camper's plane) (Line A) is parallel to the occlusal plane of the maxillary teeth (Line B).

the posterior regions of the mouth. Their position related to the curves of Wilson (mediolateral) and Spee (anteroposterior) and to each other should allow harmonious occlusion with maximum occlusal interdigitation and canine or mutually protected occlusion. Ideally, the maxillary posterior occlusal plane should be parallel to the Camper's plane (i.e., to the mid-tragus position) (Figure 12-7). The occlusal plane of existing teeth is especially evaluated in partially edentulous patients in relationship to the final implant prosthesis. Odontoplasty, endodontic therapy, or crowns are indicated to remedy tipping or extrusions of adjacent or opposing natural teeth. A pretreatment diagnostic wax-up is strongly suggested to evaluate the needed changes before implant placement. A proper curve of Spee and curve of Wilson are also indicated for proper esthetics and are reproduced in the compensating curves for complete denture fabrication (Figure 12-8).

The occlusal plane seems like an obvious step in the patient dental evaluation. However, an evaluation of three-unit fixed prostheses in several large dental laboratories revealed most restoring dentists prepare crowns or three-unit fixed prostheses without correcting the opposing occlusal plane. Apparently, the existing occlusal plane is not routinely evaluated before the fabrication of the prosthesis, or the patient and doctor have decided to compromise the final result and restore the missing teeth to the preexisting poor position. Instead, the restoring dentist should highlight to the patient the extrusion or exfoliation of the surrounding teeth, which is often obvious on a panoramic radiograph or diagnostic casts, after it is noted. The need to restore the missing tooth sooner rather than later is apparent to the patient, because the teeth are already shifting as a result of the arch collapse. If the patient cannot afford the complete treatment plan related to the missing teeth, the opposing arch with the poor occlusal plane should be treated first, not the arch with the missing tooth. In this way, opposing quadrants will ultimately be restored to a proper relationship. Of course, the missing

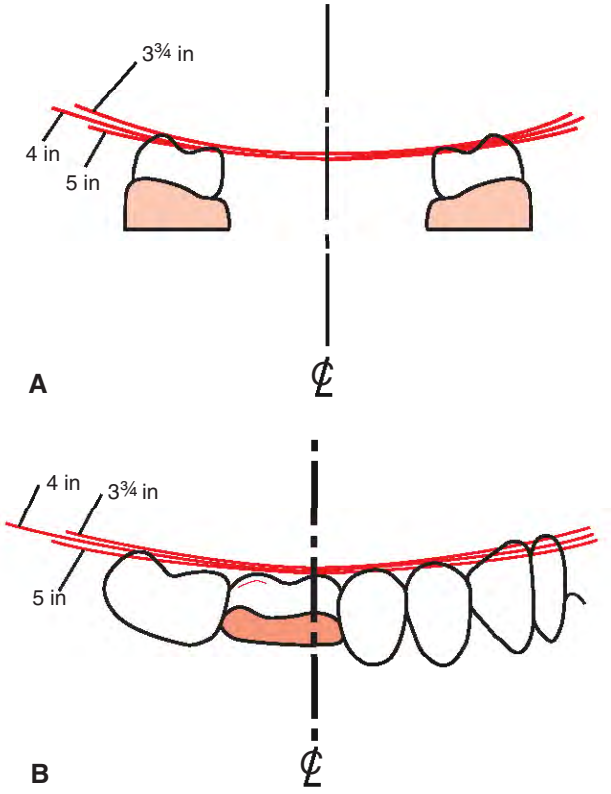


Figure 12-8 **A,** The curve of Spee is also similar to the radius of a 4-inch sphere and is related to skull size. **B,** The curve of Wilson is evaluated before reconstruction in the region. The radius of the average curve corresponds to the radius of a 4-inch sphere.

tooth should be replaced before the occlusal plane is compromised again. An occlusal plane analyzer may be used on diagnostic casts to evaluate pretreatment conditions and assist in intraoral occlusal plane correction. Occlusal analyzers are fabricated in several sizes. The average size corresponds to a 4-inch sphere and provides a starting point for ideal curves of Wilson and Spee. Any discrepancy observed on the cast may be corrected in the mouth. A laboratory-assisted template may be fabricated with this intent. In the laboratory, a vacuum or press fit of an acrylic shell is prepared over the cast. The occlusal plane analyzer is then used to evaluate and correct an improper occlusal plane. A hand piece is used to grind the acrylic shell and protruding occlusal cusps on the duplicate diagnostic cast. The clear acrylic shell is then taken intraorally and inserted over the teeth. Any cusp extending through the acrylic shell is recontoured to the level of the surrounding acrylic. As such, the occlusal plane is rapidly corrected to an ideal condition (Figure 12-9).

The natural dentition opposing a partially edentulous ridge also must be carefully examined and often needs modification before surgical placement of the implants, especially in the posterior regions of the mouth. The opposing teeth have often drifted or tipped into the opposing edentulous site as a result of improper or missing opposing occlusal contacts.

WWW.HIGHDENT.IR

Figure 12-9 **A,** A Misch Occlusal Analyzer is fabricated in three sizes as follows: 3/4-inch, 4-inch, and 5-inch sphere. The occlusal plane of the patient is evaluated before the restoration of the opposing arch. **B,** A press-form (vacuum) shell is placed over a duplicate study cast of the patient. The template and teeth are adjusted so the casts follow the Misch Occlusal Analyzer more accurately. **C,** The areas on the cast are marked to indicate the areas to modify intraorally. The modified template is inserted in the mouth, and the dental regions above the template are recontoured. **D,** Intraorally, the correction is performed using the template.

The CHS in the edentulous site may be significantly reduced as a result of posterior extrusion or exfoliation. The implant drills and implant body insertion often require a posterior crown height space of more than 8 mm from the ideal plane of occlusion, so the hand piece, drill, or implant may be inserted at the correct position and angulation.

The partially edentulous posterior ridge with facial resorption may require implant insertion more medial in relation to the original central fossa of the natural dentition. Enameloplasty of the stamp cusps of the opposing teeth is often indicated to redirect occlusal forces over the long axis of the implant body and may be determined with the diagnostic casts and modified in the mouth before the opposing arch impression and bite registration at the final impression appointment. Then, at the metal try-in or final prosthesis delivery, the final modifications of the opposing dentition are made.

The existing tooth and arch relationships do not need to be perfect before implant treatment. However, because implant dentistry always concerns the replacement of teeth, at least a partial rehabilitation process must occur in the patient before implant placement. The goal would be to identify and restore the prosthetic parameters within normal limits. The correct tooth positions should be first determined, so even if the total treatment time is extended over several years, at least each segment will aim toward a consistent goal. Too often the restoring dentist assumes the patient wants the cheapest or fastest treatment related to each treatment session. As a consequence, the mouth is restored one tooth at a time, fitting the restoration into the patient's present occlusal condition, which usually worsens over time and never improves on its own. As a result, after the patient has been in the same practice for several decades, the mouth is in poorer condition than when the patient started. Although it is easier to restore an entire mouth to the correct occlusal relationships at one time, it is also possible to obtain a similar result one tooth at a time, as long as each step proceeds along the predetermined course of treatment.

WWW.HIGHDENT.IR

SPECIFIC CRITERIA

After the five elements of the existing teeth (restorations) have been evaluated and modified where necessary, several other conditions may modify and hinder the course of implant treatment if overlooked (see Box 12-1 and Appendix A). These conditions should be considered before the final treatment plan is presented to the patient and include the following:

1. Lip lines
2. Maxillomandibular arch relationship
3. Existing occlusion
4. CHS
5. Temporomandibular joint status
6. Extraction of hopeless or guarded-prognosis existing teeth
7. Existing prostheses
8. Arch form (ovoid, tapering, square)
9. Natural tooth adjacent to implant site
10. Soft tissue evaluation of edentulous sites

A large number of these items may be evaluated on the mounted diagnostic casts. Others require the direct observation of the patient. A checklist is helpful to methodically gather the data, which directly influence the treatment plan.

LIP LINES

Lip in Repose

Lip positions are evaluated, including resting lip line, maxillary high lip line (smile), and mandibular low lip line (speech) in relation to the vertical position of the teeth. The lip line positions are especially noted if anterior teeth are to be replaced. The resting lip positions are highly variable, but in general are related to the patient's age. Older patients show less maxillary teeth at rest and during smiling, but demonstrate more mandibular teeth during sibilant sounds.[4] Prosthetic guidelines for incisal edge position established relative to esthetics, phonetics, and occlusion are applied.[1-8,29-35]

A common removable prosthetic guideline is a 1- to 2-mm incisal edge display with the lip at rest, regardless of the patient's age. Instead, the goal should be to position the prosthetic teeth in the most likely location for the patient's natural teeth. A male shows fewer teeth than a female of the same age. In a 50-year-old male, the maxillary incisal edge is often level with the upper lip at rest. This is a similar position for a 60-year-old female. The average upper lip is 20 to 22 mm for women and 22 to 26 mm for men. The maxillary incisal edge is usually at an average of 22 to 24 mm from the floor of the nose depending on the length and contour of the lip. For a short upper lip, the standard guideline for incisal edge of the central incisor would not be acceptable, because this would decrease the height of the maxillary arch.

The position of the maxillary incisor in relation to the maxillary lip and the age of the patient is much more variable than the position of the canine. The lip bow in the center of the upper lip rises several millimeters on some females and is barely obvious on others. The higher the lip bow, the more central incisor surface is seen on the patient, regardless of age. Men rarely exhibit an exaggerated lip bow and therefore have a more consistent incisor edge to lip position. The canine position at the corner of the lip is not affected by the lip bow effect. As such, it is a more consistent position and usually corresponds to the length of the resting lip position from 30 to 60 years of age in both males and females.[8]

In the natural dentition, the maxillary lip is most often longer than the incisal edge after the patient is 65 years old. However, most patients desire the maxillary teeth to be at least slightly visible. Therefore it is not unusual that the restoring dentist will concede this issue. Yet it is risky to extend the maxillary tooth position to decrease the age of the smile without considering the consequences of an increased crown height on moment forces. If pontics rather than implants support the anterior crowns, the poor biomechanical condition is magnified.

An alternative to increasing the length of the anterior teeth may be to increase the thickness of the alveolar ridge. This extra support brings out the lip and also raises the vermillion border. As a result, the teeth are not longer, but the border of the lip is higher. In addition, if the added width to the ridge is with autologous bone, replacing teeth with implants rather than pontics further improves the situation. The fuller maxillary lip may also look younger, because vertical age lines may also be reduced.

High Lip Line

The maxillary high lip line is determined while the patient displays a natural, broad smile.[36,37] There are three categories of maxillary lip lines: low, average (ideal), and high ("gummy"). The low lip line displays no interdental papilla or gingiva above the teeth during smiling. The high lip demonstrates all of the interdental papilla and more than 2 mm of tissue above the cervices of the teeth. The clinical characteristics of the average or ideal esthetic smile include a full length of crown exposure (crowns of normal height), a normal tooth position and alignment (lateral incisors may not be completely straight), a normal tooth form, the interdental papilla, and minimal gingival exposure over the cervicals of the teeth (lip at the free gingival margin of the centrals and canines) (Figure 12-10). Approximately 70% of the adult population has a smile line within a few millimeters of the free gingival margin.

WWW.HIGHDENT.IR

Figure 12-10 **A,** An ideal high smile line exposes the clinical crown and the interdental papillae. **B,** The patient in **A** has a full arch implant–supported FP-3 pink and white porcelain-metal prosthesis (shown here in a retracted position).

Figure 12-11 **A,** A low, high smile line shows no interdental papillae during smiling. **B,** The patient in **A** has a full arch implant–supported FP-3 prosthesis.

The FP-1 prosthesis in implant dentistry attempts to reproduce a normal crown contour. However, with a high lip position during smiling, this goal must also include the soft tissue drape around the crown. As a consequence, the aesthetic requirements are much more demanding and often mandate additional surgical steps to enhance the soft and hard tissues before the crown restoration. The selection of an FP-2 and an FP-3 fixed prosthesis is often based solely on the evaluation of the high lip line. An FP-2 prosthesis is easier to fabricate, because it requires fewer porcelain bake cycles.

Approximately 15 to 20% of adults have a low lip line and do not show the interdental papilla when smiling (more males than females) (Figure 12-11). In these patients, the soft tissue drape does not require a primary focus and can often be compromised with a FP-2 restoration, when the patient is notified before treatment. However, an average to high lip position during smiling contraindicates this restoration type because of poor cervical esthetics. The pink porcelain restoration (FP-3) to replace the soft tissue may be esthetic, but is rarely the treatment of choice for single-tooth replacement. On the other hand, in multiple missing adjacent anterior teeth, the pink porcelain is often the treatment of choice, because the soft tissue drape is usually unable to be ideal, even with bone and tissue grafts.

In a completely edentulous patient, the labial flange of the patient's existing denture may be removed and the lip position evaluated before the completed treatment plan of a fixed restoration. When the lip needs the support of the labial flange for esthetics, yet a fixed restoration is planned, onlay grafts with hydroxylapatite (HA), connective tissue, or autograft or allograft may be indicated to increase labial tissue thickness for proper lip support.

A gummy or high smile line occurs in 14% of the young female patients and 7% of young male patients (Figure 12-12).[36] The normal clinical crown width/height ratio is 0.86 for the central incisor, 0.76 to 0.79 for the lateral incisor, and 0.77 to 0.81 for the canine. If the patient demonstrates a band of gingiva over the cervical areas of the teeth, the height of the clinical crowns are evaluated, relative to their width. Esthetic crown lengthening is often a good option when the height of the central clinical crown is less than 10 mm (and the width is greater than 8 mm). Often the effect of crown lengthening is a dramatic improvement and may be accomplished at the same time as the implant surgery.

In patients with a high lip line who are missing all their anterior teeth, the prosthetic teeth can be made

WWW.HIGHDENT.IR

Figure 12-12 **A,** A high smile line exposes all of the clinical crown, the interdental papillae, and the full gingival margin above the teeth. **B,** This patient has a full arch implant–supported FP-3 porcelain-metal prosthesis.

longer (up to 12 mm) instead of the average 10 mm height to reduce the gingival display and result in a more esthetic restoration. Therefore the height of the maxillary anterior teeth is determined by first establishing the incisal edge by the lip in repose. Second, the high smile line determines the height of the tooth (from 9 to 12 mm). Third, the width of the anterior teeth is determined by the height/width ratios.

The cervical third of the maxillary premolars is also observed during a high smile line. It is not unusual to reveal the cervical third and gingiva of the premolar with a high lip line. These teeth should not appear too long or unnatural in height. Resorption may also cause the implants to be inserted more palatally in this area. The position of these crowns may then be too palatal and therefore affect the esthetic result. Bone grafts are the primary method to eliminate the need for ridge laps or the addition of pink porcelain at the gingiva. They are also indicated to reduce crown height.

Mandibular Lip Line

The mandibular low lip position is often neglected, with disastrous esthetic results. The mandibular incisors are more visible in middle age and older patients during speaking than maxillary teeth. In addition, lower central incisors are often visible in their incisal two thirds during exaggerated smiles.[37,38] Although the maxillary high lip line is evaluated during smiling, the mandibular low lip position should be assessed during speech. In pronunciation of the *S* sound, or sibilants, some patients may expose the entire anterior mandibular teeth and gingival contour. Patients are often unaware of this preexisting lip position and blame the final restoration for the display of the mandibular gingiva, or complain that the teeth look too long. Therefore it is recommended to make the patient aware of these existing lip lines before treatment and emphasize that these lip positions will be similar after treatment. An FP-3 mandibular restoration may be indicated to restore the patient with a low mandibular lip position.

MAXILLOMANDIBULAR ARCH RELATIONSHIP

After the maxillary anterior teeth position, OVD, and mandibular anterior teeth position are evaluated, the maxillomandibular relationships are assessed in the vertical, horizontal, and lateral planes. An improper skeletal position may be modified by orthodontics or surgery. It is far better to discuss these options with the patient before implant surgery, because the implant placement may compromise the final prosthetic result if the arch positions are altered after implant insertion. Compromises of the final result should be discussed with patients when orthognathic surgery or orthodontic therapy is declined by patients with skeletal discrepancies.

Arch relationships are often affected in edentulous ridges. The anterior and posterior edentulous maxilla resorbs toward the palate after tooth loss.[39] The width of the alveolar ridge decreases 40% within a few years, primarily at the expense of the labial plate. Consequently, implants are often placed lingual to the original incisal tooth position. The final restoration is then overcontoured facially to restore the incisal two thirds in the ideal tooth position for esthetics. This results in a cantilevered force on the implant body. The maxilla is affected more often than the mandible, because the incisal edge position in the esthetic zones cannot be modified and is dictated by esthetics, speech, lip position, and occlusion. Anterior cantilevered crowns from maxillary anterior implants often require additional implants splinted together and an increase in the anteroposterior distance between the most distal to most anterior implant positions to compensate for the increased lateral loads and moment forces, especially during mandibular excursions.

An anterior cantilever on implants in the mandibular arch may correct an Angle's skeletal Class II jaw relationship. The maxillary anterior teeth support the lower lip at rest in both Angle's skeletal Class I and Class

WWW.HIGHDENT.IR

II relationships. A traditional complete mandibular denture cannot extend beyond the anatomical support or neutral zone of the lips without decreasing stability of the prosthesis. However, with implants, the denture teeth may be set in a more ideal esthetic and functional position. The anterior cantilever in the mandible is also dependent upon adequate implant number and anteroposterior (A-P) distance between the splinted implants. To counteract the anterior cantilever effect, the treatment plan should provide increased implant support by increasing the surface area by number, size, design, or A-P implant position. In these cases, an RP-4, designed to prevent food impaction, may facilitate daily care and help control the occlusal forces compared with an FP-3 prosthesis.

The palatal resorption pattern of the maxilla, paired with the anterior rotation of the mandible in long-term, complete denture patients may mimic a Class III relationship on a lateral cephalometric radiograph. However, in this condition, Class III mandibular mechanics do not apply (primarily vertical chewers with little to no anterior excursions during mastication or parafunction). On the contrary, these patients exhibit a full range of mandibular excursions and can contribute significant lateral forces on the maxillary restoration, which is cantilevered off the implant base to obtain a Class I esthetic restoration. Therefore additional splinted implants are suggested in the maxilla with the widest A-P distance available. This usually requires sinus grafts and posterior implants in the first or second molar position splinted to the anterior implant support.

Transversal arch relationships include the existence of posterior crossbites, which occur frequently in implant dentistry. Edentulous maxillary posterior arches resorb palatally and medially after tooth loss. Sinus grafts can restore available bone height, but the ridge still remains medial to the opposing mandibular tooth central fossa. This is especially pronounced when opposing a Division C–h or moderate atrophic mandible, because the mandible widens after the residual alveolar ridge resorbs. For example, when mandibular implants are used in C–h bone volume for implant support opposing a complete denture, the posterior teeth may be set in crossbite (especially when out of an esthetic zone) to decrease the moment forces developing on the maxillary posterior teeth, causing denture instability.

EXISTING OCCLUSION

Maximal intercuspation (MI) is defined as the complete intercuspation of the opposing teeth independent of condylar position, sometimes described as the best fit of teeth regardless of the condylar position.[9] *Centric occlusion* is defined as the occlusion of opposing teeth when the mandible is in centric relation (CR).[9] This may or may not coincide with the tooth position of maximal intercuspation. Its relationship to CR—a neuromuscular position independent of tooth contact with the condyles in an anterior, superior position—is noteworthy to the restoring dentist because of the potential need for occlusal adjustments to eliminate deflective tooth contacts and the evaluation of their potential noxious effects on the existing dentition and for the planned restoration. Correction of the problems before treatment presents many advantages and may follow a variety of approaches depending on the severity of the incorrect tooth position: selective odontoplasty (a subtractive technique), restoration with a crown (with or without endodontic therapy), or extraction of the offending tooth. The existing occlusion is best evaluated with facebow mounted diagnostic casts and open-mouth bite registration in CR.

Controversy exists as to the necessity to have MI harmonious with CR occlusion. A vast majority of patients around the world do not have such a relationship, yet they do not exhibit clinical pathology or accelerated tooth loss. Therefore it is difficult to state these two positions must be similar. What is important is to evaluate the existing occlusion and the mandibular excursions to consciously decide whether the existing situation should be modified or be maintained. In other words, dentists should determine whether they are going to ignore or control the occlusion of the patient (Figure 12-13).

As a general rule, the more teeth replaced or restored, the more likely the patient is restored to CR occlusion. For example, if a completely edentulous mandible is to be restored with an implant-supported fixed prosthesis, the CR occlusion position provides consistency and reproducibility between the articulator and the intraoral condition and slight changes in OVD to position anterior implant abutments in a more favorable restoration position may be studied and implemented on the

Figure 12-13 The existing occlusion is evaluated to decide whether the maximal intercuspation (MI) is similar to centric occlusion (the mandible is in centric relation). The mandibular excursions are also evaluated. The lack of canine contact and premature first premolar contact and the uneven occlusal plane indicated correction of the occlusion before final reconstruction.

WWW.HIGHDENT.IR

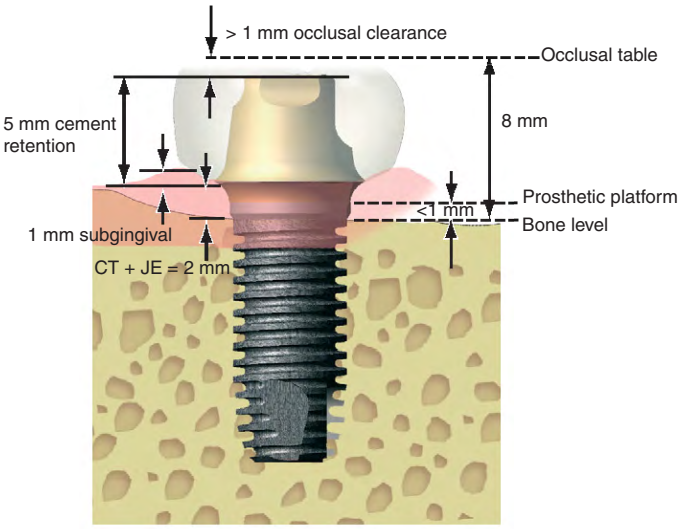


Figure 12-14 The crown height space (CHS) is measured from the occlusal plane to the level of the bone.

articulator without the need to record the new occlusal vertical position on the patient.

On the other hand, when one anterior tooth is being replaced, the existing MI position is often satisfactory to restore the patient, even though a posterior interference and anterior slide into full interdigitation may be present. The underlying question that helps determine the need for occlusal correction prior to restoration of the implant patient is the observation of negative symptoms related to the existing condition. This may include temporal mandibular joint conditions, tooth sensitivity, mobility, tooth fractures or abfraction, or porcelain fracture. The fewer and less significant the findings, the less likely an overall occlusal modification is required before restoration of the patient. However, to properly assess these conditions, the dentist must not ignore them before treatment.

CROWN HEIGHT SPACE

The *interarch distance* is defined as the vertical distance between the maxillary and mandibular dentate or edentate arches under specific conditions (e.g., the mandible is at rest or in occlusion).[9] A dimension of only one arch does not have a defined term in prosthetics; therefore Misch proposed the term *crown height space* (CHS).[40] The CHS for implant dentistry is measured from the crest of the bone to the plane of occlusion in the posterior region and the incisal edge of the arch in question in the anterior region (Figure 12-14). The ideal CHS for an FP-1 fixed implant prosthesis should range between 8 and 12 mm. This space accounts for the biological width, abutment height for cement retention or prosthesis screw fixation, occlusal material strength, esthetics, and hygiene considerations around the abutment crowns. Removable prostheses often require more than 12 mm of CHS for denture teeth and acrylic resin base strength, attachments, bars, and oral hygiene considerations.[41,42]

Biomechanic Consequences of Excessive Crown Height Space

Mechanical complication rates for implant prostheses are often the highest of all complications reported in the literature [43,44] and are often caused by excessive stress applied to the implant-prosthetic system. Implant body or component failure may occur from overload and result in prosthesis failure and bone loss around the failed implants.[43] Crestal bone loss may also be related to excessive forces and often occurs prior to implant body fracture.

The biomechanics of CHS are related to lever mechanics. The issues of cantilevers and implants were demonstrated in the edentulous mandible where the length of the posterior cantilever directly related to complications or failure of the prosthesis.[44] Rather than being a posterior cantilever, the CHS is a vertical cantilever and therefore is also a force magnifier.

When the direction of a force is in the long axis of the implant, the stresses to the bone are not magnified in relation to the CHS (Figure 12-15). However, when the forces to the implant are on a cantilever or a lateral force is applied to the crown, the forces are magnified in direct relationship to the crown height. As discussed in Chapter 6, Bidez and Misch evaluated the effect of a cantilever on an implant and its relation to crown height.[45,46] When the crown height is increased from 10 to 20 mm, two of six of these moments are increased 200%. When the available bone height is decreased, the CHS is increased.

An angled load to a crown also magnifies the force to the implant. A 12-degree force to the implant increases

WWW.HIGHDENT.IR

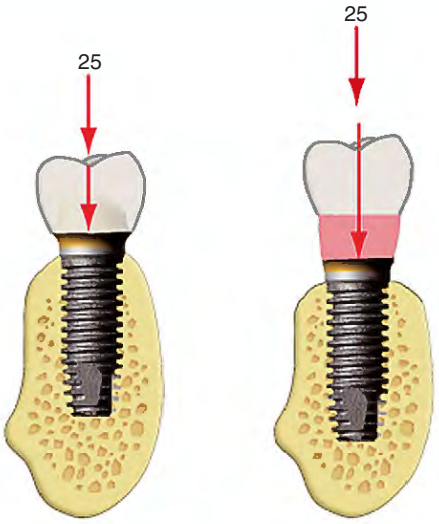


Figure 12-15 The crown height is not a multiplier of force when the load is in the long axis of the implant. However, any angled force or cantilever increases the force and the crown height magnifies the effect.

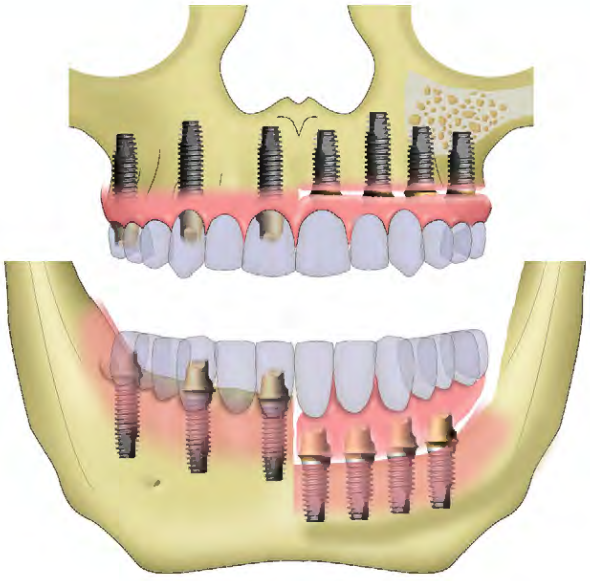


Figure 12-16 The greater the crown height space (CHS), the more implants are required to restore the patient (right side of drawing). The less the CHS (left side), the fewer the implants to restore the patient.

the force by 20%. This increase in force is further magnified by the crown height. For example, a 12-degree angle with a 100-N force will result in a force of 315 N-mm on a crown height of 15 mm.[46] Maxillary anterior teeth are usually at an angle of 12 degrees or more to the occlusal planes. Therefore even implants placed in an ideal position are usually loaded at an angle. In addition, maxillary anterior crowns are often longer than any other teeth in the arch, so the effects of crown height cause greater risk. The angled force to the implant may also occur during protrusive or lateral excursions, because the incisal guide angle may be 20 degrees or more. Anterior implant crowns will therefore be loaded at a considerable angle during excursions compared with the long axis of the implant. As a result, an increase in the force to maxillary anterior implants should be compensated for in the treatment plan.

Most forces applied to the osteointegrated implant body are concentrated in the crestal 7- to 9-mm bone, regardless of implant design and bone density. Therefore implant body height is not an effective method to counter the effect of crown height. Moderate bone loss before implant placement may result in a crown height–bone height ratio greater than 1, with greater lateral forces applied to the crestal bone than in abundant bone (in which the crown height is less). A linear relationship exists between the applied load and internal stresses.[47,48] Therefore the greater the load applied, the greater the tensile and compressive stresses transmitted at the bone interface and to the prosthetic components. The greater the CHS, the greater number of implants usually required for the prosthesis, especially in the presence of other force factors. This is a complete paradigm shift to the concepts advocated originally with many implants in greater available bone and small crown heights and fewer implants with greater crown heights in atrophied bone (Figure 12-16).

Box 12-2 Excessive Crown Height Space: Treatment Planning Options to Decrease Stress

- Shorten cantilever length
- Minimize buccal and lingual offset loads
- Increase the number of implants
- Increase the diameters of implants
- Design implants to maximize the surface area
- Fabricate removable restorations (less retentive) and incorporate soft tissue support
- Remove the removable restoration during sleeping hours to reduce the noxious effects of nocturnal parafunction
- Splint implants together, regardless of whether they support a fixed or removable prosthesis

Because an increase in the biomechanical forces are in direct relationship to the increase in CHS, the treatment plan of the implant restoration should consider stress-reducing options whenever the CHS is increased. Methods to decrease stress are presented in Box 12-2.[40,41]

Excessive Crown Height Space

Crown height space is excessive when greater than 15 mm. Treatment of excessive CHS as a result of vertical resorption of bone before implant placement includes surgical methods to increase bone height or stress reduction methods to the prosthesis. Several surgical techniques may be considered to increase

WWW.HIGHDENT.IR

bone height, including block onlay bone grafts, particulate bone grafts with titanium mesh or barrier membranes, interpositional bone grafts, and distraction osteogenesis.[41,42,49]

Bone augmentation may be preferred to prosthetic replacement. Surgical augmentation of the residual ridge height will reduce the CHS, improve implant biomechanics, and often permit the placement of wider body implants with the associated benefit of increased surface area. Although prosthetics is the most commonly used option to address excess CHS, it should be the last option employed. Gingiva-colored prosthetic materials (pink porcelain or acrylic resin) on fixed restorations or changing the prosthetic design to a removable restoration should often be considered when restoring excessive CHS.

In the maxilla, a vertical loss of bone results with a more palatal ridge position. As a result, implants are often inserted more palatal than the natural tooth position. Removable restorations have several advantages under these clinical circumstances. The removable prosthesis does not require embrasures for hygiene and may be removed during sleep to decrease the effects of an increase in CHS on nocturnal parafunction. It may also improve the deficient lip facial support. The overdenture may have sufficient bulk of acrylic resin to permit denture tooth placement without infringement of the substructure and to decrease the risk of prosthesis fracture. However, it has identical requirements to a fixed prosthesis, because it is rigid during function (hidden cantilever situation).

In the case of removable prostheses with mobility and soft tissue support, two prosthetic levers of height should be considered. The first is the height of the attachment system to the crest of the bone. The greater the height distance, the greater the forces applied to the bar, screws, and implants. The second CHS to consider is the distance from the attachment to the occlusal plane. This distance represents the increase in prosthetic forces applied to the attachment. Therefore in a CHS of 15 mm, an O-ring may be 7 mm from the crest of bone, resulting in a lever action of 7 mm applied to the implants. The distance from the rotation point of the O-ring to the occlusal plane may be an additional 8 mm. Under these conditions, a greater lever action is applied to the prosthesis than to the implant interface. This results in increased instability of the restoration under lateral forces.[42]

A CHS greater than 15 mm means a large amount of metal must be used in the substructure of a traditional fixed restoration to keep porcelain to its ideal 2-mm thickness (Figure 12-17). Control of surface porosities of metal substructures after casting becomes increasingly difficult, because their different parts cool at different rates.[50] If not controlled properly, both of these factors increase the risk of porcelain fracture after loading.[51] For excessive CHS, considerable weight of the prosthesis (approaching 3 oz of alloy) may affect maxillary trial placement appointments, because the restoration does not remain in place without the use of adhesive. Because noble metals must be used to control alloy's heat expansion or corrosion, the cost of such implant restorations is dramatically increased. Proposed methods to produce hollow frames to alleviate these problems include using special custom trays to achieve a passive fit, which can double or triple the labor costs.[52]

An alternative method to fabricate fixed prostheses in situations of CHS 15 mm or greater is the fixed complete denture or hybrid prosthesis, which has a smaller metal framework, denture teeth, and acrylic resin to join these elements together (Figure 12-18). This type of fixed prosthesis is often indicated for implant restorations with a large CHS. On occasion, undercontoured interproximal areas are designed by the laboratory in restorations of large CHS space to assist oral hygiene, and have been referred to as *high-water restorations*. This is an excellent method in the mandible;

Figure 12-17 A large crown height space with a traditional porcelain-metal prosthesis requires a large amount of metal in the substructure with increased risk of complications such as porosities and fracture during cooling.

Figure 12-18 A hybrid prosthesis uses a cast metal substructure (smaller in size than porcelain-to-metal), acrylic, and denture teeth to restore the patient.

WWW.HIGHDENT.IR

however, it results in food entrapment, affects air flow patterns, and may contribute to speech problems in the anterior maxilla.

Because crown height is a considerable force magnifier, the greater the crown height, the shorter the prosthetic cantilever should extend from the implant support system. In crown heights of more than 15 mm, no cantilever should be considered unless all other force factors are minimal. The occlusal contact intensity should be reduced on any offset load from the implant support system. Occlusal contacts in CR occlusion may even be eliminated on the most posterior aspect of a cantilever. In this way, a parafunction load may be reduced, because the most cantilevered portion of the prosthesis is only loaded during functional activity while eating food.

Reduced Crown Height Space

Issues related to CHS are accentuated by an excessive CHS that places more forces on the implant and prosthetic system, and reduced CHS makes the prosthetic components weaker. A reduced CHS has biomechanical issues related to a reduced strength of implant material or prosthetic components, an increased flexibility of the material, and a reduction of retention requirement of the restoration. The fatigue strength and flexure of a material is related to its radius to the power of 4. In fixed restorations, the flexure of the reduced-diameter material may cause porcelain fracture, screw loosening, or uncemented restorations. Therefore in the situation of reduced CHS, material failures are more likely (Box 12-3).

Skeletal discrepancies (deep bite), reduced OVD from attrition or abrasion, minimal bone atrophy after tooth loss, and supraeruption of unopposed teeth may all result in less than ideal space for prosthetic replacement of the dentition. Traditional prosthetic and restorative procedures are indicated to restore the proper OVD and plane of occlusion. However, on occasion, even when the opposing arch is corrected, the CHS may still be less than ideal (<8 mm). The 8-mm requirement for CHS consists of 2 mm occlusal material space, 4-mm minimum abutment height for retention, and 2 mm above the bone for the biological width dimension (which does not include the sulcus, as a crown margin may be 1 mm subgingival for retention or esthetics).

Box 12-3 Reduced Crown Height Space

1. Structural integrity problems of a restoration increase with a reduced CHS.
2. Surgical procedures during implant placement may increase a CHS.
3. Complications of an insufficient CHS may be increased by the surgical position of the implant (i.e., poor angulation, implant platform several millimeters above the bone).
4. Different implant systems have a different minimum CHS related to the height of the prosthetic components.

CHS, Crown height space.

When the reduced OVD is in partially edentulous patients, the OVD may be restored by orthodontics, which is the preferred method. This correction may also require a surgical orthognathic surgery, such as a LeFort I osteotomy and superior repositioning. However, prosthetics is a common approach and may involve an entire arch.

When the opposing teeth are in the correct position and the CHS is insufficient, additional space may be gained surgically with osteoplasty and soft tissue reduction of one arch, provided adequate bone height remains after the procedure for predictable implant placement and prosthetic support (Figure 12-19). If a removable implant-supported prosthesis is planned, an aggressive alveoloplasty should often be performed after tooth extraction to provide adequate prosthetic space.

Additional prosthetic space can also be obtained in many clinical situations by soft tissue reduction, especially in the maxilla. Soft tissue reduction should be performed in conjunction with second-stage surgery if the implants heal in a submerged location. This allows the thicker tissue to protect the implants from uncontrolled loading by a soft tissue–supported prosthesis during healing. If the implants heal permucosally, the reduction procedures should be done during implant placement. Soft tissue reduction procedures may include gingivectomy, removal of connective tissue, or apical repositioning of flaps. Efforts should be made to maintain adequate keratinized tissue around the implants. Soft tissue reduction also has the benefit of decreased probing depths around the implants. However, the definition of CHS is from the bone to the occlusal plane, and, therefore, although the prosthetic space is improved, the CHS remains similar when only soft tissue reduction is performed. Too little CHS can be further complicated when the surgeon places the implant above the bone.

When the CHS is less than ideal, the following prosthetic parameters should be identified[42]:

1. Available space
2. Abutment taper
3. Surface area of abutment
4. Cement type
5. Surface finish
6. Occlusal topography and material
7. Load on final restoration
8. Fit of restoration to abutment
9. Retention of prosthesis
10. Implant manufacturer
11. Implant platform to occlusal plane dimension

The consequences of insufficient CHS include a decrease in abutment height (which may lead to

WWW.HIGHDENT.IR

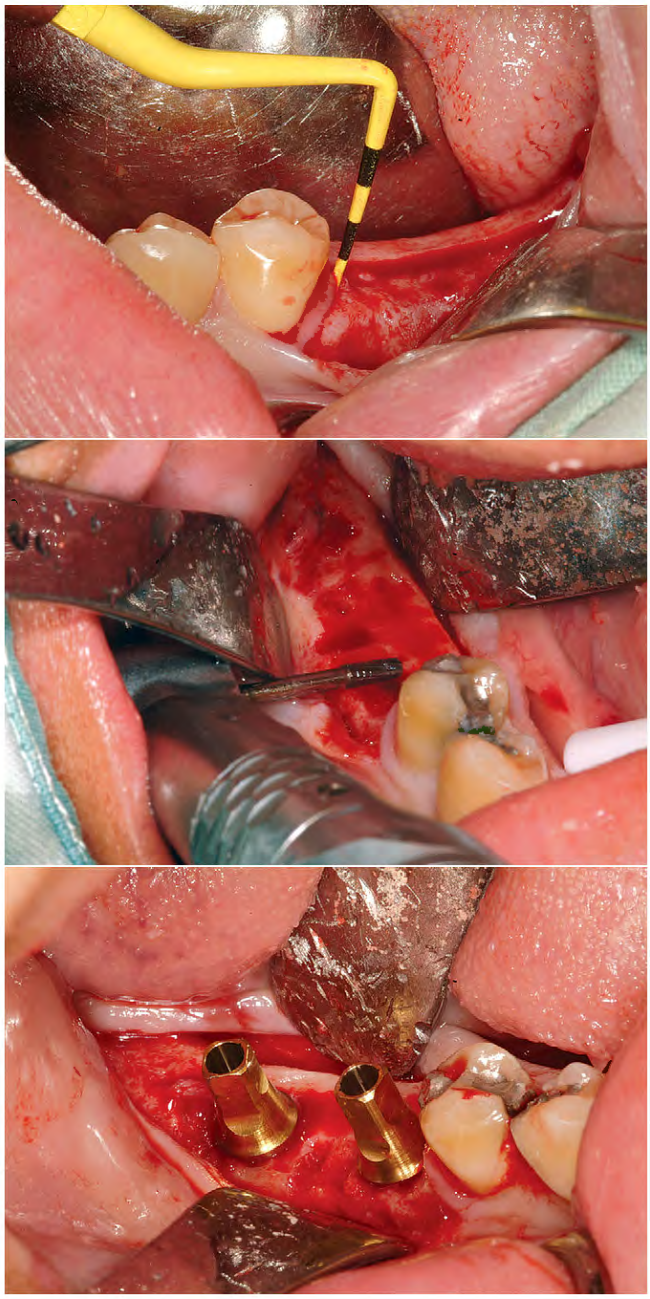


Figure 12-19 **A,** A reduced crown height space (CHS) results in short abutments, less cement retention, and increase of flexibility of the metal in the prosthesis. A mandibular square arch form has a small anteroposterior (A-P) distance. **B,** An osteoplasty increases the CHS before implant insertion. **C,** The implants may be positioned with an increase in CHS and fewer prosthetic complications.

inadequate retention of the restoration), inadequate bulk of restorative material for strength or esthetics, and poor hygiene conditions compromising long-term maintenance.[53] In addition, the final restoration flexes inversely to the cube of the thickness of material. A fixed prosthesis half as thick will flex eight times as much and will further result in loss of cement retention, loosening/fracture of fixation screws, or porcelain fracture.[54] Inadequate thickness of occlusal porcelain or acrylic, or unsupported occlusal material caused by inadequate metal substructure design, may also result in complications such as component fracture.

Minimum restorative requirements vary in function of the implant system. The minimum restoration space may be determined by limiting the occlusal material to 1 mm and reducing the abutment height to the top of the retaining screw. The smallest minimum restoration spaces correspond to 4.21 mm for 3I Osseotite, 4.35 mm for Replace Select (Nobel BioCare), 4.5 mm for Biohorizons, and 4.56 mm for Frialit 2 systems. The greatest restoration space requirements are found in Astra (6.6 mm), Lifecore (6.84 mm), and Straumann (7.05 mm) systems.[42]

When fabricating a cemented restoration, the restoration technique (indirect versus direct) may be influenced by the CHS. Because additional abutment height for retention may be gained by a subgingival margin, the indirect technique (making an implant body level impression) may have an advantage over a direct intraoral impression. An implant body level impression permits the subgingival restoration to be placed more than 1 mm subgingival, with greater accuracy, and therefore represents benefit in a reduced CHS situation, especially when the soft tissue is several millimeters thick. The indirect technique is also used for custom abutments, which can be designed with increased diameter to increase the overall surface area for retention. A custom abutment may also be fabricated to decrease the total occlusal convergence angle to increase retention for cemented prostheses.

The retention and resistance difference between a 3-mm-high and a 5-mm-high implant abutment may be as great as 40% for a 4.5-mm-diameter abutment. Less than 3 mm of abutment height indicates a screw retained crown, 3 to 4 mm requires a screw retained or resin-cemented restoration, and greater than 4 mm of abutment height allows the operator's preference. Splinting implants together, regardless of whether they are screw retained or cement retained, can also increase retention.

Conditions such as cement hardness, surface condition of the abutment, and occlusal material (porcelain versus metal) are also to be considered in limited CHS situations. The occlusal material is important to consider in reduced CHS for two primary reasons. When metal is used as the occluding surface, it is possible to provide greater retention for the prosthesis as a result of an increase in abutment height. The abutment height may be greater because the occlusal space required above the abutment is only 1 mm, whereas porcelain requires 2 mm of occlusal space and acrylic resin requires 3 mm or more. Another factor is the strength of the material. Metal occlusal surfaces provide the greatest resistance to fracture and should be considered when there is limited CHS. When a screw is used to retain the crown, the strength of occlusal porcelain is reduced by 40%. Acrylic resin requires the most dimension for strength and

WWW.HIGHDENT.IR

much more likely to fracture when the CHS is limited. This is why acrylic resin overdentures require more CHS than a porcelain-metal fixed prosthesis.

The surgeon may magnify the prosthetic problem of limited CHS by placing the implant at an angle to the ideal position. Angled abutments lose surface area of retention from the abutment screw hole and further compromise the limited space conditions. In addition, a 30-degree taper on an abutment to correct parallelism loses more than 30% of the abutment surface area and dramatically decreases the retention for the abutment.

Overdentures also exhibit greater complications in situations of reduced CHS. Removable prostheses have space requirements for elements such as a connecting bar, type, and position of attachments and restorative material (metal versus resin). According to English, the minimum CHS for individual attachments is 4.5-mm CHS for locator-type attachments and between 12 and 15 mm for a bar and O-rings.[55] Marinbach reports the ideal CHS for removable prostheses is >14 mm and the minimum height is 10.5 mm.[42] The lowest possible profile attachment should be used in situations of reduced CHS to fit within the contours of the restoration, provide greater bulk of acrylic resin to decrease fracture, and allow proper denture tooth position without the need to weaken the retention and strength of the resin base.

Overdenture bars may be screw retained or cement retained. The most common method of retention for a fixed prosthesis is cement retention. The most common method of bar retention by almost the same percentage for overdentures is screw retention. Yet the advantages of cement retention for a fixed prosthesis also apply to an overdenture bar. Therefore, in minimum CHS situations, the screw-retained bar has a clear advantage, but in ideal to excessive CHS situations, the cemented bar should be considered.

TEMPOROMANDIBULAR JOINT

The temporomandibular joint (TMJ) may exhibit signs and symptoms of dysfunction. Symptoms include pain and muscular tenderness experienced by the patient. Noises or clicking in the joint during opening, deviation of the mandible during jaw opening, and limited jaw movements are signs of potential dysfunction observed during the patient examination. Patient complaints or signs gathered during this phase should be carefully evaluated before further reconstructive treatment.

Palpation of the temporalis, masseter, and internal and external pterygoid muscles is part of the TMJ examination. The muscles should not be tender during this process. Parafunction may contribute to TMJ disorders and is a direct source of muscle tenderness. Under these conditions, the muscles are usually hypertrophied as a result of the excess occlusal forces. The masseter and temporalis muscles are easily palpated. The lateral pterygoid muscle is often overused in this patient profile, yet is difficult to palpate. The ipsilateral medial pterygoid muscles can be as diagnostic and is easier to evaluate in the hamular notch region. It acts as the antagonist to the lateral pterygoid muscle in hyperfunction and, when tender, is a good indicator of overuse of either muscle.

Deviation to one side on opening indicates muscle imbalance on the same side as the deviation and possible degenerative joint disease.[56,57] The patient should also be able to perform unrestricted mandibular excursions. Maximal opening is noted during this exam and is normally greater than 40 mm from the maxillary incisal edge to the mandibular incisal edge in an Angle's skeletal Class I patient. If any horizontal overjet or vertical overbite exists, it is subtracted from the 40-mm minimum opening measurement.[58] The range of opening without regard to overlap or overbite ranges from 38 to 65 mm in men and 36 to 60 mm in women, from one incisal edge to the other.

The practitioner is encouraged to carefully evaluate the TMJ status. It is beyond the scope of this text to address the methods of treatment of TMJ dysfunction. However, many patients with soft tissue–borne prostheses and TMJ dysfunction benefit from the stability and exacting occlusal aspects that implant therapy provides. As such, these patients may benefit from implant support to improve their condition.

EXTRACTION OF TEETH WITH HOPELESS OR GUARDED PROGNOSIS

Maintaining natural teeth in health, function, and esthetics is a primary goal of all dentists. In the past, the maintenance of a natural tooth was paramount, because tooth replacement techniques were costly and not as predictable as repairing the natural tooth. However, advanced repair procedures such as apicoectomy, furcation treatment, or functional crown lengthening may have a lower success rate than an implant to replace the tooth. Therefore, on occasion, the natural tooth is significantly compromised and extraction with replacement of an implant is indicated. A tooth may be considered for extraction because of prosthetic, endodontic, or periodontal considerations. On rare occasions, extraction is considered rather than orthodontics to restore the teeth in a more esthetic or functional position.

Caries on a natural tooth is most often able to be removed and the tooth restored. However, on occasion, the tooth is unrestorable after the decay is removed. A prosthetic axiom is to have at least 1.5 to 2 mm of tooth structure for a crown with a cervical ferule effect. In addition, adequate retention and resistance from the tooth preparation should exist. As a result of the caries, endodontic therapy, post and core, and functional crown lengthening may be required. Thus the procedures to save the tooth are costly and not predictable.

WWW.HIGHDENT.IR

Figure 12-20 **A,** A devital tooth with an endodontic lesion of more than 5 mm has less than an 88% treatment success rate. **B,** The postoperative follow-up of the tooth indicates endodontic success. It may now be restored with less risk of failure. If not successful, extraction rather than retreatment is considered, because the retreatment success rate is 65%.

On occasion, the end result may not be predictable or esthetically pleasing. For example, when a central incisor requires considerable functional crown lengthening, the gingival margin may be compromised and have a poor esthetic result.

A patient with a history of high decay rate with recurrent caries under a crown, requiring endodontics with a post and core before restoration, may also be better served with an extraction and implant insertion. The repeated recurrent decay can be eliminated, at least for that tooth, with an implant. When caries extends within the root canal, the outer structural walls of the natural root may be too thin for a predictable post or restoration. As a result, extraction and implant insertion has a better prognosis.

Endodontic considerations may also consider tooth extraction rather than traditional treatment. When the root canal cannot be accessed because of abnormal root anatomy, an extraction and implant insertion may be considered. On occasion, the endodontic procedure is compromised or an apicoectomy has a moderate to high risk of paresthesia. An implant after extraction may be less invasive and have less risk of paresthesia. A tooth with a "split root" syndrome may have undergone root canal therapy, with pain still present during function. Rather than an apicoectomy, an extraction and implant insertion is usually a definitive treatment that eliminates more predictably pain during function.

A vital tooth has endodontic success rates above 93%, whereas a nonvital tooth has an 89% rate. A large periapical lesion (larger than 5 mm) compromises the success rate of traditional endodontics. A nonvital tooth with large periapical pathology has a success rate of 78% (Figure 12-20).[59,60] Therefore endodontic therapy should be performed and evaluated over several months before post, core, and crown treatment. A retreatment of an endodontic tooth with a periapical lesion has a reported success rate of 65%. As a result, consideration for extraction and implant replacement may be considered for devital teeth with more than 5-mm apical radiolucencies that do not resolve or endodontic retreatment when periapical lesions are present.

The existing teeth in a partially edentulous patient should be evaluated for longevity and existing disease. Implant dentistry has modified the treatment plan philosophy in these patients. Advanced periodontal disease may be addressed with extraction of questionable abutments more frequently than in the past, provided the resulting edentulous area offers sufficient bone for predictable endosteal implant placement and a predictable prognosis.[61] Herodontics are discouraged when the prognosis is poor or failure of treatment may result in inadequate bone for implant placement. The cost of the questionable periodontal treatment may result in the patient's inability to afford the more predictable implant therapy later. This is especially noted when the existing available bone around the tooth roots is compromised in height, especially in the posterior mandible. Unsuccessful periodontal treatment and continual bone loss may render the remaining bone inadequate for extraction and placement of implants. As

WWW.HIGHDENT.IR

a result, when 10 mm of bone is all that remains from the mandibular canal to the remaining bone around the periodontally involved teeth, consideration is given to the projections of periodontal therapy.

The etiology of furcation involvements includes bacteria as well as plaque in the furca with extension of inflammation in the region with loss of interradicular bone. This leads to a progressive and site-specific loss of attachment in most individuals. A first molar furcation entrance cannot be accessed with hand instruments 58% of the time.[62] In addition, pulpal pathoses with accessory canals in the furca may cause problems. Vertical root fracture after endodontic therapy may also occur.

Furcation treatment of molars may include root amputation. The lowest success rate for root resection was found on mandibular distal root resection (75%) (Figure 12-21). Even when successful, the missing root indicates endodontics, core and crown, and the replacement of the distal root. An implant replaces the whole tooth with a higher success rate and often lower cost. A maxillary molar that has lost bone to the furcation has lost almost 30% of the root surface area of support. Therefore when a tooth has a short root or is multirooted, a considerable functional crown lengthening may compromise the remaining support or result in a furcation involvement. The endodontics, post and core, and functional crown lengthening may not be as predictable as extraction and implant insertion. In addition, the cost of conventional treatment may be twice the cost of an implant.

Traditional methods to save a tooth have increased in cost over the years. Multirooted endodontic therapy now approaches the cost of an implant surgery. When functional crown lengthening and endodontic post treatment is also required, the fees are usually greater than extraction and implant insertion. Therefore part of the equation of whether to extract or treat a tooth may also relate to the cost of the service provided. The natural molar tooth that requires endodontics, root amputation, post and core placement, and nevertheless a compromised root with poor root surface area may be cost prohibitive for the service provided.

An implant in the site after tooth extraction is often less expensive and more predictable long term. However, the recent trend to extract teeth with a good prognosis after endodontic or periodontal treatment is discouraged. Implants are not yet 100% predictable, and implants should not be substituted for natural teeth presenting a good or even fair prognosis. Table 12-1 summarizes the decision-making protocol involving a natural tooth abutment.

The dentist evaluates the natural teeth for their quality of health with widely used prosthetic, periodontal, and endodontic indexes. After this is accomplished, the dentist obtains an estimate of longevity and decides whether to extract or to treat and maintain the tooth, following a 0-, 5-, or 10-year rule. If the natural tooth has a favorable prognosis for more than 10 years, it is included in the treatment plan. The decision to use it or not as an abutment requires additional information, but few reasons support removal of the tooth to restore the partially edentulous patient.

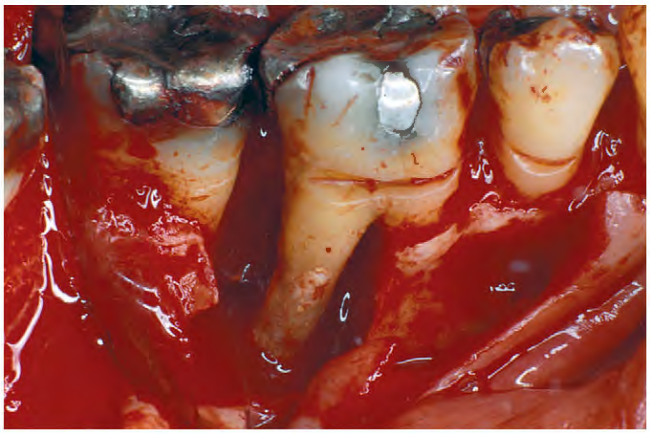


Figure 12-21 A mandibular first molar with a distal root resection generally has a success rate of 75%. Even when successful, the mesial root requires endodontic treatment, core, and crown and the distal root needs replacement. Therefore an implant or three-unit fixed partial denture is indicated. It is more cost-effective to extract, implant, and fabricate one crown, even when bone grafting is indicated.

Table 12-1 Extract or Maintain Natural Tooth: 0-, 5-, and 10-Year Rule

Prognosis	Protocol
>10 years	Keep the tooth and restore as indicated.
5 to 10 years	Independent implant restoration. If the natural tooth must be included with implants in the restoration, make it a "living pontic" by adding implants on each side and splint together.
<5 years	Extract the tooth and graft the site or consider an implant.

If the natural tooth prognosis (after periodontal, endodontic, or restorative therapy when necessary) is in the 5- to 10-year range, independent implant-supported prostheses are indicated. If the edentulous region does not provide sufficient implant support for an independent restoration, then placement of as many implants as possible around the tooth, with treatment alternatives that will permit removal of the tooth without sacrificing the restoration, is indicated. For example, a coping may be placed on the tooth with a 5- to 10-year prognosis, and the tooth may act as "living pontic" in the final restoration, surrounded by sufficient implant support. Whether the tooth is missing or present does not matter. In this way, the prosthesis may be removed in the future and the tooth may be removed (if indicated). The prosthesis essentially is maintained without compromise.[64-66]

WWW.HIGHDENT.IR

The copings on the teeth should be designed with a different path of insertion than fixed partial dentures and should be cemented with permanent cement, whereas the fixed implant prosthesis usually is cemented with a weaker (soft access) or temporary cement. Thus the fixed partial denture path of removal differs from that of the natural tooth coping and, along with the weaker cement, allows the prosthesis to be removed while the coping remains permanently cemented on the tooth. The preparation of copings on natural teeth often requires additional removal of tooth structure to prevent overcontoured restorations and as a consequence may mandate endodontic therapy.

If hygiene is poor with patients with a high caries index or with Grade II or III furca involvement in molars, the tooth most often is considered in the 0- to 5-year category and is considered for extraction, especially when other teeth in the same quadrant are missing or hopeless, or only 8 to 10 mm of bone remains between the crest of bone and the opposing landmark.

A less than 5-year prognosis for a natural tooth adjacent to an edentulous site, despite restorative or periodontal therapy, warrants extraction of the tooth, with grafting and planning for additional implant abutment support as part of the initial treatment plan. This treatment scenario may often be faster, easier, and less costly over a 5- to 10-year period compared with maintaining a questionable adjacent tooth.

Mandibular molars with Grade I furcation involvement often are placed in the 5- to 10-year prognosis category. However, maxillary molars are at higher risk of furca complications and are lost 33% of the time within 5 years. Mandibular molars have a 20% failure at this same reference time. After the molar has a Grade II or higher furca, it has a greater risk of failure and may be placed in the 0- to 5-year category.[67-70]

The dentist should evaluate teeth especially next to multiple edentulous sites. A natural tooth distant from the future implant restoration site is less likely to affect the implant reconstruction and alter the treatment sequences in this site. However, failure of a natural tooth adjacent to an implant site may cause failure of the adjacent implant and almost always (whether failure occurs or not) causes the restoration to be delayed and compromised. Therefore if the practitioner is not sure whether the tooth is in the 0- to 5-year or 5- to 10-year category, the tooth more often should be considered to have the poorer prognosis.

EXISTING PROSTHESES

When present, existing prostheses are evaluated for proper design and function. A removable partial soft tissue–supported restoration opposing the proposed implant-supported prosthesis is of particular interest. The occlusal forces vary widely as the underlying bone remodels. The patient may not even wear the opposing removable partial denture in the future, which will dramatically modify the occlusal conditions. Therefore continued maintenance and follow-up evaluations are indicated, including relines and occlusal evaluation.

The patient should be asked whether esthetic desires are met with the current restorations. It is not unusual that the prosthesis is completely acceptable, yet the patient wishes a different shade or contour for the teeth. If unacceptable to the patient, the reasons for dissatisfaction are noted. In addition, the existing restorations are evaluated throughout the mouth for clinical harmony. It is better to leave a poor esthetic restoration that is in occlusal harmony than to provide one that is esthetic but improper in position, because the latter may influence all future restorations. Pontic regions of existing prostheses may often be improved with the addition of connective tissue grafts.

An acceptable preexisting maxillary removable prosthesis, which will be replaced with a fixed implant prosthesis, may be used as a template for implant reconstruction when fabricating an implant-supported fixed or removable implant prosthesis. The thickness of the labial flange of the existing denture is evaluated and is often removed to evaluate the difference in lip position and support. If implants may be correctly inserted, yet additional lip support is needed once the labial flange is eliminated, an HA, connective tissue, or acellular dermal onlay graft is usually indicated. This graft is not intended for implant support or placement, but to enhance the support of the labial alveolar mucosa to improve maxillary lip support.

ARCH FORM

The edentulous arch form in the horizontal plane is described as ovoid, tapering, or square. In the edentulous patient, the ovoid arch form is the most common, followed by the square, then the tapered form. The square arch form may result from the initial formation of the basal skeletal bone. However, the presence of a square arch form is more common in maxillary implant patients as the result of labial bone resorption of the premaxilla region when anterior teeth are lost earlier than the canine. The tapering arch form is often found in skeletal Class II patients as a result of parafunctional habits during growth and development. It is not uncommon to find different arch forms in the upper and the lower arches.

Two different arch forms are to be considered for implant prostheses. The first arch form is of the residual edentulous bone and determines the A-P distance for implant support. The second arch form is of the replacement teeth position. The dentate and edentulous arch form as not necessarily related, and the worst

WWW.HIGHDENT.IR

situation in the maxilla corresponds to a square residual arch form that supports a tapered dental restoration. The cantilever off the available bone is greatest in this combination (Figure 12-22).

The most ideal biomechanical arch form depends on the restorative situation. The tapering residual ridge arch form is favorable for anterior implants supporting posterior cantilevers. The square dental arch form is preferred when canine and posterior implants are used to support anterior teeth in either arch. The ovoid arch form has qualities of both tapered and square arches.

The arch form is a critical element when anterior implants are splinted together and support a posterior cantilever restoration. For these conditions, a square arch form provides a poorer prognosis than a tapered arch form. The A-P distance or A-P spread is the distance from the center of the most anterior implant to a line joining the distal aspect of the two most distal implants.[55,71] It provides an indication as to the amount of cantilever that can be reasonably planned. When five anterior implants in the mandible are used for prosthesis support, the cantilevered posterior section of the restoration should not exceed 2.5 times the A-P spread, when all patient force and stress factors are low. The actual length of the cantilever depends not only on implant position, but also on other stress factors including parafunction, crown height, implant width, and number.

In other words, the predominant factors to determine the cantilever length are related to stress, not the A-P distance.[45,46] For example, the distance between two implants supporting a cantilever (C) form a Class I lever. For implants 10 mm apart and a 10-mm posterior cantilever, the following forces are applied: a 25-lb force on cantilever C results in a 25-lb force on the most anterior implant from the cantilever (A) and 50 lb for the nearest implant to the cantilever (B), which acts as a fulcrum. An interimplant distance of 5 mm with the same 10-mm cantilever and a 25-lb force applied on C results in a 50-lb force on A and a 75-lb force on B. The diminution in the distance between implants significantly increases the forces to both implants. But, in the first example, if a patient with parafunction bites with a 250-lb force on C, the force on implant A is 250 lb and the force on implant B is 500 lb. In other words, parafunction is much more meaningful in terms of force than the interimplant distance when designing a cantilever. Therefore A-P distance is only one stress factor to evaluate for cantilever length. Parafunction, crown height, masticatory dynamics, arch position, opposing arch, direction of force, bone density, implant number, implant width, implant design, and A-P distance are all factors to be considered. When the force factors are low and the area factors (implant number, width, and design) are high, the cantilever length may be as much as 2.5 times the A-P distance.

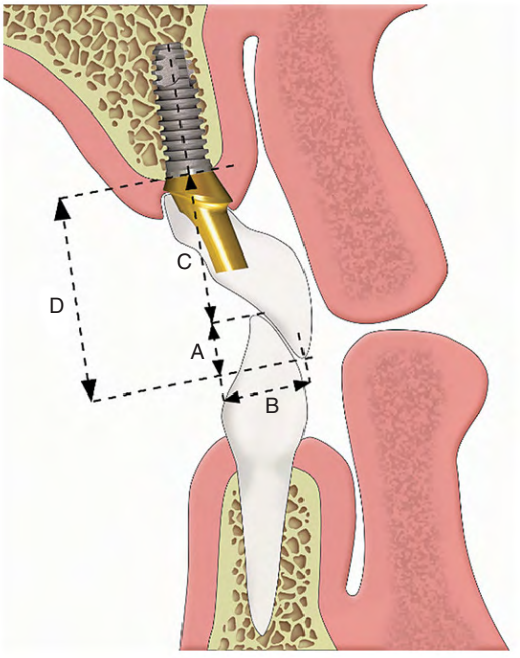


Figure 12-22 The dental arch form may be different than the arch form of the residual arch. A tapered dentate arch form on a square residual bone form is the worst combination, because the anterior teeth are cantilevered from the implant abutments.

As mentioned previously, anterior endosteal implants often may not be inserted in their ideal location in the maxilla as a result of labial plate resorption and inadequate bone width at the implant site. This not only requires implant placement more palatally compared with the original natural teeth, it may also negate the lateral and central positions and require the use of the canine regions in more advanced atrophic arches. The resulting restoration is a fixed, anteriorly cantilevered prosthesis to restore the original arch form. Under these conditions, greater stresses are placed on the dentate tapered arch forms compared with dentate square arch forms, all other factors being identical. The maxillary anterior cantilever to replace teeth in a dentate-tapered arch form requires the support of additional implants of greater width and number to counteract the increase in lateral load and moment force. For example, not only are the canine implants necessary, two more anterior additional implants are suggested, even if bone grafting is required before their placement. In addition, additional posterior implants in the first to second molar region splinted to the most anterior implants are highly suggested. Therefore if a maxillary arch form requires this treatment approach, at least eight implants (four on each side) and an increased A-P distance from molar implants splinted to incisor implants is suggested. In the maxilla, the recommended anterior cantilever dimension is less than for the posterior cantilever in the mandible, because of poor bone density and forces directed outside the arch during excursions (see Chapter 17).

WWW.HIGHDENT.IR

Box 12-4 Teeth Adjacent to Implant Site

1. Abutment options
2. Extract or maintain
3. Transitional abutment
4. Adjacent bone anatomy
5. Cantilevers
6. Implants connected to teeth
7. Natural abutment mobility
8. Splinting natural abutments
9. Natural and implant pier abutments

Box 12-5 Natural Abutment Evaluation

1. Abutment size
2. Crown/root (implant) ratio
3. Endodontic status
4. Root configuration
5. Tooth position (in the arch)
6. Parallelism
7. Root surface area
8. Caries: restorability
9. Periodontal status

NATURAL TEETH ADJACENT TO IMPLANT SITE

A common prosthetic axiom is to provide the partially edentulous patient with a fixed prosthesis whenever possible. Implant dentistry often may provide the additional abutments necessary to fulfill this goal, regardless of the number of teeth missing. The ability to add abutments in specific locations, rather than being limited to a particular remaining natural abutment that may not always be in optimum health, enables the dentist to expand this prosthetic axiom to most patients. The dentist may use implants as independent support for the restoration or rarely, along with natural teeth in the same prosthesis. In either situation, the treatment plan is strongly influenced by the dental evaluation of the remaining natural abutments adjacent to the edentulous site. Natural teeth may require additional therapy before the final prosthesis can be completed. It is best to communicate with the patient regarding all required treatment involved in the rehabilitation process before the surgical placement of the implants. Otherwise, treatment outcome sequences and cost may conflict with the originally projected result and lead to dissatisfaction, the need to modify the original treatment plan, or a poorer prognosis.

Whether considered for abutment support or not, teeth adjacent to a partially edentulous site are evaluated thoroughly and from a different perspective than the rest of the dentition. Often the adjacent tooth exhibits bone loss next to the edentulous site and presents a less than optimal quality of health. In addition, the available bone characteristics immediately adjacent to the tooth are highly influenced by its presence. Often this is a determining factor in the choice between an independent implant prosthesis, a traditional fixed partial denture, or a removable restoration. When multiple teeth are missing, the treatment becomes even more complex with additional restorative options, such as whether implants and natural teeth may serve as abutments in the same prosthesis.

The dental criteria of the adjacent tooth to an edentulous space addressed in this section are outlined in Box 12-4, as well as important parameters to evaluate in considering implants and teeth in the same restoration. Additional considerations to help assess the restorability of teeth adjacent to potential implant sites appear in Box 12-5.

Abutment Options

Several options are available for the adequate restoration of an edentulous segment. Under ideal conditions, placing implant abutments in sufficient number to fabricate a completely implant-supported prosthesis has several advantages. The most common cause of failure of tooth-supported fixed prostheses is caries of the abutment teeth.[72-74] Unrestored natural teeth do not decay as often as restored teeth, and implant abutments do not decay. The second most common cause of fixed prosthesis failure is endodontic failure or complications of a natural tooth abutment. Implant abutments do not need endodontic therapy. As a result, the 10-year survival rates indicate a greater than 25% improved survival rate for implant prosthesis compared with fixed partial dentures supported by natural teeth.[75,76] Natural teeth abutments compared with unrestored natural teeth are more difficult to clean, collect and retain more plaque, are often more temperature or contact sensitive, and are more subject to future prosthetic periodontal or endodontic treatment. Caries, endodontic problems, or both may cause not only a loss of the fixed prosthesis more than 25% of the time within 10 years, but also almost as often lead to the failure and extraction of at least one of the natural tooth abutments. As a result, an independent implant restoration is the treatment of choice for almost every multiple-tooth edentulous site in a partially edentulous patient.[77]

Natural teeth respond to occlusal forces differently than implants. A light force produces most of the recorded movement of a tooth, whereas the amplitude of implant movement is related directly to the force applied.[78-80] In arches with implant and natural abutments, it is easier to adjust two independent prostheses. When planning an independent implant prosthesis, instead of using a natural tooth as one of the terminal abutments, the dentist usually requires the addition of at least one more implant. An increase in implant

WWW.HIGHDENT.IR

abutment number enhances the implant-bone interface and therefore reduces the stress to the support system and improves the ability of the fixed restoration to withstand additional forces, when necessary. In addition, because of the additional retentive units, uncemented or unretained restorations occur with less frequency. Unretained restorations are the third most common complication reported in fixed prosthodontics.[73,74] Abutment screw loosening is a complication reported for implant prostheses, especially during the first year.[43,81-87] The increase in implant number also decreases the amount of forces on the abutment screws, and thus the risk of abutment screw loosening; as a result, many reasons justify the use of a sufficient number of implants for an independent prosthesis. So many advantages exist for an independent implant-supported fixed prosthesis with multiple units that such a treatment is always the first choice when possible. Unfortunately, completely implant-supported fixed prostheses in partially edentulous patients are not always feasible and carry a higher surgical risk. Thus the natural tooth occasionally may be considered a potential abutment. However, the dentist should consider splinting of implants and natural teeth within the same prosthesis only when the surface area of the implant support does not permit replacement of the total number of missing teeth and additional implant placement is not a possibility.

Transitional Natural Abutments

On occasion, because of the lengthy aspect of implant treatment, when bone regeneration procedures are indicated before implant placement, initially maintaining strategic teeth (even with a poor prognosis) as interim restoration abutments may be desirable. These teeth are often terminal abutments that support a fixed temporary restoration, protect edentulous implant or graft areas from mastication trauma, and avoid the use of a removable soft tissue–borne partial interim prosthesis. After these teeth are extracted after implant healing, their position is often an ideal implant site for the final restoration. The implant then is placed in a second surgical phase. This approach is beneficial to avoid soft tissue–supported restorations on bone augmentation sites but may extend treatment by 6 months.

The transitional abutment scenario is most common in a full-arch rehabilitation patient who has a full-arch fixed restoration on periodontally involved teeth. The prognosis of these abutments may be poor, mandating their extraction (less than 5-year survival category). However, if all these dental elements are extracted, the patient usually is given a full immediate denture as a temporary prosthesis while grafting and implant insertion phases are performed. The psychologic and physiologic changes associated with a denture, even if a temporary solution, may have dramatic consequences for the patient, who may benefit greatly from a stepped approach in which a few poor, short-term, asymptomatic dental elements are maintained while all others are extracted for the sole purpose of providing the patient with a fixed temporary restoration.[88]

The careful selection of the transitional abutments must not hinder the implant treatment. However, an extended treatment time with additional implant placement surgery may be required. For example, four dental elements dispersed throughout the arch may be kept for the temporary restoration. Meanwhile, other sites are extracted, grafted, and implanted. When these implants are healed and ready to be restored, the temporary natural abutments may be extracted and additional implants placed. The healed implants now may support the transitional prosthesis. The new implants, on occasion when the bone density and biomechanical factors permit, may be immediately loaded.

The advantages of the transitional abutment procedures are that a fixed prosthesis maintains the patient throughout treatment, protecting the implant surgical site during the submerged healing phase. Disadvantages include additional cost, increased time, risk of implant site contamination if any problem or flare-up of natural abutments occurs, and increased risk for the initial implants because the foundation is not completely sufficient for support until the additional implants are healed. The dentist should weigh the advantages and risks of such a treatment carefully before proposing it to the patient.

A fixed interim prosthesis also may be supported by three to six additional implants placed in function immediately at insertion to permit the fabrication of a temporary fixed prosthesis while all other implants are submerged. The dentist evaluates these additional implants at the time of final prosthesis fabrication and may or may not include them, depending on their status at that time. Transitional mini-implants also have been developed to that effect.[89] However, caution is needed in using additional implants of normal or minimized dimensions, because the volume of bone used for their placement may be of strategic value during treatment and risks being destroyed by fibrous tissue formation or bone resorption when immediately loaded, which may affect a final implant prognosis. Also, if a final implant fails, the alternate site already has been used and may be unavailable. Further research is needed to establish the exact range of application of these alternatives. Such treatment options are indicated only on a case-by-case basis.

Adjacent Bone Anatomy

The edentulous bony structure adjacent to a natural tooth varies in height, width, length, and angulation and is a reflection of the history of the former tooth.

WWW.HIGHDENT.IR

If the ridge topography is not ideal for endosteal implant placement in the site immediately adjacent to the natural abutment, the dentist should consider a bone graft or a pontic. An osteoplasty needed to obtain adequate bone width in the area adjacent to a natural tooth may compromise the adjacent natural root support, increase the crown height of the final restoration, and affect the esthetic outcome. Therefore osteoplasty to gain additional width is rarely indicated in this situation.

If an ideal prosthodontic abutment position is adjacent to a natural tooth and inadequate bone width is available, augmentation of the edentulous site before implant insertion may improve the bone anatomy without compromising the natural abutment. However, inadequate bone height adjacent to a tooth offers a poorer prognosis for augmentation than in other situations. In general, to grow bone in height is more difficult than to grow it in width. However, when the inadequate bone height of the edentulous site includes the region adjacent to a natural root, the ability to grow additional bone height becomes even more unpredictable and usually unsuccessful. Bone height augmentation is not predictable on a natural tooth root with a horizontal defect. If the natural tooth root has lost bone adjacent to the site, the bone augmentation in height usually will not occur above the position of bone on the root. An alternative for inadequate bone height next to a natural tooth is orthodontic extrusion along with the bone graft. The orthodontic movement will increase bone height next to the tooth and improve the bone graft prognosis. However, the tooth usually requires endodontics and restoration after the orthodontic process.

An implant apically positioned more than 3 mm to 4 mm below the cement-enamel junction (CEJ) and interproximal bone level of the natural tooth root presents potential soft tissue contour problems (Figure 12-23). The soft tissue between the tooth and implant creates a more shallow slope, unlike the steep drop of the level of the bony crest between the elements. Under these conditions, a soft tissue pocket greater than 6 mm may result in the implant crown adjacent to the natural tooth. Therefore when a bone graft for height in a multitooth edentulous site is required to place an implant adequately adjacent to a natural root, the dentist should consider a pontic to replace the missing element next to the natural tooth. The pontic may be supported by a cantilever from implants or teeth or using dual support from teeth and implants.

Figure 12-23 The implant in the mandibular right first premolar site is positioned more than 6 mm below the canine cement-enamel junction. Although bone is present on the distal of the canine, the soft tissue slope around the implant creates a soft tissue pocket greater than 5 mm and increases the risk of peri-implant complications.

In case of inadequate bone volume adjacent to a tooth, the dentist considers treatment options in this order: (1) graft the site if inadequate in width to permit Division A or B implant placement, (2) cantilever a pontic from two or more natural teeth or two or more Division A implants, and (3) fabricate a fixed prosthesis with one pontic connecting an implant with one or two teeth, depending on the adjacent tooth status (Figure 12-24).

Cantilevers

Cantilevers in fixed prostheses result in moment loads or torque on the abutments.[90-94] They are used more frequently for implant-supported prostheses than natural teeth abutments, and several widely diverging guidelines have been recommended for their use, ranging from no extension at all to several teeth.[90-96] The force on the cantilever may be compared with a class I lever. The distance between the most anterior and most distal abutments is divided into the length of the cantilever to determine the mechanical advantage to the farthest abutment from the cantilever. For example, if the implants are 10 mm apart and a distal cantilever of 15 mm is present, the mechanical advantage is 1.5 times. A 25-lb compressive load (chewing force) is magnified to a tensile load of 37.5 lb on the most anterior abutment. The abutment closest to the cantilever acts as a fulcrum and receives the sum of the two loads, or a compressive load of 62.5 lb.

The most common complication for a cantilevered restoration is uncementation of the abutment farthest from the cantilever. This occurs because cement is about 20 times weaker in tension compared with compression forces. For example, the compressive strength of zinc phosphate cement is 12,000 psi, but its tensile strength before fracture is only 500 psi. Takayama has suggested that the cantilever should not extend beyond the distance between the implants to keep the mechanical advantage less than one times this distance.[97] The most common distance between two implant centers is 7 to 8 mm so that the outer dimensions of the implants may be 3 mm apart and the crowns on the implants are similar in size to a premolar. Thus the size of the cantilever should not be greater than a premolar of similar size when two implants support the prosthesis.

Ideally, a cantilever should extend mesially, rather than distally, to reduce the amount of occlusal force

WWW.HIGHDENT.IR

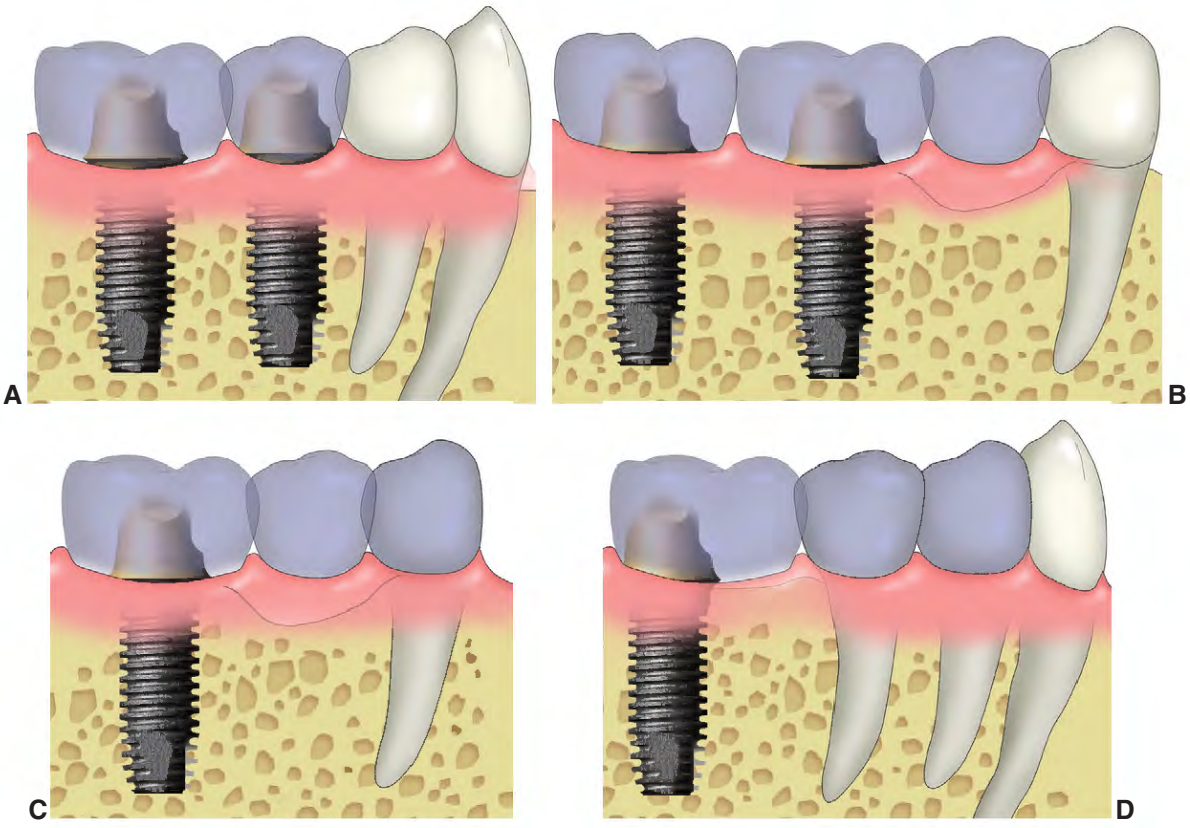


Figure 12-24 **A,** When the inadequate bone adjacent to a tooth can be grafted for implant placement and an independent prosthesis, this is the treatment of choice. **B,** When the inadequate bone adjacent to a tooth cannot be grafted, one option is to cantilever the missing tooth from the anterior teeth or from posterior implants. The posterior implants permit the replacement of more than one tooth but require at least two implants. **C,** When the inadequate bone adjacent to a tooth cannot be grafted, another option is to insert an implant more distal and make a three-unit fixed partial denture by connecting the implant to the nonmobile tooth. **D,** When the inadequate bone adjacent to a tooth cannot be grafted and the tooth is slightly mobile, one option is to insert an implant more distal and make a four-unit fixed partial denture by connecting the implant to two anterior teeth (when the most anterior tooth is nonmobile).

on the lever.[98] The most important factor in determining the length of the cantilever is the amount of force the patient places on the cantilever. In other words, the amount of force generated against the cantilever is more critical than the other factors, including the cantilever length and mechanical advantage. In addition, an angled force is more detrimental than a force in the long axis of the abutments.

The crown height also influences the amount of the force on the cement and bone interface. As such, the cantilever magnifies any other force factor presented and therefore should be used with caution. When cantilevers are used in the final restoration, the occlusion on the cantilevered pontics should be reduced, with no contact on the pontic during mandibular excursions.

Cantilevers on two implants should not be used when force factors are moderate to severe or when other force factors are present (Figure 12-25). Instead, additional implants or grafting and implants positioned without cantilevers typically reduce complications.

When the cantilever forces are too great, the dentist also should consider joining the implants to adjacent teeth to eliminate the cantilever effect.

Implants Connected to Teeth

Although rare, the most common scenario for which a root form implant may be joined to a natural tooth as a terminal abutment is in the posterior regions of the mouth. For example, if a patient is missing the first and second molars in a quadrant (with no third molar present), the segment requires at least two implants of proper size and design to independently restore these two teeth. If adequate bone exists in the second molar and distal half of the first molar but inadequate bone exists in the mesial half of the first molar, a premolar-size pontic is required. The pontic may be cantilevered from the anterior natural teeth or the posterior implants. Either of these options may result in complications because of tensile forces on the cement seal of the abutment farthest from the pontic.

An alternative may be to join the implant to a natural tooth, if all other factors are favorable. This plan is more likely in the presence of a division C–h ridge in the pontic region, when inadequate bone height adjacent to the natural tooth decreases the prognosis of a vertical bone graft. Another scenario in favor of this treatment plan is when the posterior implants are of a

WWW.HIGHDENT.IR

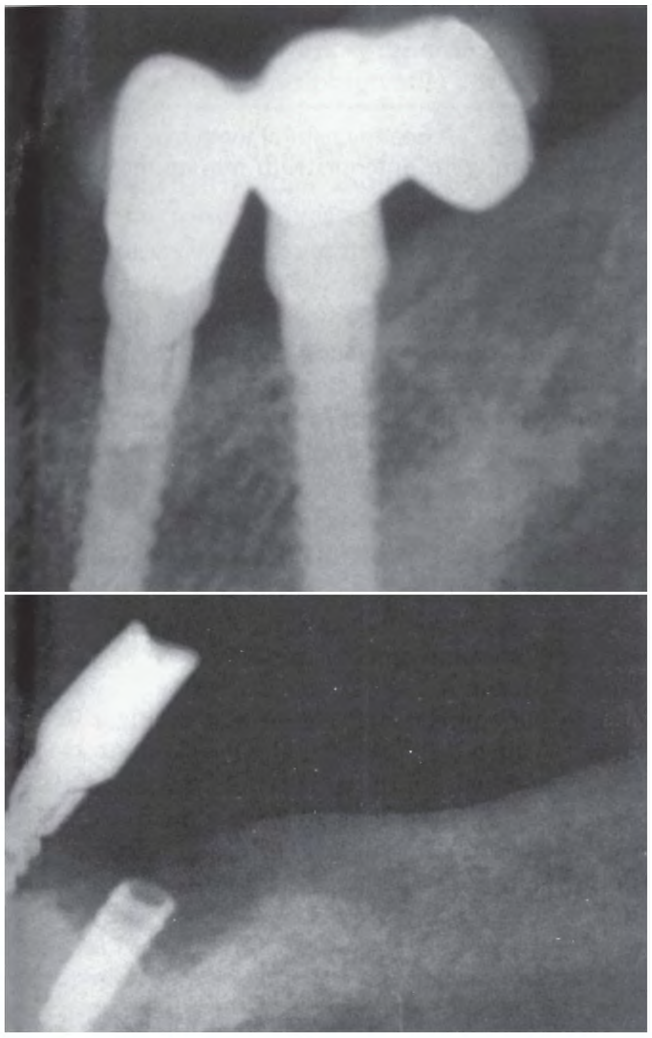


Figure 12-25 **A,** A three-unit fixed restoration with one posterior cantilever. The implants are less than 3.75 mm in diameter. The opposing arch was natural teeth. **B,** The distal implant fractured. Before the fracture, crestal bone loss was evident on both implants. This suggests too great a force was being applied to the support system. The bone loss increases the crown height and directly increases the force to the implants. The smaller diameter implants increase the stress to the bone and have less resistance to fatigue fracture.

more narrow diameter than usual. When two Division B root forms are used in the posterior mandible to replace molars, there should be no cantilever to magnify the force on the implants. Posterior pontics should not be cantilevered from even two splinted Division B root form implants. An additional root form implant or natural tooth is required as an abutment for the fixed prosthesis. When an additional implant insertion is not an option, the posterior implants may be joined by a rigid connector (i.e., a solder joint) to natural teeth within the prosthesis, provided all dental factors are favorable.

The connection of natural teeth and osteointegrated implants within a single rigid prosthesis has generated concern and publications, with studies and guidelines for both extremes (Figure 12-26).[99-106] In other words, some articles report problems, whereas others state that

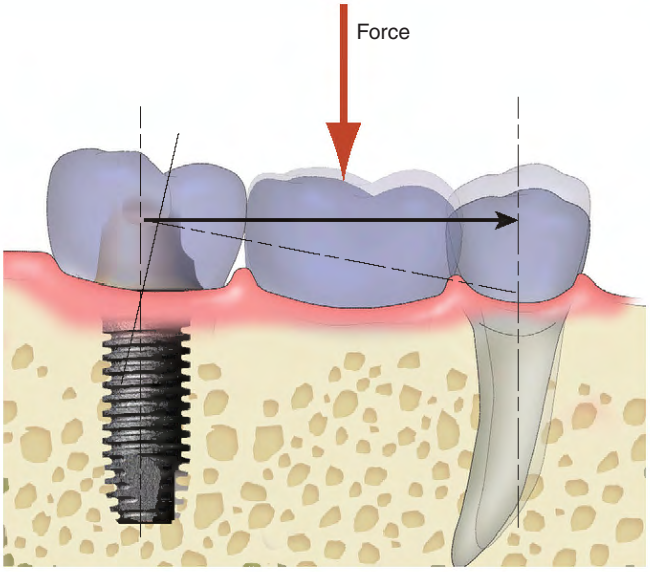


Figure 12-26 Splinting a rigid implant to a natural tooth has caused concerns relative to the biomechanical differential in movement between the implant and tooth. Because the tooth moves more than the implant, the implant may receive a moment force created by the "cantilever" of the prosthesis.

no problem exists. To be more specific to a particular situation, more information is required to design a successful treatment plan. Two designs are available for the connection of implants and teeth within the same prosthesis: a conventional fixed partial denture or a fixed partial denture with a nonrigid connector. To address this issue, the mobility of the natural abutment must be assessed.

Mobility

The mobility of potential natural abutments greatly influences the decision to join implants and teeth more than any other factor. In the implant-tooth rigid fixed prosthesis, five components may contribute movement to the system: the implant, the bone, the tooth, the prosthesis, and implant/prosthetic components.

Vertical Movement

The tooth exhibits normal physiologic movements in vertical, horizontal, and rotational directions. The amount of movement of a natural tooth is related to its surface area and root design. Therefore the number and length of the roots; their diameter, shape, and position; and the health of the periodontal ligament primarily influence a tooth mobility.[107] A healthy tooth exhibits no clinical mobility in a vertical direction. Actual initial vertical tooth movement is about 28 µm and is the same for anterior and posterior teeth.[80] The immediate rebound of the tooth is about 7 µm and requires almost 4 hours for full recovery, so additional forces applied within 4 hours depress the tooth less than the original force.[78] The vertical movement of a rigid implant has been measured as 2 to 3 µm under a 10-lb force and is due mostly to the viscoelastic properties of the underlying bone (Figure 12-27).[80]

WWW.HIGHDENT.IR

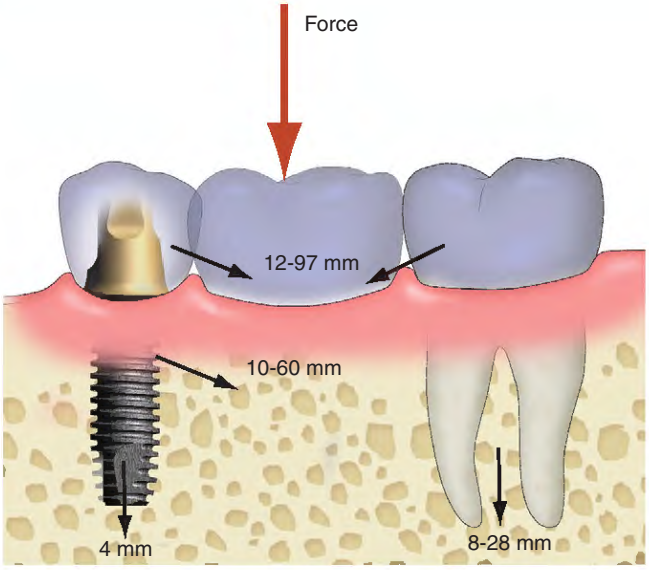


Figure 12-27 A three- or four-unit precious metal prosthesis with an implant and a posterior tooth rigidly splinted has some inherent movement. The implant moves apically 0 to 5 µm, and the tooth moves apically 8 to 28 µm but can rotate up to 75 µm toward the implant because of a moment force. The metal in the prosthesis can flex from 12 to 97 µm, depending on the length of the span and the width of the connecting joints. The abutment-to-implant component movement may be up to 60 µm because of abutment prosthetic screw flexure. As a result, a vertical load on the prosthesis creates little biomechanical risk when joined to a nonmobile tooth, because of the design.

Prosthesis Movement

The fixed prosthesis that connects a tooth and implant also illustrates movement. Under a 25-lb vertical force, a prosthesis with a 2-mm connector fabricated in noble metal results in a 12-µm movement for one pontic and 97-µm movement for a two-pontic span.[108-110] Therefore the fixed partial denture movement helps compensate for some difference in vertical mobility of a healthy tooth and implant. Rangert et al.[106] reported an in vitro study of a fixed prosthesis supported by one implant and one natural tooth and showed that the abutment/gold cylinder screw joint of the system acts as a flexible element. The inherent flexibility matched the vertical mobility of the natural tooth. Therefore the minimal movement of the tooth and the fact that implant, prosthesis, and abutment components have some mobility indicates the risk is small in the vertical direction with the biomechanical difference of implant and tooth in the same prosthesis when one or two pontics separate these units.

Horizontal Movement

Horizontal tooth mobility is greater than vertical movement. A very light force (500 g) moves the tooth horizontally 56 to 108 µm (Figure 12-28). The initial horizontal mobility of a healthy, nonmobile posterior tooth is less than that of an anterior tooth and ranges from 56 to 75 µm, which is two to nine times the vertical movement of the tooth. Initial horizontal mobility is even greater in anterior teeth and ranges from 90 to 108 µm in healthy teeth.[79]

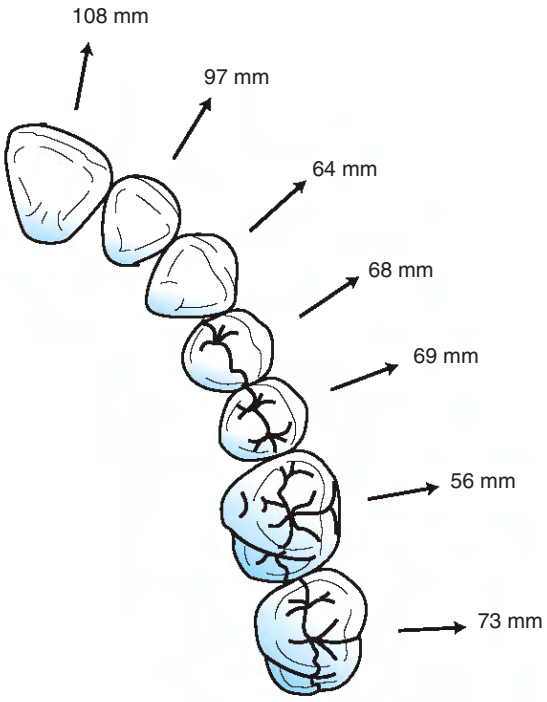


Figure 12-28 A healthy natural tooth may move laterally from 56 to 108 µm, with anterior teeth moving more than posterior teeth.

Muhlemann found that tooth movement may be divided into initial mobility and secondary movement.[78] The initial mobility is observed with a light force, occurs immediately, and is a consequence of the periodontal ligament. If an additional force is applied to the tooth, a secondary movement is observed, which is related directly to the amount of force. The secondary tooth movement is related to the viscoelasticity of the bone and measures up to 40 µm under considerably greater force.

Implant Movement

The implant-bone interface also exhibits lateral movement. Sekine et al.[80] evaluated the movement of endosteal implants with rigid fixation and found a range of 12 to 66 µm of movement in the labiolingual direction. Komiyama[111] measured 40 to 115 µm of implant movement in the mesiodistal direction under a force of 2000 g (about 4.5 psi) and a labiolingual range of 11 to 66 µm. The greater implant movement in the mesiodistal dimension corresponds to the lack of cortical bone between the implants in this direction compared with the thicker lateral cortical plates present in the labiolingual dimension. Therefore the mobility of implants varies in direct proportion to the load applied and the bone density and reflects the elastic deformation of bone tissue.

An interesting note in implant mobility is that no significant difference was related to implant length. This finding further confirms that implant length is not the primary factor for implant support, even in the presence of lateral loads. Bone density affects this condition more than implant length. These mobility

WWW.HIGHDENT.IR

Figure 12-29 An implant joined to an anterior tooth is at greater risk of biomechanical overload. The lateral force on the tooth causes greater movement than a vertical force. Anterior teeth receive more lateral loads than posterior teeth. The lateral loads are transferred to the implant. Lateral loads on the implant increase the amount of crestal bone stresses.

characteristics corroborate the findings of Fenton et al.,[112] who applied a 500-g load for 4 seconds to maxillary anterior teeth and osseointegrated implants. The implants were displaced a mean 10 µm with a rapid elastic return (less than 1 millisecond), whereas the teeth showed a mean displacement of 57 µm with a prolonged viscoelastic return. Therefore when all factors are considered, an implant moves vertically and horizontally, the abutments and prosthesis flex, and the tooth has apical and lateral movements.

Guidelines for Joining Implants to Teeth

A vertical movement/force placed on a posterior implant joined to a healthy posterior tooth causes mesial tension on the implant. The implant can move vertically 3 µm and mesially 40 to 115 µm, and a noble metal-fixed prosthesis with one pontic allows mesiodistal movement of 6 µm. Therefore a natural tooth with no clinical mobility could be connected rigidly to an osseointegrated implant because the implant, bone, and prosthesis compensate for the slight tooth movement. There is extensive documentation that implants can be connected rigidly to stable teeth.[84,85,106,113-116] However, the occlusion should be modified to allow the initial occlusal contacts on the natural tooth so that the implant does not bear the major portion of the initial load.[117]

The mobility of healthy anterior incisor teeth often is recorded as 1 with a range of movement from 90 to 108 µm. Therefore visual clinical evaluation by the human eye can detect movement greater than 90 µm. When the mobility of a natural tooth can be observed, mobility is greater than 90 µm and too great to be compensated by the implant, bone, and prosthesis movement. When the vertical posterior tooth movement, vertical implant movement, mesiodistal implant movement, and prosthesis movement are compared with the same conditions of an anterior tooth with lateral loads, the biomechanical risk factors do not correlate. Therefore one primary condition for joining an implant to natural teeth is the lack of observable clinical movement of the natural abutment.

Another requisite to join an implant to a natural tooth is that no lateral force should be designed on the prosthesis. Lateral forces increase the amount of tooth movement and decrease the amount of implant movement (faciolingual versus mesiodistal). Horizontal forces placed on an implant also magnify the amount of stress at the crestal bone region.[117] Therefore implants rarely should be connected to anterior teeth because (1) anterior teeth often exhibit greater clinical mobility than the implant can tolerate and (2) the lateral forces applied to the restoration during mandibular excursions are transmitted to the natural tooth and implant abutments (Figure 12-29).

When the natural abutment exhibits clinical horizontal movement or conditions promote horizontal forces against the abutment tooth, two options can be selected for the final prosthesis. The first, and the option of choice, is to place additional implants and to avoid the inclusion of natural abutments in the final prosthesis. The other option is to improve stress distribution by splinting additional natural abutments until no clinical mobility is observed.

Guidelines for Splinting Dental Units

Splinting natural teeth does not decrease the mobility of a tooth significantly after the prosthesis is removed; however, the overall prosthesis movement is decreased, especially when the splinted units form an arch. If posterior contacts cannot be eliminated in lateral excursions as a result of skeletal relationships or when opposing a removable prosthesis, splinting often is safer to reduce the risk of long-term complications. In addition, splinting natural abutments also decreases the amount of load to each abutment (when a 150-psi load is distributed to all splinted abutments, the resultant force on each abutment is decreased).[118,119]

The number of teeth to splint together is the number required to eliminate prosthesis movement. The initial dental evaluation may include acid etching and bonding potential mobile natural abutments to each other to determine how many teeth must be joined to reduce the prosthesis clinical mobility to zero. The dentist applies the following prosthetic guidelines:

1. The last tooth connected in the splint should not be mobile. In other words, to decrease mobility, at least the last tooth in the splint (and sometimes more) should be rigid.

WWW.HIGHDENT.IR

2. The terminal abutments in the splint should not have poor retention form.

The natural abutment connected to a rigidly fixated implant should not exhibit clinical mobility or poor retentive form. These same two criteria should be considered for the natural tooth used as a secondary abutment when splinting teeth in a fixed partial denture. A classic axiom for splinting teeth in prosthodontics reads, "It is unadvisable to employ the last tooth as a splinted abutment if it lacks a degree of firmness comparable to its healthy neighbor, because the strain on the firm abutment could be destructive."[120] Implant prostheses may use additional secondary natural abutments to decrease the movement of the prosthesis so that rigid fixation of the implant will not be compromised. However, if the last abutment is mobile, it does not serve the intended purpose. A general guideline is not to end a fixed prosthesis on the weakest splinted abutment. The weak tooth does not offer additional support and further burdens the healthier abutments.[121,122]

In addition, if cement failure occurs or the restoration needs retrieval, the partially retained prosthesis is more difficult to retrieve from the mobile abutment, resulting in more frequent coronal fracture and other complications. The natural teeth exhibit some faciolingual movement, which varies from 56 to 108 µm in health. The discussion here is to reduce tooth movement when it is visible so that the mobile teeth may be connected to the implants. Although the teeth move in a faciolingual direction, different regions of the arch have different directions of movement relative to each other. In other words, the faciolingual direction of the anterior teeth corresponds to the mesiodistal direction of the posterior teeth; therefore, if these dental units are splinted to each other, the splint may become nonmobile.

A dental arch may be described as a five-sided structure. The posterior teeth move in a similar direction to each other, the canines move in a different direction, the anterior teeth move in a third direction, the contralateral canine moves in yet a different direction in comparison, and the other posterior component of the arch moves in a similar direction as the first. The more dental sections are connected, the more rigid the structure. As a general rule, three or more sections rigidly connected create an overall nonmobile dental structure. Even mild to moderate mobile dental units may become a nonmobile unit. This approach to joining implants to mobile teeth usually is limited to conditions when the multiple sections of the dental arch already require restoration. Rarely would one consider crowning eight or more teeth solely to splint to the implant component. Instead, the use of a natural tooth pier abutment may be indicated.

Nonrigid Connectors

Although nonrigid connectors have been advocated in the literature,[67] a nonrigid connector in a unilateral prosthesis rarely is indicated in fixed prostheses[122] and has proved detrimental in implant- and teeth-supported restorations.[103,123] Nonrigid connection reportedly does not improve the stress distribution between the different abutments[124-126] and also has been reported to have caused migration of the natural teeth.[99,103-105,127] If the nonrigid connector exhibits any clinically observed mobility, it moves more than the implant. As such, the implant-supported part of the restoration is cantilevered to the attachment. In addition, the nonrigid, or mobile, attachment adds cost, creates overcontoured abutments, impairs daily hygiene, and does not decrease the clinical tooth movement.

Implants should not be joined to mobile teeth with rigid attachments, which basically add a cantilever on the implant (the tooth acting as a living pontic). If the natural teeth are too mobile in relation to the implant in the same prosthesis, several complications may occur and be detrimental to tooth and implant.[128,129] If the prosthesis is cemented, movement may break the cement-implant abutment seal. Cement does not adhere as well to titanium as to dentin. In addition, the mobile tooth will move rather than break the cement seal. In a screw-in prosthesis, the coping screw often will loosen or break in the implant.

After the prosthesis is loose from the implant, greater stress is applied to the natural mobile tooth. However, until the implant retainer attachment is lost, the greatest stress is transmitted through the prosthesis to the implant and bone section. Crestal bone loss or implant breakage from fatigue are then likely complications.

Regardless, when implants are joined to teeth that act as a terminal abutment, a definitive cement should be used for the natural tooth. Reports of intrusion of the natural tooth connected to an implant include the use of temporary cement to lute a coping to the natural abutment, leaving the final restoration uncemented on the coping,[103-105,127] or the use of a nonrigid connector.

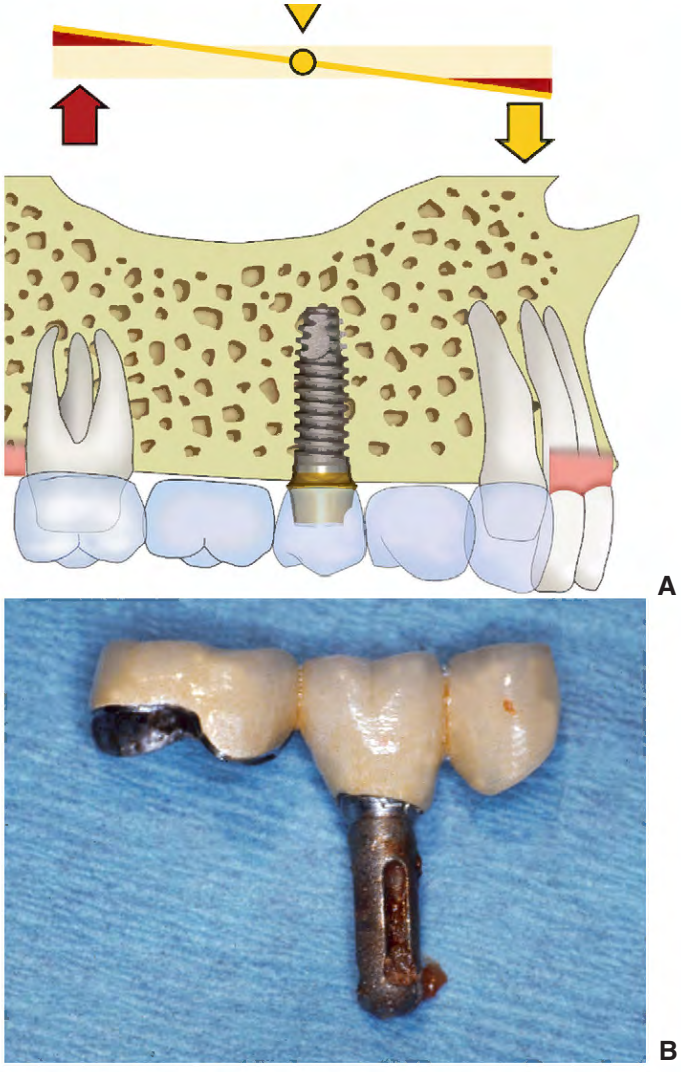
A possible explanation for tooth intrusion could be that the tooth is pushed vertically 28 µm, but wants to rebound only 8 µm. The fixed prosthesis rebounds immediately and pulls on the tooth. The cement seal eventually breaks, causing a space to develop, which is first occupied by air. The prosthesis then acts as an orthodontic appliance and continually pushes the tooth in a vertical direction. Eventually, the space is occupied by saliva, and hydraulics continue the downward force during mastication. The tooth eventually submerges or intrudes from the prosthesis (Figure 12-30).

Pier (Intermediary) Abutments

A pier abutment is one between two other abutments, sometimes referred to as intermediate abutment. The intermediate abutment may be an implant or a natural tooth, and each type plays a different role in the overall treatment. When an implant serves as a pier abutment between two natural teeth, the difference in movement between implant and tooth may increase the

WWW.HIGHDENT.IR

Figure 12-30 An implant should be joined to a natural tooth with a rigid connection, and the natural tooth crown should be cemented with definitive cement. If the cement seal fails, the tooth may intrude from the prosthesis.



A

B

Figure 12-31 **A,** When an implant acts as a pier abutment, the biomechanical risk of uncemented restorations is increased, especially under lateral loads. The more rigid implant may act as the fulcrum of a class 1 lever. The cement seal breaks on the more rigid tooth or the least retentive abutment. **B,** This implant was overloaded and failed because the cement seal broke on the natural tooth. The compressive force on the pontic led to a tensile force on the tooth, and the implant acted as a fulcrum. Cement is 10 times weaker under tension. After the cement seal broke from the tooth, all the loads were applied to the implant only, which then failed from overload.

complication rate compared with one tooth joined to two implants. The pier implant exhibits less movement than a terminal abutment and acts as the fulcrum of a Class I lever (Figure 12-31).

This problem is magnified by a longer lever arm such as a pontic between the implant and tooth. A pier implant abutment may cause complications even when joined to nonmobile teeth as terminal abutments. The cement tensile strength is often 20 or more times less than the compressive strength. Therefore when the implant acts as a fulcrum, an uncemented abutment (usually the least mobile tooth or least retentive crown) is a common consequence, with decay being the next most common occurrence.

Uncemented restorations are a common complication in fixed partial dentures, even when all aspects of treatment are within acceptable limits. Any condition that may increase this problem, such as the one presently addressed, should be carefully avoided. For most clinical situations, an additional implant can be placed in at least one of the sites next to a tooth to provide the support needed to fabricate an independent, cantilevered, implant-supported prosthesis. The better option is to perform bone grafting, place implants in both terminal abutment locations next to the natural teeth, and avoid connecting implants to teeth (Figure 12-32).

When bone grafting is not an option and additional implants cannot be inserted, a mobile attachment can be used to restore the implant pier abutment between two natural nonmobile teeth (Figure 12-33). The nonrigid attachment may connect the implant and the least retentive crown to prevent the implant pier abutment from acting as a fulcrum. In conventional fixed prostheses, the "male" portion of a nonrigid attachment usually is located on the mesial aspect of the posterior pontic, whereas the "female" portion is in the distal aspect of the natural pier abutment tooth. This prevents mesial drift from unseating the attachment.[122] However, an implant does not undergo mesial drifting, and the nonrigid connector location is more flexible.

When a natural tooth rather than an implant serves as a pier abutment between two or more implants, the situation is completely different from the previous scenario. When the two or more implants may support the load of the prosthesis alone, the natural tooth becomes a living pontic. In other words, in absence of the tooth, the dental unit would be a pontic.

Because the tooth has greater mobility than the implant and does little to contribute to the support of the prosthetic load, it is referred to as a pontic with a root, or a living pontic (no more than one adjacent site

WWW.HIGHDENT.IR

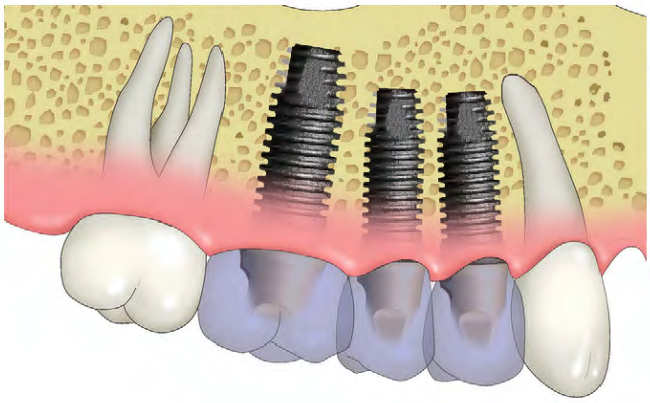


Figure 12-32 A better option is to graft the sites and place implants in terminal abutment locations and to fabricate an independent prosthesis.

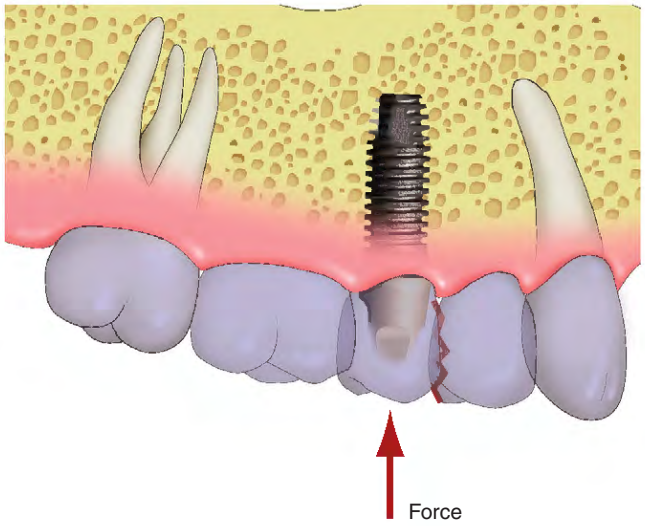


Figure 12-33 When grafting and additional implants are not an option, a mobile attachment may be used to prevent the pier implant from acting as a fulcrum.

should be a pontic) (Figure 12-34). This scenario is best when no additional pontics are between the implants and the tooth. On occasion, multiple implants are splinted together to cantilever one or two pontics, yet a healthy, natural tooth is positioned among the implants. The tooth essentially is ignored in the development of the treatment plan, other than the dentist having to fabricate a crown rather than a pontic in the splinted prosthesis (Figure 12-35).

For a natural pier abutment between two implants, a stress breaker is not indicated. One advantage of keeping the natural tooth, even though it does not contribute to the support of the prosthesis, is the proprioceptive aspect of the periodontal complex.[123,128] Implant prostheses have higher bite forces during mastication than natural tooth restorations because of the decrease in occlusal awareness. A living pontic may decrease the interaction of the forces found during function.

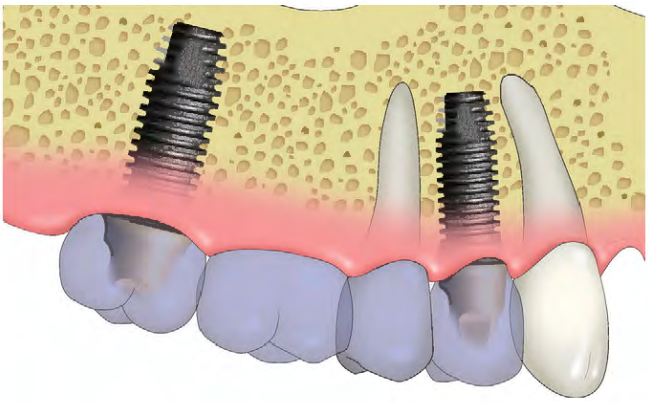


Figure 12-34 When a natural tooth serves as a pier abutment between two or more implants, the tooth may act as a living pontic. No stress breaker is needed in this situation.

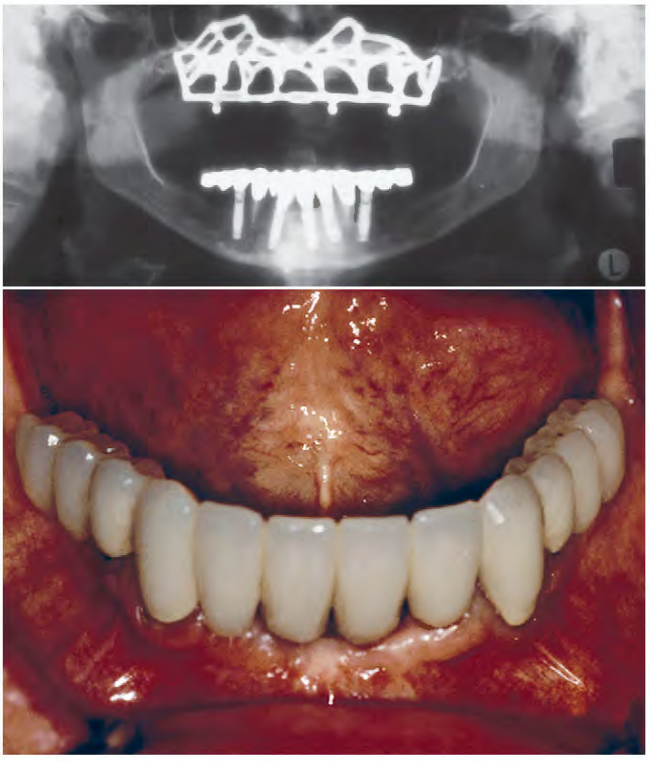


Figure 12-35 **A,** The mandibular restoration has five implants and two natural teeth splinted together, so a cantilever may be used to replace the posterior teeth. The five implants support the load of the restoration. The natural teeth pier abutments act as living pontics. **B,** An intraoral view of the fixed prosthesis. The natural teeth may give some proprioception to the restoration, especially when in the canine position. The support of the restoration is primarily from the implants, which surround the teeth and are splinted together.

Natural Abutment Evaluation

The evaluation of a potential abutment adjacent to an edentulous site includes the following: (1) abutment size, (2) crown/root (implant) ratio, (3) tooth position, (4)parallelism, (5) caries, (6) root configuration, (7) root surface area, (8) endodontic status, and (9) periodontal status.

Abutment Size

Uncemented restorations are one of the most common complication of fixed prostheses.[72-74] After the

WWW.HIGHDENT.IR

crown on the natural abutment becomes uncemented, a significant concern is caries. Decay may proceed rapidly and result in loss of the abutment, creating a need for endodontic treatment, post and core, a new prosthesis, or an abutment with even poorer retention. These same conditions exist if the natural retainer becomes uncemented from an implant-tooth restoration. In addition, the implant is at greater risk. The fixed prosthesis then acts as a cantilever with a dramatic increase in moment force on the implant. Crestal bone loss, prosthesis or abutment screw fracture, implant fracture, or mobility and failure of the implant are likely complications.

When natural and implant abutments are combined in the same prosthesis, uncementation occurs more frequently on the implant. Tooth mobility fatigues the cement seal and increases the forces on the implant. The parameters of retention are similar for a tooth or implant and mainly are influenced by the diameter and height of the abutment.[129-131] Molars are more retentive than premolars because of their increased surface area, all other factors being equal. Wider implant abutments are more retentive than narrower ones. Limited crown height because of limited interarch space also decreases retention. Splinting of teeth with limited crown height to improve retention often compromises access for hygiene in the interproximal areas. Instead, crown lengthening is often indicated in case of limited interarch space to improve the retention of the prosthesis and the esthetic result without compromising home care. A customized abutment of larger diameter can be used on an implant abutment of reduced height. Crowns of reduced size require minimal tapering and additional retentive elements such as grooves or boxes to limit the path of insertion and direction of dislodgment.[130-132]

Crown/Root Ratio

The crown/root ratio represents the height of the crown from the most incisal or occlusal position to the crest of the alveolar ridge around the tooth compared with the height of the root within the bone. This criterion is most important when lateral forces are expected against the crown, as in mandibular excursions. The lateral forces act as a Class I lever on the tooth, with the fulcrum at the crest of the bone. As the crown height increases, the root height decreases, creating a force multiplier.

The crown/root ratio is indicative of the risk of mobility and amount of additional stress the tooth may sustain when used as a fixed partial denture abutment. A patient with a history of periodontal disease may show an increased crown/root ratio, yet no abutment mobility. However, the long-term risk of mobility is increased if the tooth is used as an abutment for a prosthesis. Lateral forces are most detrimental in this situation because of the increased moment force. Splinting may be indicated to distribute stress, and occlusal schemes must be modified to protect these abutments from horizontal stresses.[133] The most ideal crown/root ratio for a fixed prosthetic abutment is 1:2, but this is rarely observed. A more common condition is 1:1.5, and a 1:1 ratio is the minimum requirement when opposing natural teeth or implants and when serving as an abutment for an implant-tooth prosthesis.[134] In addition, the doctor and patient must realize that teeth with an increased crown/root ratio often are restored with a FP-2 or FP-3 prosthesis. A high lip line during smiling and low lip line during speech should be evaluated carefully to determine the prosthetic design. Crown/implant ratio is not considered in a similar way as a crown/root ratio. The implant does not rotate around a center located two thirds down the endosteal/root portion, as does a tooth. Instead, it captures the force at the crest of the ridge. The implant length does not affect its mobility and does not affect its resistance to a lateral force. Although a minimum height requirement does exist and approaches 9 mm, implants greater than 12 mm in healed bone sites do not demonstrate clinical benefit. This is not to say crown height is not important. Crown height is a vertical cantilever on a tooth or implant and will magnify angled, lateral, or cantilever forces. However, the effect of crown height cannot be reduced by increasing the length of the implant. Instead, the dentist should consider reduced cantilever lengths or reduction of angled forces on the prosthesis.

Tooth Position

The dentist considers tooth position next to the edentulous site, including whether the tooth is in the anterior region of the mouth, the intermediate position, or the posterior region. Regardless of arch position, some considerations remain similar. When the natural tooth adjacent to the implant site is in the anterior region, greater mobility and often lateral directions of force are present. Therefore under these conditions the implant is rarely connected to a natural tooth as a terminal abutment. The most common situation in which an implant may be connected rigidly to a natural tooth as a terminal abutment is in a posterior edentulous site with a second or first premolar adjacent to the potential implant site.

The bone adjacent to a natural tooth often is compromised, especially in the long-term edentulous Kennedy-Applegate Class I or Class II patient. Under these conditions, the edentulous site is often deficient in width and height. As previously mentioned, bone grafting in width is much more predictable than height, especially in the posterior mandibular regions.

A sinus graft may provide adequate height of bone for endosteal implants in the posterior maxilla, but onlay grafts on the posterior mandible are much less predictable, and nerve repositioning before implant placement is fraught with potential complications. Bone width augmentation is a usual treatment plan, and bone grafting for height in the posterior maxillas has become a routine procedure, but the posterior mandible is less often a candidate for height augmentation, unless block grafts or more advanced grafting techniques are selected.

WWW.HIGHDENT.IR

When adjacent teeth have been missing for a long time, the remaining natural abutment often has drifted from its ideal position and frequently exhibits tipping, tilting, rotation, or extrusion. The dentist should consider correction of the natural abutment position in the original treatment plan for the partially edentulous patient, whether or not the natural abutment is joined to the implant. A good habit to form is to evaluate and correct any dental unit that will contact the new restoration. Enameloplasty to improve the occlusion or change the contact shape and position next to the implant prosthesis is not unusual. The path of insertion of the implant prosthesis and the size and shape of the interproximal space also may require modification. Treatment also may consist of a crown when beyond the ability merely to reshape the tooth. Orthodontic movement to correct interarch or gross occlusal correction, especially when skeletal patterns require improvement, may be indicated. One can plan orthodontic treatment along with the healing phase for rigid fixated implants. One also may use orthodontic treatment to develop available bone for an implant next to a natural tooth. Moving the tooth slowly through the bone to a more remote position generates bone growth and an improved implant site.

Parallelism

As previously discussed, clinical movement can be eliminated by splinting natural abutments. As such, splinting mandibular incisors is more common in implant dentistry than traditional prosthodontics. These teeth often are crowded or rotated. In addition, the path of insertion of a prosthesis that includes anterior and posterior dental units often requires more extensive tooth preparation.

Some of the indications for attachments in a fixed or removable partial denture include joining nonparallel teeth or splinting anterior and posterior teeth in the same prosthesis. The attachment should usually be rigid in design, size, and fabrication. All of these factors limit the path of insertion of the final prosthesis. Several abutments may need endodontic therapy to achieve this goal. If this is not explained to the patient before treatment begins and endodontic therapy is required, the patient often feels that inadequate treatment has been rendered.

Endodontic therapy or posts and crowns for overlapping anteriors still may provide inadequate embrasures for hygiene. This condition not only compromises esthetics but also may result in the loss of more than one tooth because of periodontal disease. Selective extraction of incisors may even be indicated if rotations or overlapping of teeth creates an unfavorable environment for daily maintenance.[118]

Caries

The dentist should eliminate all carious lesions before implant placement, even when the teeth will be restored with crowns after implant healing for the final prosthesis. Rigidly fixated implants usually require several months of healing after initial placement. The progression of the decay may alter the final treatment plan, with a decrease in crown retention and increased risk for endodontic therapy, posts, cores, or even loss of a desired abutment. Should endodontic therapy be indicated, the obturation of the canals ideally should be completed before implant surgery to avoid possible confusion in the differential diagnosis, if both treatments were overlapping in time and location. If caries are eliminated at the same time as implant surgery, elimination of caries should be performed before reflection of any tissue.

Root Configuration

The natural root configuration may affect the amount of additional stress the tooth may withstand without potential complications.[118] Tapered or fused roots and blunted apexes are examples of decreased ability to withstand the additional occlusal loads required for a fixed prosthesis. The maxillary second molar often presents these varied root configurations. Additional implants and independent implant-supported restorations usually are indicated in the presence of these conditions, rather than the use of these teeth as terminal abutments. Root dilaceration or curvatures improve the support quality of an abutment tooth. However, such root morphology also is likely to encroach on the adjacent available bone volume and increase the risk of implant placement. This is exemplified best in the maxillary canine and first premolar region.

The canine presents a distal angulation of 11 degrees and has a distal root curvature in 60% of the cases. As such, the first premolar edentulous site is limited. An implant inserted into this site usually should be shorter and should follow the angulation of the canine rather than that of the second premolar. The dentist must evaluate carefully any adjacent natural tooth with curved roots at the apex before implant placement.

Roots with a circular cross section do not represent as good a prosthodontic abutment as those with an ovoid cross section. Therefore the maxillary premolar is a better abutment than the maxillary central incisor, although their root surface areas are similar.[130]

The maxillary lateral incisor may exhibit less lateral mobility than the central incisor as a result of its cross-sectional anatomy.[79] All these factors from traditional prosthodontics are also part of the implant candidate's dental evaluation.

Root Surface Area

In general, the greater the root surface area of a proposed abutment tooth, the greater the prosthetic support. Posterior teeth provide greater periodontal surface area and greater support than anterior teeth. Teeth affected by periodontal disease lose surface area and represent poorer support elements for a prosthesis. For a maxillary first molar, bone loss to the beginning of the root furcation corresponds to a root surface area loss of 30%.[134] Ante's law requires the root surface area of the abutment teeth to be equal to or greater than that of the teeth replaced by the pontics of the fixed restoration.[135] Although empirical at its inception,

WWW.HIGHDENT.IR

Ante's law has withstood the test of time and still serves as a clinical guideline.

Endodontic Evaluation

The natural abutment adjacent to or included in a combined tooth and implant-supported prosthesis should present a healthy pulpal status or successful endodontic treatment. If the pulpal or endodontic status of an abutment is questionable, the prudent treatment is endodontic therapy. In this way, the abutment crown may be evaluated for retention, need for post and core, and any other related criteria before final prosthodontic treatment. Potential lesions of endodontic origin are evaluated best before implant surgery because an exacerbation of the lesion during early implant healing may result in a pathway of destruction to the adjacent implant site, implant failure, and extensive bone loss.

In the literature, success has been reported as low as 47% to as high as 98%. However, most studies report success in the range of 85% to 90% at 5 or more years.[136-138] As such, when endodontic treatment has a good prognosis and the tooth may be restored adequately, root canal treatment is in order. However, a number of implant failures each year are attributed to adjacent tooth endodontic failure. At first, this may seem contradictory. Implant healing failures are rare in most practices and account for less than 2% of implants inserted, when using a classic two-step approach. However, when these 2% failures are evaluated, a large number of failures occur next to natural teeth that had an endodontic complication during early implant healing.

Assessment of endodontic success before implant surgery often is difficult.[139-141] The patient may be symptom free, yet a low-grade infection is present at the apex. The healing implant interface is more prone to complications with such a tooth having this condition because the healing interface is weaker than the previous bone condition and a pathway may exist to the developing implant interface. If a tooth is asymptomatic, but has a past endodontic treatment and periapical radiolucency, consideration should be given to retreatment or extraction. When the periapical lesion is 5 mm or greater, the success of endodontic retreatment is not predictable.

During the treatment planning phase, teeth adjacent to the edentulous segment should be scrutinized for potential endodontic problems, keeping in mind that the preparation of a tooth for a crown has a 3% to 6% risk of pulpal death as a consequence of the procedure. In addition, past periodontally involved teeth are at greater risk of pulpal disease after tooth preparation.

Periodontal Status

The periodontal evaluation of natural abutments to be connected to implants is identical to the evaluation of other fixed partial denture abutments. Special attention may be directed to the adjacent implant site, which may be contaminated by bacteria during periodontal surgery.

The incision line and flap design for implant placement often includes the abutment teeth. The implant surgeon should decide whether periodontal therapy is indicated on the abutment teeth before or at the same time as implant placement. A reduction in the number of surgical procedures is a noteworthy benefit to the patient; however, active infection should be minimized during implant placement. Therefore the pathologic condition of the abutment teeth most often is addressed before the soft tissue reflection in the region of the implant osteotomy. Dental prophylaxis and oral hygiene considerations are usually scheduled before implant surgery.

In summary, a completely implant-supported restoration is desirable. Grafting the edentulous site or the use of additional implants is the treatment of choice.

However, when insufficient implant support is available, the natural teeth may be considered as potential abutments. The most important natural tooth criterion for implant-tooth–supported restorations is tooth mobility. A clinical assessment of zero mobility often allows a rigid connection between the tooth and implant. However, if mobility is present, the practitioner should design the prosthesis to include more natural abutments and return the dental elements to zero mobility or consider an independent implant restoration. Splinting natural teeth is the usual method to reduce mobility.

Several additional factors are critical for dual implant-tooth support of a fixed prosthesis: crown size, crown/root ratio, tooth position, parallelism, caries, root configuration, root surface area, endodontics, and periodontal status. Although these same criteria are important for any fixed restoration, each presents unique aspects in implant- and tooth-supported prostheses.

SOFT TISSUE SUPPORT

The evaluation of the soft tissue support is primarily concerned with the treatment planning of RP-5 (overdenture) prostheses, which gain dual support from implants and edentulous ridges. The following factors need to be evaluated: ridge shape, size, parallelism, and palate shape. Large jaws with little resorption provide a better support than smaller sized ones with greater atrophy, in either the maxilla or mandible. The size of the soft tissue can not be solely evaluated on a radiograph, because it is highly dependent on the position of the muscle attachments. High muscle attachments on abundant to moderate bone (Division A or B) may be lowered by vestibuloplasty procedures in conjunction with implant surgery.

Prosthesis support depends on the shape of the residual ridge and, in the maxilla, the palatal vault. A square ridge form yields optimal resistance and stability. A relatively flat one represents a compromised factor for retention and stability, although support is still adequate. Tapering ridges on the palatal vault usually equate with poor stability.[142-144] Ridge parallelism is also evaluated. The edentulous ridge parallel to the occlusal plane is

WWW.HIGHDENT.IR

Figure 12-36 **A,** The patient has a collapsed occlusal vertical dimension and a poor occlusal plane. When the final result is not clear to the restoring dentist, a treatment prosthesis is often a benefit to assess and reestablish prosthetic parameters. **B,** An acrylic removable partial denture in place is used as a treatment prosthesis to reestablish the proper occlusal vertical dimension, jaw position, plane of occlusion, temporomandibular joint status, speech, and potential prosthetic result.

most favorable for soft tissue support. If ridges are divergent, stability of the denture will be greatly affected.

The lateral throat form in a maxillary denture or RP-5 restoration is evaluated. A soft palate slope is favorable when it has a long, gradual slope from the junction of the hard and soft palate,[145] which allows a greater extension of the posterior palatal seal and enhances retention. On the other hand, a soft palate Class III, which drops abruptly, may lead to soreness, loss of valve seal, and gagging. These elements are of great diagnostic value in the evaluation of the maxillary fully edentulous patient who may consider an implant-supported overdenture. A greater number of unfavorable anatomical structures may direct the treatment plan toward an RP-4 prosthesis with greater implant support and no soft tissue support to address all the needs of the patient.

It must be emphasized to the patient that a partial or total soft tissue–borne prosthesis will not stabilize bone loss. To the contrary, bone loss will continue and may even be accelerated, because the prosthesis is more often worn and bite forces are increased. As a result, all soft tissue–borne devices should be considered transitional dentures. They all require repeated relines, rebasing, and refabrication to replace the missing bone. A totally implant-supported restoration (fixed or removable) does not require soft tissue support and may be considered a definitive restoration.

Many soft tissue–supported restorations are fabricated because the patient can not afford a totally implant-supported restoration, especially in the completely edentulous patient. However, the doctor often forgets that if a patient cannot afford the ideal treatment today, it does not mean the patient cannot afford any further treatment later. For example, if a patient needs four first molars replaced, but cannot afford all restorations at this time, the doctor most often can still restore one of the molars. Then a few years later the next tooth may be restored. Eventually, the four molars are treated and the arch form and occlusion restored. In similar fashion, a patient who can afford only two implants to retain a mandibular denture could possibly afford further treatment later. Therefore a lifetime strategy for health should be established, which may include the addition of more implants in the future to reduce and eventually halt the continued bone loss and consequences on esthetics and function.

PRETREATMENT PROSTHESES

FIXED RESTORATIONS

Pretreatment prostheses in restorative dentistry are often indicated to obtain a diagnosis, improve soft tissue health before fabricating soft tissue–borne restorations, reestablish the OVD, evaluate esthetic considerations, or treat TMJ dysfunction (Figure 12-36). Restoration of implant patients may also require treatment prostheses for similar reasons. In addition, the pretreatment prosthesis may be used to select a prosthetic option, to progressively load bone to improve its strength, and as a transitional restoration to protect a healing bone graft or implant. Immediate loading of an implant system often uses a transitional prosthesis out of occlusion in a partial edentulous situation. In the completely edentulous immediate load restoration, the transitional prosthesis has no cantilevers in nonesthetic areas. Treatment prostheses may also help evaluate the psychologic attitude of a patient before irreversible implant procedures (Box 12-6).

Diagnosis in medicine is the first step to establishing a treatment for a disease or disorder. Likewise, to establish a treatment plan for a partially or completely edentulous patient, a proper diagnosis should be established. A treatment prosthesis may be required to help in this process. For example, questionable teeth

WWW.HIGHDENT.IR

Box 12-6 Implant Treatment Prostheses

1. Help with diagnosis
   a. Crown lengthening
   b. Occlusal plane
   c. Hopeless teeth
2. Evaluate the psychologic profile of the patient
   a. Denture before implant surgery
3. Improve soft tissues before final impression for implant overdentures
4. Postoperative to implant surgery
5. Evaluate occlusal vertical dimension
6. Evaluate temporomandibular joint dysfunction
7. Improve implant position related to final tooth position
8. Evaluate esthetics before surgery
9. Evaluate hygienic contours of fixed restorations
10. Determine whether removable restoration is required for maxillary lip support (RP versus FP)
11. Protect bone graft or implants during healing
12. Patient's financial and compliance management
13. Progressive bone loading
14. Phonetics and esthetics for full arch implant–fixed prostheses on complete edentulous patients

RP, Removable prosthesis; *FP,* fixed prostesis.

may require initial restoration to assess their prognosis related to whether or not the extraction of the tooth and implant replacement therapy is required.

A treatment prosthesis may correct the existing occlusal plane, identify extruded teeth, and indicate whether endodontic therapy, crown lengthening, or extraction is required to complete the final treatment plan. Remember, after prosthetic crown lengthening is performed, at least 4 mm of tooth structure is supracrestal (2 mm for connective tissue and junctional epithelial attachment and 2 mm to create a ferrule effect with the crown to reduce the risk of root fracture). Also, the crown/root ratio is increased and the mobility of the tooth should be evaluated. Excessive mobility may require additional implants, splinting teeth, or even extraction and additional implant insertion.

A long-span partially edentulous patient often wears a fixed-treatment prosthesis, which also acts as an interim prosthesis. Metal-reinforced transitional prostheses may be used when four or five pontics are present. These fixed, transitional treatment restorations may be used during bone grafts or healing of implants to decrease forces on the soft tissues and on the graft or healing implants.

REMOVABLE PROSTHESES

Treatment prostheses may be used to improve the soft tissues used for support, stability, or retention before RP-5 overdenture or complete denture restorative procedures. The first evidence of residual ridge destruction by an ill-fitting denture is often deformed and traumatized overlying soft tissue.[146] The soft tissue bed may exhibit different degrees of redundant hyperplasia, epulis, hypertrophy, or abrasions.[147,148] A tissue conditioning treatment is usually indicated to restore soft tissue health before making the final impression for the soft tissue bone prosthesis. The soft tissue conditioner may need to be replaced every 2 to 3 days, although 10 to 14 days are usually sufficient to return the soft tissue to normal condition. The existing denture can often be used as the treatment prosthesis. Additional treatment such as surgical removal of excessive hypermobile tissues is often warranted before soft tissue conditioning.

It should be noted that soft tissue conditioners are different from soft liners used in soft tissue support areas of removable prostheses. Tissue conditioners change dimensions during the first 18 to 24 hours. As such, as the tissues return to a more normal condition, the material changes dimension to allow and encourage these changes. However, the modifiers required for this reaction leach out of the material, halt the process within a day, and result in a stiff material. Soft liners, on the other hand, stay soft longer than tissue conditioners, especially when coated with a sealer. However, the material does not change dimension during the first day and therefore will not accommodate a changing tissue condition.

Most often, tissue conditioners are used to improve abused tissues before a final soft tissue impression for a removable prosthesis. In addition, these materials are used after implant surgery in regions under a removable prosthesis, while the implant-bone interface heals. The tissue conditioner may respond to the swelling and tissue changes immediately after soft tissue reflection. In addition, it is relieved over the implant site. At the suture removal appointment, the tissue conditioner is removed and replaced with a sealed soft liner. This material stays soft during extended periods and is less likely to load the implant through the soft tissue.

OCCLUSAL VERTICAL DIMENSION

Long-term edentulous patients who have been wearing the same denture may require a treatment prosthesis to restore the OVD and ridge relationship before implant treatment.[149] The OVD may gradually collapse, especially in the completely edentulous patient, as a result of continued bone loss and prosthesis occlusal wear. TMJ and myofacial dysfunction may be the further consequence of this condition. A treatment prosthesis to reestablish the proper OVD or assess a symptomatic joint helps determine the patient's specific needs regarding the dysfunction.

As the OVD decreases, the mandibular jaw rotates forward and closes in a more prognathic pseudo–Class III relationship. To place the implants in the correct angulation, the OVD should be reestablished before implant surgery so the correct position of the teeth

WWW.HIGHDENT.IR

relative to the arch is established. In the case of immediate implant loading, a treatment prosthesis is delivered at or soon after the implant surgery. The design of the prosthetic superstructure concomitant with the implant substructure is necessary for immediate loading in implant overdentures. Therefore a treatment prosthesis is indicated to establish the proper OVD and tooth position before the placement of the implants and fabrication of the superstructure bar.

As the OVD increases, the maxillomandibular relationship evolves toward a Class II relationship. This influences the position or angulation of the implant. In addition, the location of an overdenture bar may be equally influenced by variations of the OVD. The treatment prosthesis may be used to establish prosthetic position of teeth.

A pretreatment prosthesis for a completely edentulous patient before the delivery of a fixed- or removable-implant prosthesis is most often a complete denture. It is fabricated with acrylic teeth to facilitate recontouring and the addition of cold-cured acrylic for repairs or to change the OVD or lip support.

ESTHETIC ASSESSMENT

On occasion, a patient's desire for esthetic improvement may be very demanding or unrealistic. In the completely edentulous patient, a treatment denture (partial or complete) may be used to satisfy those esthetic concerns before implant surgery. Tooth shape, surface quality, size and position, tooth color, lip and soft tissue contour, tooth position, gingival color, soft tissue contour, and papilla support may all be evaluated. If the patient cannot be satisfied with the pretreatment prosthesis, it is far better to realize this before implant placement. Although demanding patients may not be satisfied with the pretreatment prosthesis, they can decide to lower expectations and continue with treatment or be referred to another dentist. If the latter is chosen, it is prudent to contact the next practitioner and inform them that another pretreatment prosthesis is indicated before implant placement.

A high lip line in the maxilla or low lip line position in the mandible may influence the need for a specific gingival contour and color in the restoration, yet the maintenance needs of the restoration may compromise the final esthetic result. A fixed restoration must be designed to allow access for proper hygiene procedures around the teeth and implants. A pretreatment prosthesis may help determine whether an implant-supported removable prosthesis rather than a fixed restoration is required to satisfy the patient's esthetic goals and desires for the restoration, yet may be removed to allow proper daily maintenance.

The maxillary vermillion border of this lip is usually altered by the loss of the maxillary anterior teeth. After bone is also lost, the natural support of the entire lip is often deficient and depends on the labial flange of the prosthesis. A fixed partial denture may require an anterior cantilever away from the soft tissue in a horizontal and vertical dimension to provide this support. A pretreatment prosthesis can provide the information required to determine whether a fixed prosthesis will compromise esthetics, support, or hygiene in this region above the teeth.

PSYCHOLOGIC ATTITUDE

The finalized treatment plan and patient's physical and mental evaluation should be assessed before implant surgery. If the restoring dentist is not sure the planned final prosthesis is compatible with the desires of the patient, or the patient's attitude and demand do not seem reasonable, further evaluation is required. A pretreatment prosthesis provides additional appointments and time for these evaluations.[150,151]

FINANCIAL BARRIERS

An additional benefit of pretreatment prostheses is that patient financial management may be facilitated and compliance issues resolved before more irreversible phases of treatment are rendered. However, it is beneficial to clearly establish the cost and emphasize the need to progress forward with treatment in an orderly fashion. Very few pretreatment or transitional restorations may be worn for years without risk of fracture, uncementation, or compromises in the bone graft or implants.

PROGRESSIVE LOAD

A pretreatment prosthesis to improve the quality of bone is most always used in D3 or D4 bone-supporting implants before the fabrication of the final restoration. Interim (provisional) acrylic restorations that gradually load bone for progressive loading may be considered pretreatment prostheses. A decrease in crestal bone loss and decrease in implant failure, especially in soft bone types, are particular advantages with progressively loaded treatment prostheses. Pretreatment prostheses also assist in the determination of the final form and function of the final prosthesis, especially for completely edentulous patients, for whom the "pretreatment" prosthesis may be the first full arch–fixed restoration they have worn after several years of wearing a complete denture.

SUMMARY

Preimplant prosthodontics for partially edentulous patients include overall evaluation of five intraoral segments as: (1) the maxillary incisal edge, (2) the OVD, (3) the

WWW.HIGHDENT.IR

mandibular incisor edge, (4) the maxillary occlusal plane, and the (5) mandibular occlusal plane. In addition, there are 10 specific criteria that affect a treatment plan: (1) lip lines, (2) maxillomandibular relationships, (3) existing occlusion, (4) crown height space, (5) TMJ status, (6) extraction of hopeless or guarded-prognosis teeth, (7) existing prosthesis, (8) arch form, (9) natural tooth adjacent to an edentulous space, and (10) soft tissue evaluation. Pretreatment prostheses are also used in an implant prosthetic evaluation process (Box 12-7).

The prosthodontic evaluation of the implant candidate borrows several conventional criteria from the evaluation of natural abutments. In addition, many of these situations require a unique approach for implant prosthodontics and may influence the implant treatment plan. The goal of the implant surgeon is to achieve predictable, rigid fixation of endosteal implants. The restoring dentist's responsibility is to maintain the implant-bone interface in an environment that satisfies all the traditional prosthodontic criteria.

Box 12-7 Treatment Sequence for Implant-Supported Reconstructions

The proposed treatment for implant-supported reconstructions performed by the implant team can be as follows:

Initial appointments

Medical and dental history
Dental evaluation and x-ray examinations
Diagnostic casts
Preliminary discussion of treatment alternatives
Decision to proceed with treatment
Initial treatment plan, case presentation, and alternatives
Clinical/laboratory procedures prior to additional diagnostic records
Extra office diagnostic orders (e.g., setup, computed tomography scans, tests for medical evaluation, consultation, and team members)
Diagnostic wax-up of final results on duplicate diagnostic casts
Final treatment plan and alternatives
Medical laboratory tests evaluated
Prescriptions and postoperative instructions
Consent forms and request for treatment forms
Pictures of existing condition

Phase I dentistry

Presurgical restorative appointment—initial caries removal, extractions, temporary teeth
Periodontal treatment, endodontic therapy, orthodontics
Occlusal vertical dimension
Occlusal plane correction, treatment prosthesis, recontour existing teeth, enamoplasty
Transitional prosthesis (removable or fixed) or diagnostic try-in; tissue conditioning
Impression for surgical guide template (if oral condition altered from initial diagnostic cast)

Preimplant reconstructive surgery osseous (grafting), soft tissue

Implant surgery

Stage I: implant placement

Healing phase

Stage II: secondary permucosal extension, initial loading

Prosthodontics: progressive bone loading
Initial abutment preparation and impression
Final abutment preparation and impression
Metal try-in or waxed teeth try-in
Initial delivery—occlusal
Final delivery—occlusal adjustment
Night guard

Maintenance

First year: every 3 to 4 months
Radiographs at 6 months, then annually for 3 years, then as required
Home care education
Fluoride on teeth
Chlorhexidine on implants

WWW.HIGHDENT.IR

References

1. Rufenacht CR: *Fundamentals of esthetics*, Chicago, 1990, Quintessence.
2. Lynn BD: The significance of anatomic landmarks in complete denture service, *J Prosthet Dent* 14:456, 1964.
3. Harper RN: The incisive papilla: the basis of a technique to reproduce the positions of key teeth in prosthodontics, *J Dent Res* 27:661, 1948.
4. Vig RG, Brundo GC: The kinetics of anterior tooth display, *J Prosthet Dent* 39:502-504, 1978.
5. Hulsey CM: An esthetic evaluation of lip-teeth relationships present in smile, *Am J Orthod* 57:132, 1970.
6. Matthews TG: The anatomy of smile, *J Prosthet Dent* 39:128, 1978.
7. Robinson SC: Physiological placement of artificial anterior teeth, *Can Dent J* 35:260-266, 1969.
8. Misch CE: Incisal edge position, the canine is the guide, *J Prosthodontics* (in press).
9. The glossary of prosthodontic terms, *J Prosthet Dent* 81:39-110, 1999.
10. Sharry JJ: *Complete denture prosthodontics*, New York, 1968, McGraw-Hill.
11. Shannon TEJ: Physiologic vertical dimension and centric relation, *J Prosthet Dent* 6:741-747, 1956.
12. Kois JC, Phillips KM: Occlusal vertical dimension: alteration concerns, *Compend Contin Educ Dent* 18: 1169-1180, 1997.
13. Niswonger ME: The rest position of the mandible and centric relation, *J Am Dent Assoc* 21:1572-1582, 1934.
14. Silverman MM: Accurate measurement of vertical dimension by phonetics and the speaking centric space, part I, *Dent Dig* 57:265, 1951.
15. Pound E: Let /S/ be your guide, *J Prosthet Dent* 38: 482-489, 1977.
16. Pound E: Utilizing speech to simplify a personalized denture service, *J Prosthet Dent* 24:586-600, 1970.
17. McGee GF: Use of facial measurements in determining vertical dimension, *J Am Dent Assoc* 35:342-350, 1947.
18. Danikas D, Panagopoulos G: The golden ratio and proportions of beauty, *Plast Reconstr Surg* 114:1009, 2004.
19. Amoric M: The golden number: applications to cranio-facial evaluation, *Funct Orthod* 12:18, 1995.
20. Haralabakis NB, Lagoudakis M, Spanodakis E: A study of esthetic harmony and balance of the facial soft tissue [in Greek (modern)], *Orthod Epitheor* 1:175, 1989.
21. da Vinci L: *The anatomy of man*. Drawings from the collection of Her Majesty Queen Elizabeth II, Windsor, United Kingdom, ca 1488.
22. Misch CE: Vertical occlusal dimension by facial measurement, *Continuum: Misch Implant Institute Newsletter*, summer, 1997.
23. Misch CE: Objectives subjective methods for determining vertical dimensions of occlusion, *Quintessence Int* 31: 280-281, 2000.
24. Mach MR: Facially generated occlusal vertical dimension, *Compendium* 18:1183-1194, 1997.
25. Ricketts RM: The biologic significance of the divine proportion and Fibonacci series, *Am J Orthod* 1:357-370, 1982.
26. Brzoza D, Barrera N, Contasti G et al: Predicting vertical dimension with cephalograms, for edentulous patients, *Gerodontology* 22:98-103, 2005.
27. Ciftci Y, Kocadereli I, Canay S et al: Cephalometric evaluation of maxillomandibular relationships in patients wearing complete dentures: a pilot study, *Angle Orthod* 75:821-825, 2005.
28. Kelly E: Changes caused by a mandibular removable partial denture opposing a maxillary complete denture, *J Prosthet Dent* 27:140-150, 1978.
29. Renner RP, Boucher LJ: *Removable partial dentures*, Chicago, 1987, Quintessence.
30. Laney WR, Gibilisco JA: *Diagnosis and treatment in prosthodontics*, Philadelphia, 1983, Lea & Febiger, pp 164-165.
31. Kokich VO, Kiyak Hl, Shapiro PA: Comparing the perception of dentists and lay people to altered dental esthetics, *J Esthetic Dent* 11:311-324, 1999.
32. Kokich, VG, Spear FM, Kokich VO: Maximizing anterior esthetics: an interdisciplinary approach: esthetics and orthodontics. In McNamara JA, editor: *Craniofacial growth series*, Ann Arbor, Mich, 2001, Center for Human Growth and Development, University of Michigan.
33. Levin EI: Dental esthetics and the golden proportion, *J Prosthet Dent* 40:244, 1978.
34. Pound E: *Personalized denture procedures: dentist's manual*, Anaheim, Calif, 1973, Denar Corp.
35. Robbins WJ: The incisal edge position in complex restorative dentistry, *Texas GP*, 12-13, spring, 2000.
36. Tjan AHL, Miller GD, Josephine GP: Some esthetic factors in a smile, *J Prosthet Dent* 51:24-28, 1984.
37. Crispin BJ, Watson JF: Margin placement of esthetic veneer crowns. Part 1: anterior tooth visibility, *J Prosthet Dent* 45:278-282, 1981.
38. Cade RE: The role of the mandibular anterior teeth in complete denture esthetics, *J Prosthet Dent* 42:368-370, 1979.
39. Pietrokovski J, Masseler M: Alveolar ridge resorption following tooth extraction, *J Prosthet Dent* 17:21-27, 1967.
40. Misch CE: Natural teeth adjacent to multiple implant sites: effect on diagnosis and treatment plan. In Misch CE, editor: *Dental implant prosthetics*, St Louis, 2005, Mosby.
41. Misch CE, Goodacre CJ, Finley JM et al: Consensus Conference Panel Report: crown-height space guidelines for implant dentistry—part 1, *Implant Dent* 14:312-318, 2005.
42. Misch CE, Goodacre CJ, Finley JM et al: Consensus Conference Panel Report: crown-height space guidelines for implant dentistry—part 2, *Implant Dent* 15:113-121, 2006.
43. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent*, 90, 121-132, 2003.
44. Bragger U, Aeschlimann S, Burgin W et al: Biological and technical complications and failures with fixed partial dentures (FPD) on implants and teeth after four to five years of function, *Clin Oral Impl Res* 12:26-43, 2001.
45. Bidez MW, Misch CE: Issues in bone mechanics related to oral implants, *Impl Dent* 1:289-294, 1992.
46. Bidez MW, Misch CE: Force transfer in implant dentistry: basic concepts and principles, *J Oral Implant* 18:264-274, 1992.
47. Kakudo Y, Amano N: Dynamic changes in jaw bones of rabbit and dogs during occlusion, mastication and swallowing, *J Osaka Univ Dent Soc* 6:126-136, 1972.
48. Kakudo Y, Ishida A: Mechanism of dynamic responses of canine and human skull due to occlusal masticatory

WWW.HIGHDENT.IR

and orthodontic forces, *J Osaka Univ Dent Soc* 6: 137-144, 1972.
49. Jensen OT, Cockrell R, Kuheke L et al: Anterior maxillary alveolar distraction osteogenesis—a prospective 5-year clinical study, *Int J Oral Maxillofac Implants* 17:507-516, 2002.
50. Bidger DV, Nicholls JI: Distortion of ceramometal fixed partial dentures during the firing cycle, *J Prosthet Dent*, 45:507-514, 1981.
51. Bertolotti RL, Moffa JP: Creep rate of porcelain-bonding alloys as a function of temperature, *J Dent Res* 59: 2061-2065, 1980.
52. Bryant RA, Nicholls JI: Measurement of distortion in fixed partial dentures resulting from degassing, *J Prosthet Dent* 42:515-520, 1979.
53. Finley JM: Personal Communication, 2005.
54. Smyd E: Mechanics of dental structures. Guide to teaching dental engineering at undergraduate level, *J Prosthet Dent* 2:668-692, 1952.
55. English CE: The mandibular overdenture supported by implants in the anterior symphysis: a prescription for implant placement and bar prosthesis design, *Dental Implantol Update* 4:9-14 1993.
56. Dawson PE: *Evaluation, diagnosis and treatment of occlusal problems*, ed 2, St Louis, 1989, Mosby.
57. Dawson PE: Determining the determinants of occlusion, *Int Periodont Rest Dent* 6:9, 1983.
58. Tanaka TT: Recognition of the pain formula for head, neck and TMJ disorders. The general physical examination, *Calif Dent Assoc J* 12:43-49, 1984.
59. Farzaneh M, Abitbol S, Friedman S: Treatment outcome in endodontics: the Toronto study. Phases I and II: orthograde retreatment, *J Endod* 30:627-633, 2004.
60. Farzaneh M, Abitbol S, Lawrence HP et al: Treatment outcome in endodontics—the Toronto study. Phase II: initial treatment, *J Endod* 30:302-309, 2004.
61. Klokkevold PR, Newman MG: Current status of dental implants: a periodontal perspective, *Int J Oral Maxillofac Implants* 15:56-65, 2000.
62. Bower RC: Furcation morphology relative to periodontal treatment: furcation entrance architecture, *J Periodontol* 50:23-27, 1979.
63. Fugazzotto PA: A comparison of the success of root resected molars and molar position implants in function in a private practice: results of up to 15-plus years, *J Periodontol* 72:1113-1123, 2001.
64. Linkow LI, Chercheve R: *Theories and techniques in oral implants*, St Louis, 1970, Mosby.
65. Balshi TJ: Osseointegration for the periodontally compromised patient, *Int J Prosthodont* 1:51-58, 1988.
66. Reider CE: Copings on tooth and implant abutments for superstructure prostheses, *Int J Periodontics Restorative Dent* 10:437-454, 1990.
67. Langer B, Sullivan DY: Osseointegration: its impact on the interrelationship of periodontics and implant dentistry, part II, *Int J Periodontics Rest Dent* 9:165-183, 1989.
68. Hamp S-E, Ravald N, Teiwik A et al: Modes of furcation treatment in a long-term prospective study, *J Parodontol* 11:11-23, 1992.
69. Muller H-P, Eger T, Lange DE: Management of furcation-involved teeth: a retrospective analysis, *J Clin Periodontol* 22:911-917, 1995.
70. Wang HL, Burgett FG, Shyr Y et al: The influence of molar furcation involvement and mobility on future clinical periodontal attachment loss, *J Periodontol* 65: 25-29, 1994.
71. English CE: Prosthodontic prescriptions for mandibular implant overdentures—part 1, *Dental Implantol Update* 7:25-28, 1996.
72. Holm C, Tidehaq P, Tillberg A et al: Longevity and quality of FPDs: a retrospective study of restorations 30, 20, and 10 years after insertion, *Int J Prosth* 16:283-289, 2003.
73. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
74. Tan K, Pjetursson BE, Lang NP et al: A systematic review of the survival and complication rates of fixed partial dentures (FPDs) after an observation period of at least 5 years. III. Conventional FPDs, *Clin Oral Impl Res* 15: 654-666, 2004.
75. Priest G: Failure rates of restorations for single tooth replacement, *Int J Prosthodont* 9:38-45, 1996.
76. Pjetursson BE, Tan K, Lang NP et al: A systematic review of the survival and complication rates of fixed partial dentures (FPDs) after an observation period of at least 5 years. I. Implant-supported FPDs, *Clin Oral Impl Res* 15:625-642, 2004.
77. Priest G, Priest J: The economics of implants for single missing teeth, *Dental Econ* 130-138, 2004.
78. Muhlemann HR: Tooth mobility: a review of clinical aspects and research findings, *J Periodontol* 38:686-708, 1967.
79. Parfitt GS: Measurement of the physiologic mobility of individual teeth in an axial direction, *J Dent Res* 39: 608-612, 1960.
80. Sekine H, Komiyama Y, Hotta H et al: Mobility characteristics and tactile sensitivity of osseointegrated fixture-supporting systems. In van Steenberghe D, editor: *Tissue integration in oral maxillofacial reconstruction*, Amsterdam, 1986, Elsevier.
81. Dixon DI, Breeding LC, Sadler JB et al: Comparison of screw loosening, rotation, and deflection among three implant designs, *J Prosthet Dent* 74:270-278, 1995.
82. US Food and Drug Administration: MDR data device experience network database, Rockville, MD, Center for Devices and Radiological Health CDRH/FDA, 1995.
83. Balshi TJ, Hernandez RE, Pryszlak MC et al: A comparative study of one implant versus two replacing a single molar, *Int J Oral Maxillofac Implants* 11:372-378, 1996.
84. Gunne J, Astrand P, Ahlen K et al: Implants in partially edentulous patients, *Clin Oral Implants Res* 3:49-56, 1992.
85. Naert I, Quirynen M, van Steenberghe D et al: A six year prosthodontic study of 509 consecutively inserted implants for the treatment of partial edentulism, *J Prosthet Dent* 67:236-245, 1992.
86. Hemmings KW, Schmitt A, Zarb GA: Complications and maintenance requirements for fixed prostheses and overdentures in the edentulous mandible: a 5-year report, *Int J Oral Maxillofac Implants* 9:191-196, 1994.
87. Gunne J, Jemt T, Linden B: Implant treatment in partially edentulous patients: a report on prostheses after 3 years, *Int J Prosthodont* 7:143-148, 1994.
88. Schnitman PA, Wohrle PS, Rubenstein JE: Immediate fixed prostheses supported by two-stage threaded implants: methodology and results, *Oral Implantol* 16:96-105, 1990.
89. Gottehrer NR, Singer G: Full team approach for provisional stabilization of edentulous implant patients, *Dent Today* 15:56-59, 1996.

WWW.HIGHDENT.IR

90. English CE: The critical A-P spread, *Implant Soc* 1:2-3, 1990.
91. Falk H, Laurell L, Lundgren D: Occlusal force pattern in dentitions with mandibular implant-supported fixed cantilever prostheses occluded with complete dentures, *Int J Oral Maxillofac Implants* 4:55-62, 1989.
92. Falk H, Laurell L, Lundgren D: Occlusal interferences and cantilever joint stress in implant-supported prostheses occluding with complete dentures, *Int J Oral Maxillofac Implants* 5:70-77, 1990.
93. White SN, Caputo AA, Anderkvist T: Effect of cantilever length on stress transfer by implant-supported prostheses, *J Prosthet Dent* 71:493-499, 1994.
94. Wang S, Hobkirk JA: Load distribution on implants with a cantilevered superstructure: an in vitro pilot study, *Implant Dent* 5:36-42, 1996.
95. McAlarney ME, Stavropoulos DN: Determination of cantilever length-anterior-posterior spread ratio assuming failure criteria to be the compromise of the prosthesis retaining screw-prosthesis joint, *Int J Oral Maxillofac Implants* 11:331-339, 1996.
96. Schackleton JL, Carr L, Slabbert JC et al: Survival of fixed implant supported prostheses related to cantilever lengths, *J Prosthet Dent* 71:23-26, 1994.
97. Takayama H: Biomechanical considerations on osseointegrated implants. In Hobo S, Ichida E, Garcia CT, editors: *Osseointegration and occlusal rehabilitation,* Chicago, 1989, Quintessence.
98. English CE: Biomechanical concerns with fixed partial dentures involving implants, *Implant Dent* 2:221-242, 1993.
99. Ericsson I, Lekholm U, Brånemark PI et al: A clinical evaluation of fixed bridge restoration supported by the combination of teeth and osseointegrated titanium implants, *J Clin Periodontol* 13:307-312, 1986.
100. Kirsch A, Mentag PJ: The IMZ endosseous two phase implant system: a complete oral implantation, *J Oral Implantol* 12:576-589, 1986.
101. Astrand P, Borg K, Gunne J et al: Combination of natural teeth and osseointegrated implants as prosthesis abutments: a 2 year longitudinal study, *Int J Oral Maxillofac Implants* 6:305-312, 1991.
102. Klinge B: Implants in relation to natural teeth, *J Clin Periodontol* 18:482-487, 1991.
103. Rieder CE, Parel SM: A survey of natural tooth abutment intrusion in implant connected fixed partial dentures, *Int J Periodontics Restorative Dent* 13:335-347, 1993.
104. Sheets CG, Earthman JC: Tooth intrusion in implant-assisted prostheses, *J Prosthet Dent* 77:39-45, 1997 (erratum appears in *J Prosthet Dent* 77:453, 1997).
105. Pesun IJ: Intrusion of teeth in the combination implant-to-natural-tooth fixed partial denture: a review of the theories, *J Prosthodont* 6:268-277, 1997.
106. Rangert B, Gunne J, Sullivan DY: Mechanical aspects of a Brånemark implant connected to a natural tooth: an in vitro study, *Int J Oral Maxillofac Implants* 6:177-186, 1991.
107. Picton DCA: On the part played by the socket in tooth support, *Arch Oral Biol* 10:945-955, 1965.
108. Phillips RW: Personal communication, 1990.
109. Phillips RW: *Skinner's science of dental materials,* ed 9, Philadelphia, 1991, Saunders.
110. Bidez MW, Lemons JE, Isenberg BF: Displacements of precious and nonprecious dental bridges utilizing endosseous implants as distal abutments, *J Biomed Mater Res* 20:785-797, 1986.
111. Komiyama Y: Clinical and research experience with osseointegrated implants in Japan. In Albrektsson T, Zarb G, editors: *The Brånemark osseointegrated implant,* Chicago, 1989, Quintessence.
112. Fenton AH, Jamshaid A, David D: Osseointegrated fixture mobility, *J Dent Res* 66:114, 1987.
113. van Steenberghe D: A retrospective multicenter evaluation of the survival rate of osseointegrated fixtures supporting fixed partial prostheses in the treatment of partial edentulism, *J Prosthet Dent* 61:217-222, 1989.
114. McGlumphy EA, Campagni WV, Peterson LJ: A comparison of the stress transfer characteristics of dental implants with a rigid or a resilient internal element, *J Prosthet Dent* 62:589-592, 1989.
115. O'Leary TJ, Dykema RW, Kafrawy AH: Reactions to osseointegrated implants rigidly attached to natural teeth, *J Dent Res* 72:347, 1993.
116. Lundgren D, Falk H, Laurell L: Prerequisites for a stiff connection between osseointegrated implants and natural teeth, *J Dent Res* 67:247, 1988.
117. Misch CE, Bidez MW: Implant protected occlusion, a biomechanical rationale, *Compendium* 15:1330-1342, 1994.
118. Laney WR, Gibilisco JA: *Diagnosis and treatment in prosthodontics,* Philadelphia, 1983, Lea & Febiger.
119. Wylie R, Caputo AA: Force distribution to periodontally involved teeth by fixed splints [abstract], *J Dent Res* 61:1030, 1982.
120. Tylman SD: *Theory and practice of crown and fixed partial prosthodontics,* St Louis, 1965, Mosby.
121. Caputo AA, Standlee JP: *Biomechanics in clinical dentistry,* Chicago, 1987, Quintessence.
122. Shillingburg HT, Fisher DW: Nonrigid connectors for fixed partial dentures, *J Am Dent Assoc* 87:1195-1199, 1973.
123. Cavicchia F, Bravi F: Free standing vs tooth connected implant supported fixed partial restoration: a comparative retrospective clinical study of the prosthetic results, *Int J Oral Maxillofac Implants* 9:711-718, 1996.
124. Ismail YH, Misch CM, Pipko DJ et al: Stress analysis of a natural tooth connected to an osseointegrated implant in a fixed prosthesis, *J Dent Res* 70:460, 1991.
125. Dimilano GP, Corrente G: Photoelastic evaluation of attachments in tooth connected implant restorations in relation to residual periodontal support, *Riv Ital Osteointegrazione* 2(Suppl 1):35, 1992.
126. Misch CM, Ismail YH: Finite element analysis of tooth to implant fixed partial denture designs, *J Prosthodont* 2:83-92, 1993.
127. Cho GC, Chee WL: Apparent intrusion of natural teeth under an implant supported prosthesis: a clinical report, *J Prosthet Dent* 68:3-5, 1992.
128. Kay HB: Free standing implant-tooth interconnected restorations: understanding the prosthodontic perspective, *Int J Periodontics Restorative Dent* 13:47-69, 1993.
129. DeClercq M, Naert I, Theuniers G et al: Damages at implant parts and prosthetical superstructures supported by osseointegrated implants, *J Dent Res* 68:901, 1989.
130. Shillinburg HT, Hobo S, Whitsett LD et al: *Fundamentals of fixed prosthodontics,* ed 3, Chicago, 1997, Quintessence.
131. Kaufmann EG, Coelho AB, Colin L: Factors influencing the retention of cemented gold castings, *J Prosthet Dent* 11:487-502, 1961.
132. Jorgensen KD: The relationship between retention and convergence angle in cemented veneer crowns, *Acta Odontol Scand* 13:35-40, 1955.

WWW.HIGHDENT.IR

133. Reynolds JM: Abutment selection for fixed prosthodontics, *J Prosthet Dent* 19:483, 1968.
134. Penny RE, Kraal JH: Crown to root ratio: its significance in restorative dentistry, *J Prosthet Dent* 42:34-38, 1979.
135. Ante IH: The fundamental principles of abutments, *Mich Dent Soc Bull* 8:14, 1926.
136. Sjogren U, Hagglund B, Sundquist G et al: Factors affecting the long-term results of endodontic treatment, *J Endod* 16:498-504, 1990.
137. Peak JD: The successof endodontic treatment in general dental practice: a restrospective clinical and radiographic study, *Prim Dent Care* 1:9-13, 1994.
138. Rapp EL, Brown CE Jr, Newton CW: An analysis of success and failure of apicoectomies, *J Endod* 17: 508-512, 1991.
139. Esposito M, Hirsch J, Lekholm U et al: Differential diagnosis and treatment strategies for biologic complications and failing oral implants: a review of the literature, *Int J Oral Maxillofac Implants* 14:472-490, 1999.
140. Brisman DL, Brisman AS, Moses MS: Implant failures associated with asymptomatic endodontically treated teeth, *J Am Dent Assoc* 132:191-195, 2001.
141. Shaffer M, Juruaz D, Haggerty P: The effect of periradicular endodontic pathosis on the apical region of adjacent implants, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 86:578-581, 1998.
142. Laney WR, Desjardins RP: Surgical preparation of the partially edentulous patient, *Dent Clin North Am* 17:611-630, 1973.
143. Zarb GA, Bolender CL, Hickey JC et al: Diagnosis and treatment planning for the patient with no teeth remaining. In Zaeb GA, Bolender CL, editors: *Boucher's prosthodontic treatment for edentulous patients*, ed 10, St Louis, 1990, Mosby.
144. House MM: Full denture technique. Notes from study club, September 1950.
145. House MM: The relationship of oral examination to dental diagnosis, *J Prosthet Dent* 8:208-219, 1958.
146. Lytle RB: Soft tissue displacement beneath removable partial and complete dentures, *J Prosthet Dent* 12:34-43, 1962.
147. Lambson GO: Papillary hyperplasia of the palate, *J Prosthet Dent* 16:636-645, 1966.
148. Lytle RB: The management of abused oral tissue in complete denture construction, *J Prosthet Dent* 7:27-42, 1957.
149. Turbyfill WF: The successful mandibular denture implant. Part two, *Dent Econ* 104-106, 1996.
150. Pound E: Preparatory dentures: a protective philosophy, *J Prosthet Dent* 15:5-18, 1965.
151. Smith DE: Interim dentures and treatment dentures, *Dent Clin North Am* 28:253, 1984.

WWW.HIGHDENT.IR

Chapter 13

Diagnostic Casts and Surgical Templates

David P. Sarment, Carl E. Misch

Diagnostic casts are critical to preimplant and treatment prosthodontics. Once the preimplant phase is satisfactory, prosthetically driven implant placement must also accommodate anatomical limitations, and possible adaptations of the original planning may be necessary. This set of requirements can be achieved using precise surgical guides. In recent years, advanced guidance has been developed to achieve predictable, precise placement. This chapter emphasizes important components of the diagnostic phase and then describes how guided placement can overcome anatomical limitations.

DIAGNOSTIC CASTS

The value of diagnostic casts or study models is critical in all of dentistry and especially in oral implantology. Many patients have been partially edentulous for an extended time. The combination of continued bone loss and dentition changes related to missing teeth greatly increases the number of factors that must be considered for oral rehabilitation compared with traditional prosthodontic treatment. The dentist selects the final prosthesis, the number and location of ideal and optional abutment sites, and the occlusal schemes before surgery. Ninety percent of dentists in the United States use a team approach to insert implants and restore implant patients.

Diagnostic casts enable the dentist to evaluate several prosthodontic criteria in the absence of the patient. These casts permit an open discussion of treatment with other practitioners and laboratory technicians for consultation. In addition to all the data provided that are similar to nonimplant treatment evaluation, the casts also assist in implant site selection and angulation requirements during the surgical phase. Surgical templates often are designed from the diagnostic casts or after diagnostic wax-up of the designed prosthesis. One set of casts may be used as a permanent record of pretreatment conditions for dental–legal issues because nonreversible procedures may be performed. The diagnostic casts and pretreatment setups also may be used for presentations to motivate the patient's acceptance of the proposed treatment (Figure 13-1).[1]

Diagnostic casts mounted using an accurate record of centric jaw relationship and maxillomandibular occlusion on a semiadjustable articulator provide much information related to treatment that influences the final prosthodontic treatment plan.[2-4] Important factors include the following:

1. Occlusal centric relation position, including premature occlusal contacts
2. Edentulous ridge relationships to adjacent teeth and opposing arches
3. Position of potential natural abutments, including inclination, rotation, extrusion, spacing, parallelism, and esthetic considerations
4. Tooth morphology, structure of potential abutments, and overall conditions (e.g., wear facets, fractures)
5. Direction of forces in future implant sites
6. Present occlusal scheme, including the presence of balancing or working contacts
7. Edentulous soft tissue angulation, length, width, locations, permucosal esthetic position, muscle attachments, and tuberosities
8. Interarch space

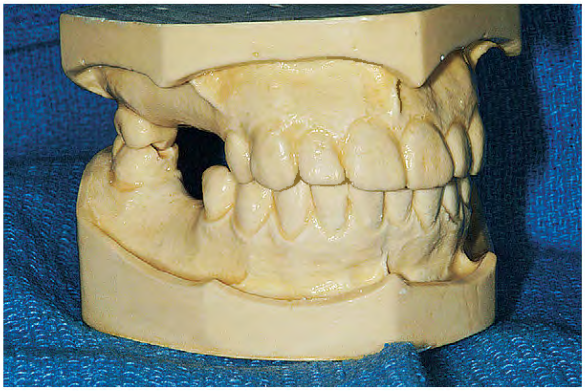


Figure 13-1 Diagnostic casts allow the dental team to evaluate a patient's intraoral condition and plan treatment. However, unmounted casts cannot be used to evaluate the relationship of one arch to another.

WWW.HIGHDENT.IR

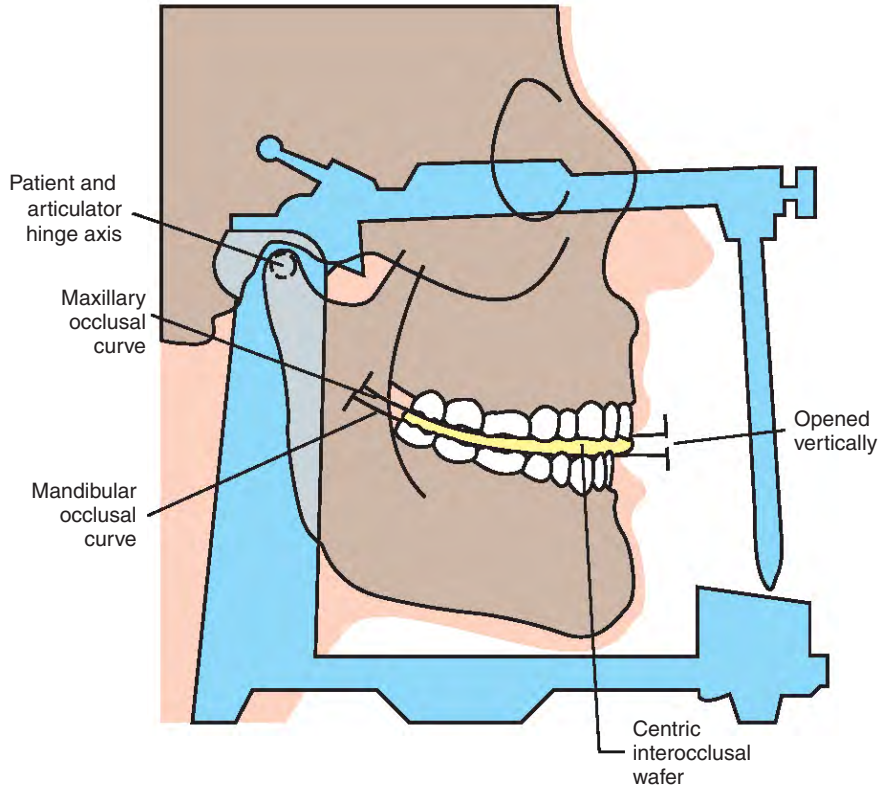


Figure 13-2 Diagnostic casts should be mounted with an open bite registration in centric relation. When the registration material between the teeth is removed, the arch of closure of the mandible should simulate that of the patient. This requires a facebow mounting from the patient to the articulator.

9. Overall occlusal curve of Wilson and curve of Spee
10. Arch relationships
11. Opposing dentition
12. Potential future occlusal schemes
13. Number of missing teeth
14. Arch location of future abutments
15. Arch form and asymmetry

The dentist should evaluate the existing occlusion before implant placement. Partially edentulous patients often have occlusal interferences as a result of tooth migration. The dentist identifies and eliminates deflective contacts before the implant prosthodontic phase. A facebow transfer and centric and eccentric occlusal records should help in mounting the casts on a semiadjustable articulator (Figures 13-2 to 13-5). Occasionally a panographic recording of mandibular movements and a fully adjustable articulator may be indicated. The diagnostic casts are mounted in centric relation occlusion with a wax spacer between the arches. Premature contacts can be detected on removal of the wax spacer. The dentist then may perform an occlusal adjustment on duplicates of the diagnostic casts and replicate them in the mouth.[5]

Research has shown almost 75% of earbow transfers are within 5 mm of the axis of the temporomandibular joint. Almost 25% of individuals have one ear higher than the other, in relation to the occlusal plane. The distance from the hinge axis to the central incisors range from 87 to 120 mm, with more than 80% within 100 to 105 mm.[5]

Kois has developed a unique facebow transfer that corresponds to the facial midline and horizontal plane to the 100-mm distance of the conditional hinge position. This technique simplifies the process of the facebow transfer and ensures the occlusal plane of the teeth is evaluated and fabricated correctly (Figures 13-6 to 13-8) (Kois Dento-Facial Analyzer System, Panadent Corp., Grand Terrace, Calif.).

A considerable prosthetic advantage is present when centric relation occlusion is harmonious with centric relation. Lack of change in the occlusal vertical dimension permits a closed-mouth centric recording during prosthetic reconstruction for the fabrication of the prosthesis, without the need for an accurate hinge axis recording of the condyles or fully adjustable articulators. When incisal edge position of the maxilla is determined, its position usually causes a steeper protrusive or excursive position than the condylar disk assembly. As a result, posterior disocclusion can be established easily. These conditions permit the reconstruction to be fabricated in the laboratory and transferred accurately to the patient.

The occlusion may require complete rehabilitation to eliminate potential unfavorable forces against the implant. Both arches may require prosthodontic treatment to establish the desired occlusal schemes. Parafunctional bruxism with loss of incisal guidance from attrition or an opposing single denture are the most common conditions that mandate more involved opposing dentition modification. The first condition often indicates a need to increase the anterior guidance

WWW.HIGHDENT.IR

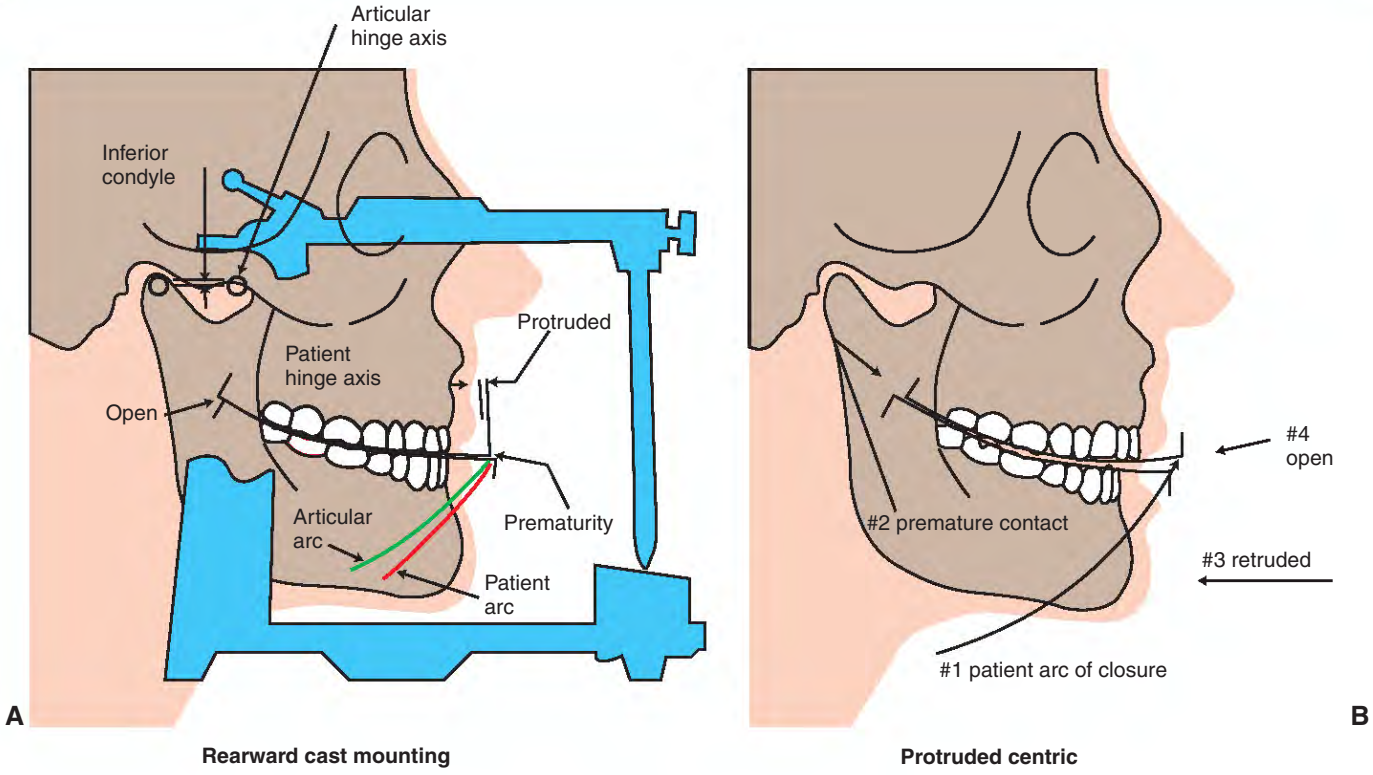


Figure 13-3 **A,** A mounted maxillary cast without a facebow and an open bite mandibular relationship do not allow proper evaluation or reconstruction of the patient's intraoral condition. For example, a maxillary cast mounted to the rear causes premature anterior contacts when the bite registration is removed. **B,** When a reconstruction fabricated on models mounted as in part **A** is placed intraorally, a posterior premature contact is observed and the anterior teeth have no contact. A maxillary cast mounted too high on an articulator also exhibits premature anterior contacts when the open bite registration is removed. When the reconstruction is placed intraorally, a posterior premature contact is observed, similar to the previous scenario. The reverse scenario is observed when the maxillary cast is mounted too far forward or inferior to the hinge axis of the patient.

Figure 13-4 An ear-mounted facebow is acceptable in most situations. However, in almost 25% of patients one ear is higher than the other. In these situations, the cross member still should be made parallel to the horizon.

for posterior disclusion in excursions, whereas the second warrants bilateral balance occlusion.

Duplicate diagnostic casts also may be mounted on an articulator for selective alterations and prewaxing to determine the desired contour, occlusal scheme, and esthetic aspects of the final restoration. The diagnostic wax-up usually is described in detail and performed by the laboratory technician. The specific laboratory communicative processes are thereby begun before actual treatment and may be modified during treatment as required. These considerations include occlusal plane correction, edentulous ridge position and its effect on implant placement, occlusion, esthetic considerations, and interarch distance. The dentist may use the altered diagnostic wax-up casts to provide a guide for provisional restorations and may evaluate them during the provisional stages of reconstruction.

The dentist, in addition to using radiographic surveys, may also use the diagnostic casts to estimate the underlying bone volume. The dentist inserts a needle equipped with an endodontic stop through the patient's mucosa overlying the implant site and measures the mucosal thickness on the crest, facial, and lingual areas. The dentist also can use a bone caliper that contains sharp beaks penetrating soft tissues at a known height. Once the calipers are inserted, bone width can be measured with the calibrated instrument.[6] The edentulous region of the diagnostic cast is cut perpendicular to the ridge. The diagnostic cast cross section then is shaded with a pencil to represent the tissue thickness observed while probing. The remaining cross section of the cast roughly estimates the bony contours under the soft tissue.[6-8]

WWW.HIGHDENT.IR

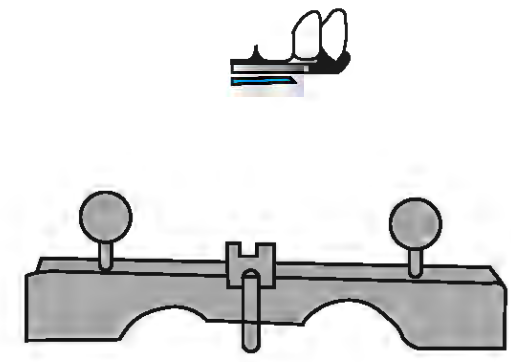


Figure 13-5 When the facebow is not parallel to the horizon with the patient, when placed onto the articulator, the maxillary cast is mounted with an incline that does not represent the patient's intraoral condition.

Figure 13-6 The maxillary incisal edge is 100 mm from the hinge axis in 80% of patients (± 5 mm). *(Courtesy Panadent Corporation, Grand Terrace, Calif.)*

RESTORATIVE DIAGNOSIS VERSUS SURGICAL DIAGNOSIS

Although a prosthetically driven implant placement is best for simplification of abutment selection, ideal force distribution,[9] and long-term success, anatomical limitations may force the surgeon to redirect the implant angulation. When buccal bone loss has occurred, modification of the treatment plan and placement of a bone graft for better implant angulation may be necessary. However, other areas such as lingual concavities cannot

Figure 13-7 Most facebows use the ear canal as a posterior reference point, because the hinge axis is within 5 mm of this point in more than 75% of the patients.

Figure 13-8 The Kois Dento-Facial Analyzer System facebow uses the midline of the face and a parallel reference plane with the incisal edge 100 mm from the hinge axis.

be grafted, and a change in angulations or implant selection is the only option. A typical instance of such limitation is in the mandibular first molar area. A panoramic or periapical radiograph does not show a possible severe lingual concavity, which can be depicted only with careful clinical examination or cross-sectional imaging. Prosthetically driven implant placement would lead only to a risk of lingual perforation. If a concavity is suggested, further radiographs such as a traditional tomogram or a computed tomography scan, together with a diagnostic radiographic template, will reveal the angulation dilemma and allow for clear communication between the restorative dentist and the surgeon. The example in Figure 13-9 represents an actual case in which the diagnostic wax-up was transferred to a scannographic template,[10] but the prosthetic requirement cannot be met surgically—an implant placed in the long axis of the diagnostic tooth would force the surgeon to perforate the lingual cortical bone. In addition, the mandibular nerve is relatively high. Therefore the dentist must make a decision to modify the angulation and later redirect the implant path with an angulated abutment, position

WWW.HIGHDENT.IR

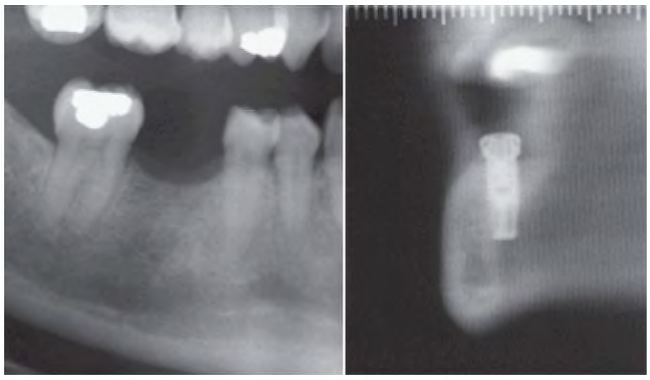


Figure 13-9 An implant following an ideal prosthetic position must have ideal bone volume and angulation. In many situations the bone width or angulation requires slight modification of the implant to avoid perforations of the lateral cortical plates. In this case, the vertical implant insertion caused a perforation through the lingual cortical plate of the posterior mandible.

Figure 13-10 A template for a partially edentulous patient should engage the teeth for stabilization and fixation during surgery. Templates for the anterior regions of the mouth should indicate the facial contour of the restoration.

a shorter implant, or avoid this site all together. A careful clinical examination with the diagnostic template in place would reveal the issue as well, but radiographic visualization is more ideal to ensure surgical success, limit liability, and communicate and discuss planning. Unfortunately, such anatomical limitations often are revealed during surgery, when decision making is more difficult and debate is impossible. Other common locations for similar limitations include the prominent premaxilla or the severely resorbed anterior part of the mandible. As discussed subsequently, precise diagnosis to rule out anatomical limitations can be followed by a thorough plan that can be transferred to the surgical field using surgical guidance.

SURGICAL GUIDES

To establish a logical continuity between diagnosis, prosthetic planning, and surgical phases, use of a transfer device is essential. The restoring dentist fabricates the surgical guide template after the presurgical restorative appointments, once the final prosthetic design, occlusal scheme, and implant location, size, and angulation have been determined. The surgical template dictates the implant body placement that offers the best combination of (1) support for the repetitive forces of occlusion, (2) esthetics, and (3) hygiene requirements. A well-developed plan should be transferred precisely, leaving little decision at the time of surgery.

Several methods of fabricating the surgical template are available. The requirements are more relevant than the options of fabrication. The template should be stable and rigid when in the correct position. If the arch being treated has remaining teeth, the template should fit over or around enough teeth to stabilize it in position (Figure 13-10). When no remaining teeth are present, the template should extend onto unreflected soft tissue regions (i.e., the palate and tuberosities in the maxilla or the retromolar pads in the mandible). In this way, the template may be used after the soft tissues have been reflected from the implant site.

The dentist should determine the ideal angulation for implant insertion on the diagnostic wax-up, and the template should relate this position during surgery. This requires at least two reference points for each implant. For that purpose, the surgical guide must be elevated above the edentulous bone. The distance between two points located respectively on the occlusal surface (central fossa or incisal edge) of the planned abutment crown and the crest of the ridge represents about 8 mm. As a result, these two points of reference can be joined by a line that represents the path of ideal implant insertion. The ideal angulation is perpendicular to the occlusal plane and parallel to the most anterior abutment (natural or implant) joined to the implant.

Other ideal requirements of the surgical template include size, surgical asepsis, transparency, and the ability to revise the template as indicated. The template should not be bulky, be difficult to insert, or obscure surrounding surgical landmarks. The surgical template must not contaminate a surgical field during bone grafts or implant placement; it should also be transparent and allow easy access for the surgeon and the assistant. Consideration of which side of the arch is being treated, where the surgeon and assistant will be seated, and whether the surgeon is right- or left-handed is recommended. In this way, the bony ridge and drills can be more easily visualized when the template is in place, and the assistant can position the irrigation without blocking the surgical view.

The surgical template should relate to the ideal facial contour. Many edentulous ridges have lost facial bone, and the template can assist in determining the amount of augmentation required for implant placement or

WWW.HIGHDENT.IR

support of the lips and face (see Figure 13-10). The surgical template may be used for a bone graft, and later the same template may be used for insertion of implants and again for implant uncovery. A study template facilitates sterilization and use for several procedures.

To construct a surgical guide, modification of the radiographic guide is often possible if an ideal wax-up of the teeth was used as a template for the radiographic guide. Ideal tooth position is already present, and enlargement of the access hole and buccal or lingual opening is achieved easily. When the long axis of the teeth is visible and can be maintained, after verifying bone availability, then enlargement of the long axis channel guarantees accurate implant guidance.[11]

An easy method to fabricate a surgical guide is to use a modification of Preston's clear splint for the diagnosis of tooth contours, tooth position, and occlusal form.[12] The diagnostic wax-up is completed to preview the tooth size, position, contour, and occlusion in the edentulous regions where implants will be inserted. No selective grinding or modification is performed on any teeth that have not been altered before surgery; otherwise, the template will not fit correctly in the mouth. A full-arch, irreversible, hydrocolloid impression is made of the diagnostic wax-up and poured in dental stone. On the duplicate cast of the wax-up teeth, a vacuum acrylic shell (0.060 to 0.080 inch) is pressed and trimmed to fit over the teeth and gingival contours of the buccal aspect of the ridge. If no natural teeth remain, the posterior portion of the template should be maintained and cover the retromolar pads or tuberosities and palate to aid in positioning.

The occlusal surface is trimmed over the ideal and optional implant sites, maintaining the facial and facioocclusal line angle of the surgical template (Figure 13-11). A black line then is drawn on the template with a marker to indicate the center of each implant and the desired angulation. This provides maximum freedom for implant placement yet communicates the ideal tooth position and angulation during surgery.

A surgical guide template with 2-mm holes through the occlusal surface of a denture tooth is too limiting for the surgeon, although it precisely identifies the ideal implant placement. While the template is in position, the crest of the edentulous ridge should be visible to avoid stripping of the facial plate of bone during the osteotomy.

In the edentulous arch the vacuum form may be fabricated from the existing removable prosthesis, if within accepted guidelines. A soft tissue liner then may be added in the tuberosity or retromolar pad regions and other soft tissue areas not involved in surgery. Acrylic resin then is added over the occlusal portion of the template where no implants are planned. The patient then occludes into this index after using petroleum jelly over the opposing teeth. In this manner, the template can be correctly positioned over the edentulous ridge during surgery once the tissue is reflected. Otherwise, a template position set too far facially or off to one side is likely (Figures 13-12 to 13-15).

Figure 13-11 A press form is made over a duplicate cast that replaces the missing teeth. The template is trimmed in the edentulous sites to indicate the teeth position and the lingual contour in the posterior regions and facial contour in the anterior region. Adjacent teeth are used to stabilize the template, and the soft tissue regions are avoided.

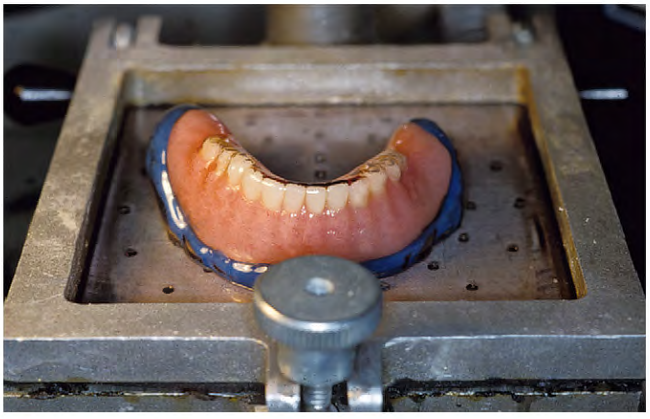


Figure 13-12 The complete edentulous template can be fabricated from a press or vacuum form over the patient's denture.

Figure 13-13 A soft tissue liner is added in the area that will not be reflected during surgery. An index also is made of the opposing dentition.

WWW.HIGHDENT.IR

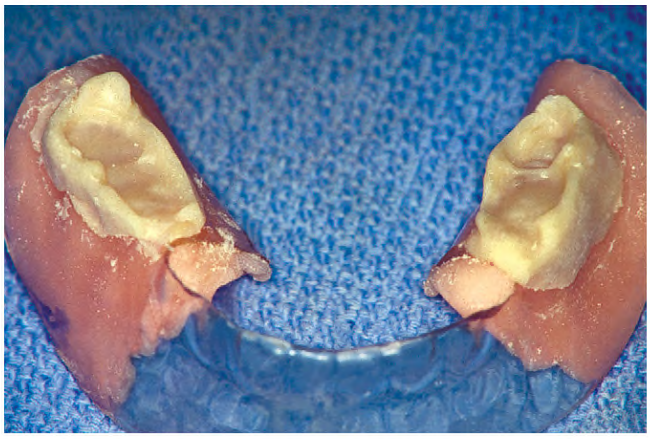


Figure 13-14 The complete edentulous template is trimmed to indicate the facial contour of the restoration in the anterior regions of the mouth. The crestal and lingual contour is completely visible for surgical access.

Figure 13-15 The template is indexed to the opposing arch once the tissue is reflected. The template then is held in position while the patient opens the mouth. The maxillary denture is removed (when present) and the mandibular bone width and angulation are noted before the initial osteotomy. The 2-mm direction indicator then may be inserted and evaluated with the template in position.

A surgical template for the complete edentulous arch also may engage the occlusal aspect of the opposing teeth.[13,14] The following are the fabrication steps on the edentulous cast mounted against the opposing dentition at the proper final occlusal vertical dimension and occlusal relationships:

1. A full wax-up of the missing teeth in the edentulous regions is performed. A hole is prepared through the middle of the central fossa of each future posterior abutment tooth and through the incisal edge position of anterior teeth.
2. On the stone model, each site chosen is drilled to a depth corresponding to the approximate soft tissue thickness measured on a panoramic radiograph (approximately 2 to 3 mm). An orthodontic wire is passed through the teeth and into the holes. This allows each pin of the template to contact bone, once the tissue is reflected during the surgery, without modifying the occlusal vertical dimension and consequently the emergence position of the implant. A small loop is made at the other end of the wire to create a retention form. The wire should approach within 1 to 3 mm of the opposing arch (Figure 13-16).
3. On the antagonist model painted with separator, an acrylic resin template is built on the occlusals that embed the retention loops of the indicator pins. Each pin must be embedded fully in the acrylic at the proper centric and vertical relationships (Figure 13-17).

Figure 13-16 A Laney-Poitras template for the edentulous arch provides complete surgical access. The denture teeth are positioned on the arch. A hole is drilled through each prospective implant site and into the cast 2 to 3 mm. *(Courtesy Yvan Poitras.)*

Once the soft tissue is reflected, the template is positioned over the teeth of the opposing arch. The patient may occlude on the pins, and each one determines the ideal center position of the teeth (Figures 13-18 and 13-19). A pilot drill can be used to mark each implant body position. The angulation of the osteotomy also can be determined with the template. The surgical guide easily determines the implant position, yet the surgeon can have the patient's mouth open and drill into the bone with complete access and vision. This template also may be used with a panoramic radiograph before surgery to determine vertical magnification or horizontal distortion (Figure 13-20). The template also may be used at stage II uncovery to find the position of each implant when soft tissue carving for fixed prosthesis type 1 (FP-1) restorations are indicated, rather than complete reflection of the tissue.

The FP-1 and FP-2 restorations require more ideal implant placement. The ideal implant position allows

WWW.HIGHDENT.IR

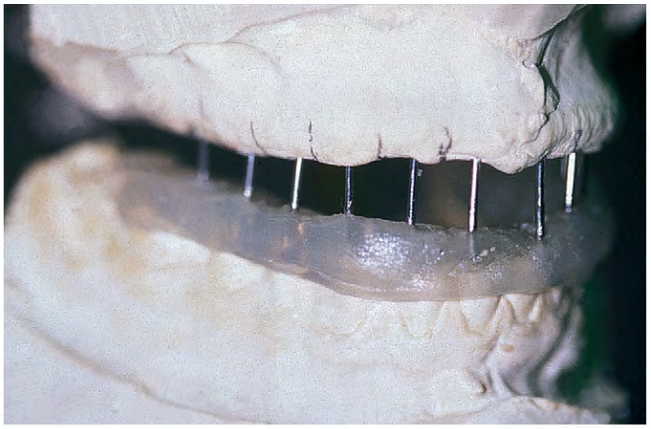


Figure 13-17 The wires representing each potential implant site and angulation are incorporated into an acrylic index of the opposing arch. *(Courtesy Yvan Poitras.)*

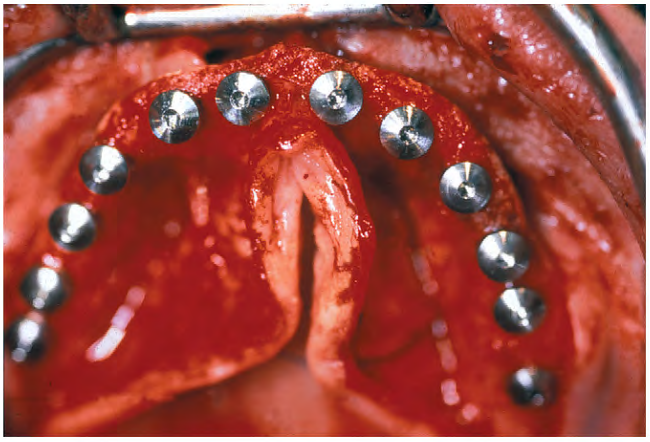


Figure 13-19 Implants are inserted into the maxilla in the appropriate positions. *(Courtesy Yvan Poitras.)*

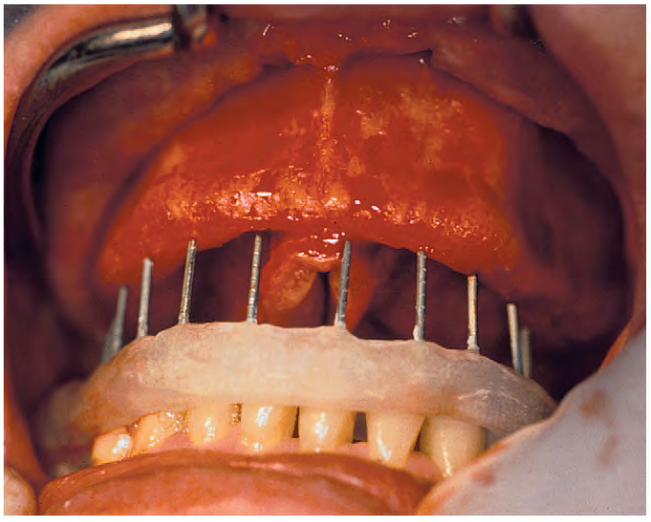


Figure 13-18 Once the tissues are reflected, the template is seated on the opposing arch and indicates the position and angulation of each implant. The patient opens, and the surgeon drills the initial site. *(Courtesy Yvan Poitras.)*

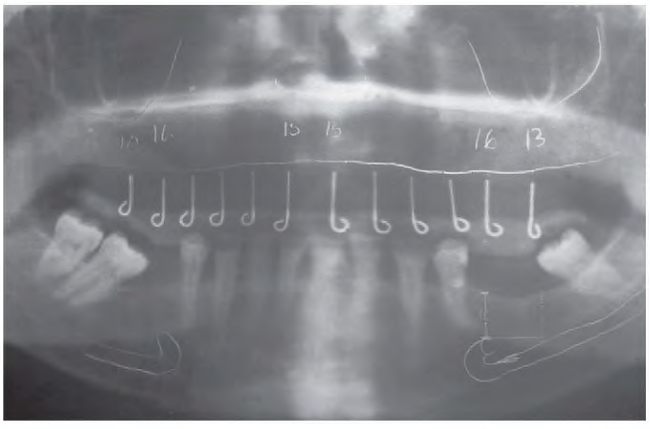



Figure 13-20 The template can be used with a panoramic radiograph. Because the height and mesiodistal position can be measured on the cast, the distortion and magnification at each implant site may be determined. *(Courtesy Yvan Poitras.)*

the placement of a straight abutment directly under the incisal edge of the final crown for a cemented prosthesis. For screw-retained prostheses, the implant should emerge toward the cingulum of the anterior tooth so that the access hole does not affect the esthetics. In an FP-3 restoration, the mesiodistal position of implant abutments may be placed without regard to the actual position of the crowns, because the soft tissue replacement region separates the crowns from the implant abutment.

An implant placed adjacent to a natural tooth should remain 1.5 to 2 mm away from the interproximal cement-enamel junction (CEJ) in esthetic regions, where the contour of the interdental papilla is a determining factor. Therefore the pilot hole should be almost 4 mm away from the natural tooth to place a 4.1-mm-diameter implant at the crest module. This requires at least a 7-mm mesiodistal space. In unesthetic regions, where the interdental papilla is not as critical, an implant placed at least 1.5 mm away from an adjacent tooth minimizes the risk of surgical error and provides easier access for hygiene and long-term maintenance.

A maxillary anterior implant placed for an FP-1 restoration requires the most careful pretreatment planning and precise implant placement. The incisal edge of the final crown, emergence profile, and labial cervical position are related to implant position.

The treatment plan for an implant in the maxillary first premolar position must reflect careful consideration for the angulation of a natural canine when present. The 11-degree average distal inclination and distal curvature of the canine root bring the apex of the root into the first premolar implant area. Therefore the implant should be angled to follow the root of the canine and prevent contact with or perforation of the natural root. A shorter implant often is indicated, especially when a second premolar is also present.

WWW.HIGHDENT.IR

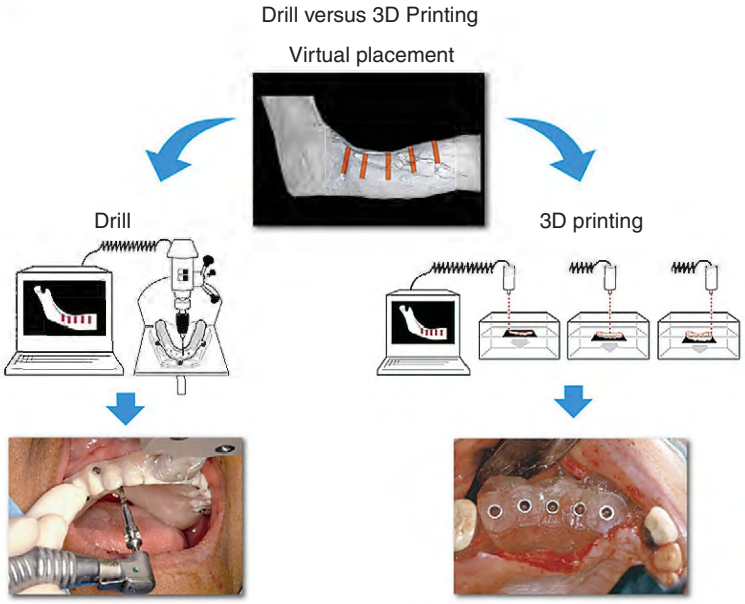


Figure 13-21 Differences between computerized drilling (*Left,* CADImplant, Burlington, Mass.) and CAD/CAM fabrication (*Right,* Materialise, Glen Burnie, Md.) for surgical guide fabrication. *(From Jabero M, Sarment DP: Advanced surgical guidance technology: a review,* Implant Dent *15:135-142, 2006.)*

ADVANCED SURGICAL GUIDANCE

Diagnosis and implant planning for complex cases with anatomical limitations and poor bone quality now can be evaluated using sophisticated radiographic techniques. Although precise positioning has long been recognized as an important goal,[15] transfer of detailed information to the surgical phase has been at best a difficult task. In fact, a single-tooth implant also requires placement accuracy, even when diagnosis is simple. A surgical guide should be used to guarantee precise placement. Studies show that modification of the radiographic guide to a surgical guide allows for a precision of less than 1 mm at the implant apex and a good control of the angulation.[11,16] However, these preclinical and clinical studies assume no modification of angulation compared with the ideal prosthetic alignment. As discussed previously, the angulation often must be modified to account for anatomical limitations. Until recently, no method existed to transfer an ideal implant position precisely to a surgical guide, especially if the long axis of the diagnostic teeth could not be used.

To refine surgical guidance, innovative developments in software technology and manufacturing techniques have been applied to fabricate highly accurate templates. More recently, peroperative surgical navigation assistance has been introduced with potential for wider acceptance in the near future (Figure 13-21).[17] These technologies allow for more accurate implant positioning by guaranteeing transfer of the implant planning to the surgical field and by forcing the surgical drills into a steady position. These technologies also open venues to new surgical techniques, such as flapless osteotomies, while improving operative timing.

Advanced surgical guides require computed tomography (CT) scanning as a prerequisite for analysis, because of the superior precision of CT compared with all other radiographic techniques.[18-20] These guides also necessitate a software-supported rendering to improve planning by using three-dimensional (3D) visualization,[21] as demonstrated by Jacobs et al.,[22,23] who reported that dentists using two-dimensional (2D) cross sections make numerous modifications during the surgical phase of treatment, whereas the addition of a three-dimensional representation improves the correlation between planned and actual placement. Jacobs et al. also found little correlation between foreseen anatomical complications and the presence of these complications at the time of surgery when only flat projections were used. Verstreken et al.[24] also found that planning was improved in regard to position, considerations of biomechanical spread, and esthetics. More importantly, software rendering that includes CT data and implant planning can be exported later to computer-aided design (CAD) software. For these applications, scannographic templates with precise visualization of diagnostic teeth are strongly recommended to visualize the prosthetic plan together with the osseous topography.[25] In addition, when advanced surgical guidance is considered, several techniques require specifically designed scannographic templates, as described subsequently. Therefore dentists who intend to use one of these methods first must be aware of the entire sequence before the diagnostic stage.

Surgical guidance can be classified in two categories. The first category includes the use of computer-aided manufacturing of guides, using virtual planning of implant positions. Guides are delivered to surgeons

WWW.HIGHDENT.IR

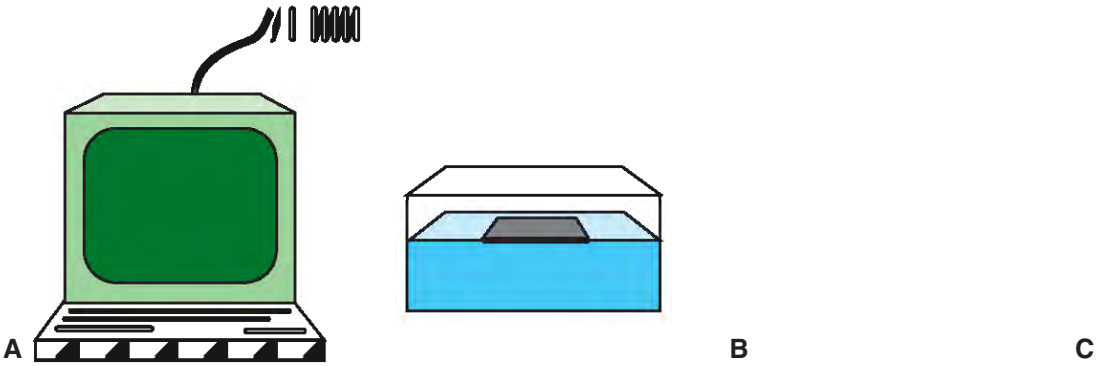


Figure 13-22 Stereolithographic fabrication of surgical guides. After scanning and planning, files are submitted to the manufacturing facility. **A,** A computer-driven laser cures a thin layer of liquid polymer. **B,** A moving table is lowered, another layer is applied, and **(C)** the process is repeated until completion of the surgical guide.

before the procedure, and no modification is possible during surgery. The second category is the use of navigation techniques. There is no guidance of the drill, but software provides real-time feedback to the surgeon in order to compare execution with planning. Therefore modifications are possible during surgery if necessary. The following sections provide a more detailed description of these modalities.

Computer-Assisted Design and Manufacturing of Surgical Guides

Multiple engineering techniques such as laser sintering[26] are available to fabricate 3D models. One of the systems (Surgiguide, CSI Materialise, Glen Burnie, Md.) uses a computer-aided manufacturing (CAM) process called *stereolithography* (Figure 13-22).[27] This rapid prototyping process has been used largely in the manufacturing industry to obtain 3D models.[27,28] A layer of liquid polymer is deposited and cured by a computer-driven laser. Additional layers or sections are stacked and polymerized until a final model is generated. For medical or dental application, the data source is a CT scan file. The accuracy of anatomical models generated by this method depends on the quality of the CT scanner and the thresholding method (the computer process that determines what is bone and what is soft tissue), but studies have shown a dimensional stability in the range of 0.6 mm.[29] Stereolithographic anatomical models have been used to anticipate reconstructive surgeries.[30,31] Santler et al.[32] and Heissler et al.[33] took advantage of 3D models to better prepare for large reconstructions on more than 300 trauma and cancer cases, whereas Runte et al.[34] used optical images as a source to build models of soft tissue contours and fabricate facial prostheses. Use of an anatomical model also has been suggested for diagnosis before sinus elevations[35] and for preparation of subperiosteal[36,37] or ramus implants.[38] Choi et al.[29] evaluated the accuracy of these acrylic models by taking linear measurements of multiple identical sets and found that it was in the range of 0.5 mm.[28] Erickson et al.[39] surveyed surgeons who used stereolithographic methods for diagnosis of surgical reconstruction and fabrication of custom implants. They found that a majority of surgeons had changed their surgical approach once they evaluated the model and thus reduced surgical time for the procedure.

For fabrication of dental implant surgical guides, the dentist's plan is used to design the guides, and CT files are used to prepare the guides to be borne on hard or soft tissue. Software programs are capable of maximizing stability and implant retention by detecting the best insertion path while avoiding undercuts within the bone (Figure 13-23). Designs also include irrigation holes, sufficient surface areas to maintain figure pressure while performing osteotomies, and other specific features such as buccal extensions if a transversal retention screw is desired. In addition, serial templates are fabricated to accommodate increasing drill diameters. Once designs are completed, the guides are processed with the stereolithographic method, and stainless steel tubes are later pressed into place.[40,41] The dentist receives the anatomical model and surgical guides by mail and can observe the anatomy before proceeding with surgery (Figure 13-24, *A*).

Because the topography is obtained from the CT scan data, this process is best suited for osseous-supported templates with wide edentulous areas (Figure 13-24, *B*). When teeth are present, high radiopacity creates blurry outlines, and stereolithographic rests cannot be contoured precisely. This is even more critical when metal-containing restorations such as crowns are present, and severe scattering masks the contours. Similarly, soft tissue surfaces are usually difficult to visualize on CT scan images. However, visualization of ridges is possible by providing a scannographic template containing a

WWW.HIGHDENT.IR

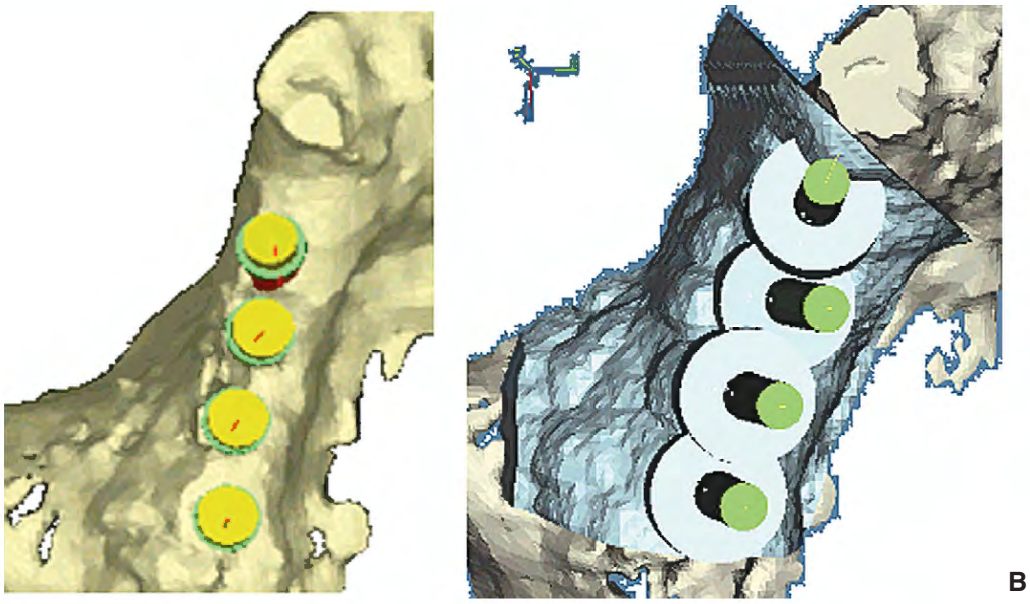


Figure 13-23 **A,** Planning has been forwarded for design and manufacturing of surgical guides. **B,** The surgical guides first are designed virtually to maximize stability and retention and then are sent for processing using the method previously described.

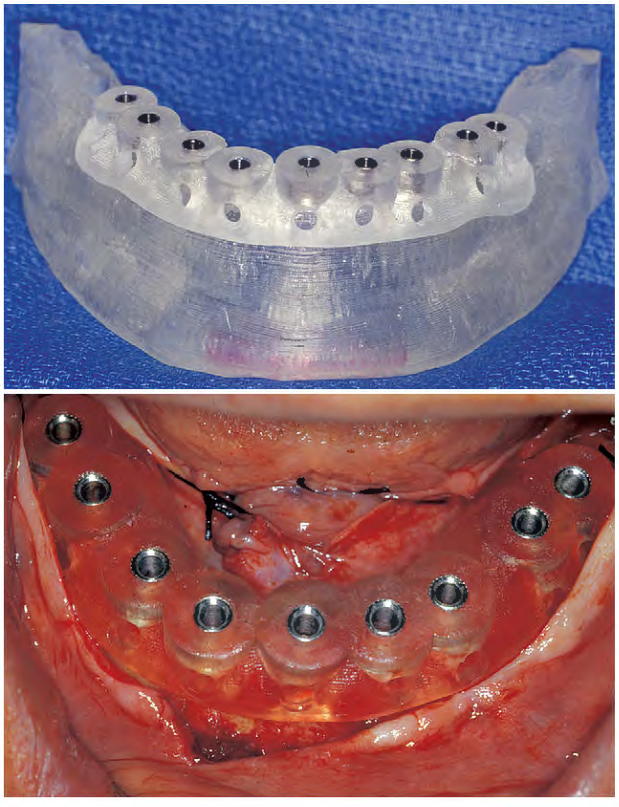


Figure 13-24 **A,** The dentist receives the anatomical model and surgical guides by mail and can observe the anatomy before proceeding with surgery. **B,** Because the topography is obtained from the computed tomography scan data, this process is best suited for osseous-supported templates with wide edentulous areas.

radiopaque base.[42] This approach is most appropriate for edentulous cases: a duplicate of the denture is processed with a radiopaque medium so that the base is visible, representing soft tissues. The surgical guides therefore can be soft tissue supported and incisions may be avoided, keeping in mind that mucosal stability is inferior to bone. Surgical guides also can be fabricated for various maxillofacial implants, such as pterygoid or zygomatic implants, where access and visualization is difficult. Finally, similar surgical guidance is applicable to the medical field, such as fusion of vertebrae in which precise osteotomies are necessary to avoid vital structures.[43,44]

A similar method uses 3D printers to fabricate guides, instead of stereolithography. A medical-grade polymer is also used to fabricate guides layer by layer. Such printers are more readily available, with the potential to be installed in dental laboratories or practices (Figure 13-25). The approach taken by i-dent Imaging (Fort Lauderdale, Fla.) is that software imports CT data, provides virtual implant planning, and is able to export a computer file to a 3D printer. The process requires CT scanning of the patient with a scannographic guide in place as well as scanning of the appliance. This guide, made by the dental technician, must contain gutta-percha landmarks simply inserted in the acrylic at the end of the guide preparation. The purpose of the double scanning is to match patient data and an accurate scannographic guide, which is then used to forward information to the 3D printer. Three-dimensional printing has the advantage of being financially accessible to dental laboratories. Technicians can become integral to the implant team by being involved in planning, guide preparation, and prosthesis fabrication after implant placement.

In contrast to systems described previously, alternative methods (Compu-Guide, Implant Logic Systems, Cedarhurst, N.Y.; and CADImplant, Burlington, Mass.) use drilling of guides.[45,46] The method necessitates the

WWW.HIGHDENT.IR

Figure 13-25 Use of three-dimensional printing for guide fabrication. After double scanning of patient and guide, data were imported and implant planning was performed using software. Note that the virtual surgical guide is immediately visible for verification before fabrication. Three-dimensional printing is performed locally, and the guide is used during flapless.

incorporation of metal markers at specific locations in the scannographic guide that therefore must be provided by the manufacturer. Once the guides are returned and used during CT scanning, the dentist creates a surgical plan using a software (SimPlant, CSI-Materialise, Leuven, Belgium) in a traditional manner. The dentist then returns the plan, model, and scannographic template for conversion of the template into the surgical guide. To achieve transfer of the plan, the model is set onto a computer-controlled milling machine, which matches the fiducial landmarks to their CT-scanned images. The plan then is transferred to the guide using the computer-driven drill press (Figure 13-26). Metal guide sleeves then are added for an ideal guidance of surgical drills. In this system, only one template is fabricated, but drill guides with incremental diameters are inserted sequentially into receiving master cylinders. Because of the ability of the guide to rest on natural teeth, this method can be applied to small edentulous spans. If increased stability is needed when few or no teeth are present, a tacking system can be added away from implant sites. Finally, the surgical guide can be converted into a provisional restoration for immediate loading cases. A similar technique has been described by Fortin et al.,[47,48] who placed reference tubes in the scannographic guide. After scanning and planning, the template also is positioned on a computer-driven drilling table to modify the appliance into a precise surgical guide.

Although these advanced methods aim at improving surgical guidance, accuracy rarely has been measured objectively. Unpublished data from the manufacturers and clinical experience (Figure 13-27) support the hypothesis that planning and actual placement are related more closely in terms of horizontal positioning (mesiodistal and buccolingual) and implant angulation. Similar results were found using a preclinical study design to compare traditional surgical guides modified from scannographic templates with SurgiGuides (Materialise). Coronal osteotomy was improved from an average of 1.5 to 0.9 mm, and the apical position at 10 mm was improved from 2.1 to 1 mm, and this improvement was due to a better angulation (from 8 to 4.5 degrees). An important note is that average enhancements also were accompanied by overall decrease in standard deviations, revealing minimization of surgical errors.[49] In another publication, van Steenberghe et al.[50] evaluated placement of 45-mm-long zygoma implants on human cadavers. They reported less than 3 degrees of deviation, and no more than a 2.7-mm discrepancy at the apex. Fortin et al.[47] found that the transfer error was less than 0.2 mm and 1.1 degrees. More recently, Di Giacomo et al. reported on a case series in which patients received CT scanning before and after implant placement, using SurgiGuides. They found that

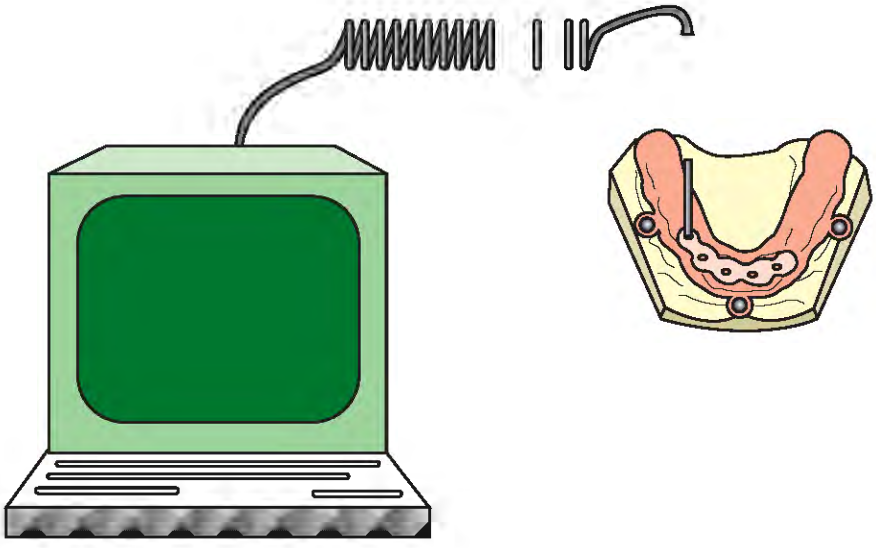


Figure 13-26 CAD/CAM computer-driven drill press system. Similar to the method shown in Figure 13-22, planning takes place on a computer. The scannographic template is repositioned on the model and registered using fiducial markers. A computer drives the table angulations to reproduce planning and convert the template into an accurate surgical guide.

WWW.HIGHDENT.IR

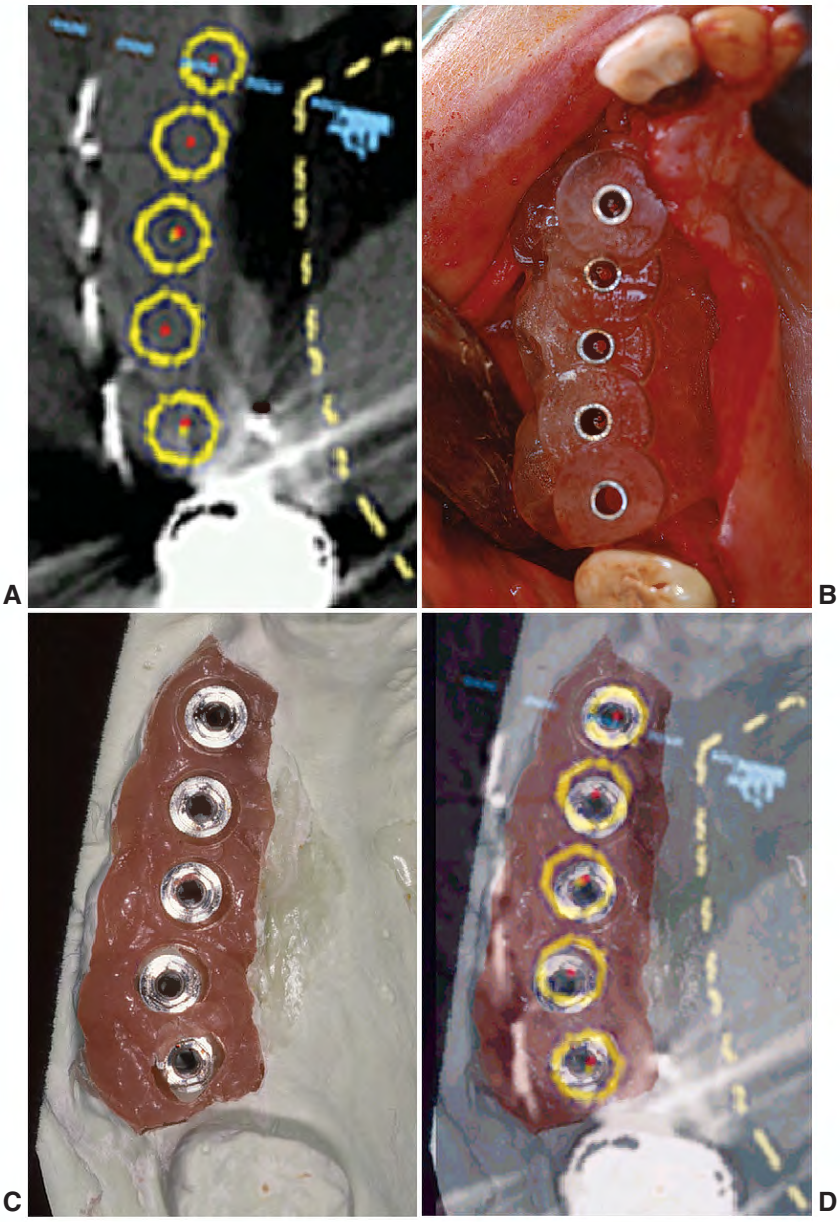


Figure 13-27 **A,** Virtual implant placement was forwarded for fabrication of surgical guides. **B,** The first surgical guide containing 2-mm metal guides is followed by identical guides with wider diameters. **C,** For comparison of planning and execution, the implant level impression was overlaid by virtual implant plan **A** to create the image **(D),** demonstrating a clinical accuracy.

placement is on average 7 degrees from planning, with the implant shoulder being a mean 1.45 mm from planning.[51]

Improvements of these surgical guides are ongoing, in particular for control of the coronoapical positioning. In a recent development, Tardieu and Vrielinck[52] proposed a modification to the first method described. These new templates (SAFE, Materialise) are secured onto the bone surface using tacking screws. Only one template is used and cylinders are replaced. This protocol also includes a limited sequence of specially designed surgical burs with stops, as well as implant carriers allowing for control of the insertion depth. Therefore another potential benefit to CAD/CAM surgical templates could be the elimination of sequential osteotomy drills, because their accuracy precludes the need for angulation correction offered by multiple enlarging osteotomies. Further research is warranted because single, larger drill osteotomies potentially can overheat the osseous surface.[53]

Surgical Navigation

Image-guided surgery, first developed for medical applications, recently has been introduced to implant dentistry.[54] The close surgical field and difficult accesses drove the need for computer guidance in medical applications such as neurosurgery, and dental implant placement may benefit from these technologies by offering a peroperative 3D assessment of osteotomies. Similar to the techniques described before, a CT scan is necessary. The scannographic guide includes fiducial markers for cross-referencing jaw positions with the CT scan, and virtual implant planning is performed

WWW.HIGHDENT.IR

using software. For surgery the handpiece is equipped with a 3D positioning device, such as electromagnetic digitizers[55] or light-emitting diodes.[56] Extraoral markers attached to the surgical guide are also necessary so that the computer can analyze the positions of the jaw and the handpiece relative to each other. Continuous reevaluation of locations and matching to the CT scan data during surgery allow for visualization of osteotomies and comparison of planning and drilling. Some computer systems are equipped with audible or visual warnings when osteotomies deviate from planning or when a vital structure is about to be entered. Research is showing that this approach, although complex, can yield favorable results perhaps, in the vicinity of 0.5 mm.[57,58]

One example of such application is marketed under the name *IGI* (Image Guided Implantology, DenX, Jerusalem, Israel). To perform a surgery using this system, a CT scan must be processed in the presence of a scannographic template attached to a manufactured arch registration device. The CT scan data is then transferred to custom software, and implant planning takes place using virtual implants. Before surgery, the registration device is repositioned, and a preliminary matching process is performed by locating radioopaque markers. Both the registration device attached to the patient and the handpiece possess light-emitting diodes (LEDs) that can be located in space via infrared cameras mounted above the dental chair. Once this registration has taken place, the surgery may begin using the reference body to locate the patient's jaw and the diode-equipped handpiece to locate the surgeon's movements. A computer screen displays real-time positioning of the drill in the mesiodistal, buccolingual, and coronoapical planes (Figure 13-28). For edentulous patients, temporary implants must be inserted because of the necessity to obtain a stable reference guide containing the markers.

Another similar system is the VirtualScope (Areall, Neuilly-sur-Seine, France), which features an advanced registration method allowing for elimination of positioning markers during CT scanning (Figure 13-29). The rationale for not using fiducial radiopaque landmarks in the scannographic template is that although matching of the markers and their CT image is possible, a small distortion at their level may become a severe mismatch at a distance from them. For instance, an implant's coronal position may remain accurate, whereas its apex is 2 to 3 mm from its intended location. Instead, this system offers a real-time 3D capture of the arch via an ultrasound probe. Mapping of the clinical image can be matched to the CT-scanned data and updated continuously, thus creating an accurate registration independent from a guide.[59] Flags are attached to the handpiece and the ultrasound probe, and their position in space is located by two sets of cameras above the surgeon. The reformatted CT scan, the implant plan, and the actual drill position are viewed at all times through glasses worn by the surgeon. In future versions, a semirigid arm also will connect to the handpiece so that position and angle will be guided by the computer while the surgeon applies the pressure. In addition, a similar registration approach will be used for periapical radiographs. The film holder position will be recorded in relation to an edentulous space. Multiple films will be taken at various angles, and the computer will be able to create a 3D view of the site, thus eliminating the need for a tomogram or a CT scan for small-span implant restorations. Interestingly, this

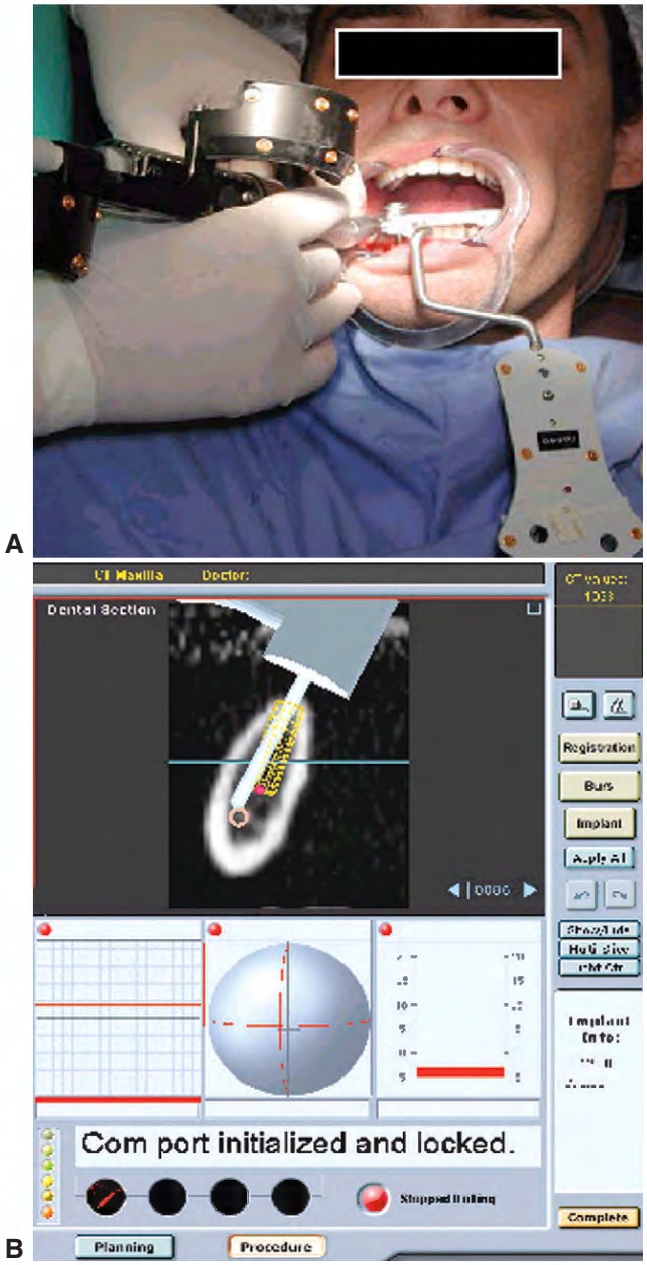


Figure 13-28 Image-guided surgery. **A,** The surgical handpiece and patient arch are registered in real time, allowing for **(B)** visualization and comparison of drilling with preplanning. Significant deviations are avoided by using an alarm or automatic handpiece shutdown.

WWW.HIGHDENT.IR

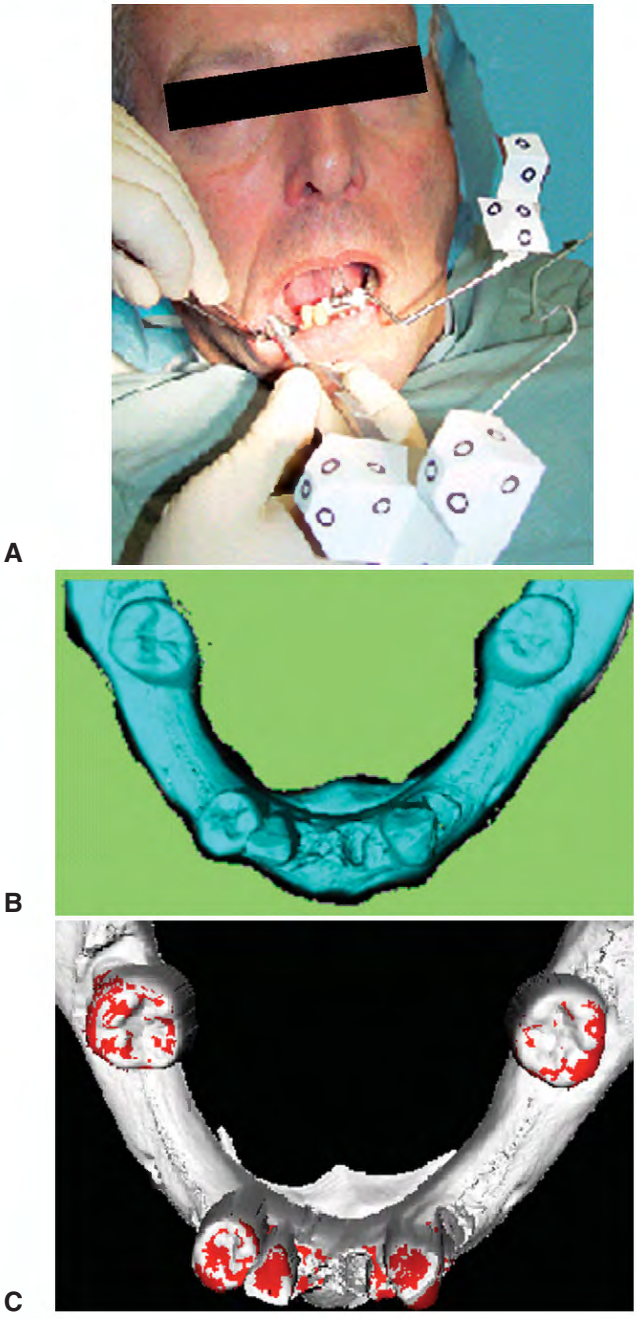


Figure 13-29 Image-guided surgery **(A)** using a real-time updated ultrasonic image of the patient arch **(B). C,** Other functions are similar to the method described.

system is being developed for dental applications but is also of interest for medical applications, such as for the ear, nose, and throat or neurosurgery.

Other similar methodologies are available, such as the system reported by Wanschitz et al.,[60] in which the handpiece and mandible are positioned using LEDs. In the latest version, the surgeon wears an optical tracking system that allows simultaneous visualization of the surgical field and the plan in the glasses.[61]

These promising computer-guided technologies are currently under development, although most are marketed already. Manufacturers claim a precision within less than 1 mm at the osteotomy entrance and high control of angulations.[43,62] Using the LED localization approach and a tracking system, Wanschitz et al.[60] performed an in vitro test of accuracy and found it to be within less than 1 mm. Further studies are necessary, but clinical application is beginning and will likely grow rapidly once costs are reduced.

SUMMARY

Surgical models and guidance have acquired a new dimension with the integration of CAD/CAM technology and computer-guided surgery. With the advent of low-radiation cone-beam computed tomography, now available in small practical units, access to CT data is simplified and, in turn, advanced diagnosis and fabrication of CAD/CAM surgical guides becomes more realistic. Precision has been improved and uncertainty and surgical time have been reduced, thus addressing complex rehabilitation with greater confidence. In addition, predictable positioning allows for better prosthetic outcome by simplifying abutment selection and avoiding complex laboratory fabrication when misalignment must be corrected. In addition, novel techniques are emerging that may enable the preparation of the final prosthesis before implant placement. Precise guidance is crucial to such complex reconstruction so that minimal adaptation is performed after surgery. Future technical improvements likely will allow dentists to access these technologies while controlling costs, reducing surgical time, and minimizing restorative steps.

References

1. Thompson EO: Constructing and using diagnostic models, *Dent Clin North Am* 1:67-84, 1963.
2. Laney WR: Critical aspects of removable partial denture service. In Goldman HM, editor: *Current therapy in dentistry,* St Louis, 1968, Mosby.
3. Misch CE, editor: *Patient evaluation booklet,* Plattsburgh, NY, 1994, Medigraphics.
4. Desjardins RP: Tissue integrated prostheses for edentulous patients with normal and abnormal jaw relationships, *J Prosthet Dent* 59:180-187, 1988.
5. Dawson PE: *Evaluation, diagnosis and treatment of occlusal problems,* St Louis, 1989, Mosby.
6. Wilson DJ: Ridge mapping for determination of alveolar ridge width, *Int J Oral Maxillofac Implants* 4:41-44, 1989.
7. Gershkoff A, Goldberg NI: The implant lower denture, *Dent Dig* 55:490-493, 1969.
8. Spiekerman HS: Special diagnostic methods for implant patients. In Spiekeman HS, editor: *Implantology,* Stuttgart, Germany, 1995, Thieme.
9. Quirynen M, Naert I, van Steenberghe D: Fixture design and overload influence marginal bone loss and fixture success in the Brånemark system, *Clin Oral Implants Res* 3:104-111, 1992.
10. Basten CH, Kois JC: The use of barium sulfate for implant templates, *J Prosthet Dent* 76:451-454, 1996.

WWW.HIGHDENT.IR
مرجع تخصصی دندانسازان و دندانپزشکان

11. Naitoh M, Ariji E, Okumura S et al: Can implants be correctly angulated based on surgical templates used for osseointegrated dental implants? *Clin Oral Implants Res* 11:409-414, 2000.
12. Preston JD: A systemic approach to the control of esthetic form, *J Prosthet Dent* 35:393-402, 1976.
13. Poitras Y: *The complete edentulous maxilla surgical template,* Monte Carlo, Monaco, 1996, International Congress of Oral Implantologists.
14. Laney WR, Tolman DE: The Mayo clinic experience with tissue integrated prostheses. In Albrektsson T, Zarb GA, editors: *Brånemark osseointegrated implant,* Chicago, 1989, Quintessence.
15. Engelman MJ, Sorensen JA, Moy P: Optimum placement of osseointegrated implants, *J Prosthet Dent* 59:467-473, 1988.
16. Besimo CE, Lambrecht JT, Guindy JS: Accuracy of implant treatment planning utilizing template-guided reformatted computed tomography, *Dentomaxillofac Radiol* 29:46-51, 2000.
17. Jabero M, Sarment DP: Advanced surgical guidance technology: a review, *Implant Dent* 15:135-142, 2006.
18. Todd AD, Gher ME, Quintero G et al: Interpretation of linear and computed tomograms in the assessment of implant recipient sites, *J Periodontol* 64:1243-1249, 1993.
19. Butterfield KJ, Dagenais M, Clokie C: Linear tomography's clinical accuracy and validity for presurgical dental implant analysis, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 84:203-209, 1997.
20. White SC, Heslop EW, Hollender LG et al: Parameters of radiologic care: an official report of the American Academy of Oral and Maxillofacial Radiology, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 91:498-511, 2001.
21. Seipel S, Wagner IV, Koch S et al: Oral implant treatment planning in a virtual reality environment, *Comput Methods Programs Biomed* 57:95-103, 1998.
22. Jacobs R, Adriansens A, Naert I et al: Predictability of reformatted computed tomography for pre-operative planning of endosseous implants, *Dentomaxillofac Radiol* 28:37-41, 1999.
23. Jacobs R, Adriansens A, Verstreken K et al: Predictability of a three-dimensional planning system for oral implant surgery, *Dentomaxillofac Radiol* 28:105-111, 1999.
24. Verstreken K, Van Cleynenbreugel J, Martens K et al: An image-guided planning system for endosseous oral implants, *IEEE Trans Med Imaging* 17:842-852, 1998.
25. Israelson H, Plemons JM, Watkins P et al: Barium-coated surgical stents and computer-assisted tomography in the preoperative assessment of dental implant patients, *Int J Periodontics Restorative Dent* 12:52-61, 1992.
26. Abbott JR, Netherway DJ, Wingate PG et al: Computer generated mandibular model: surgical role, *Aust Dent J* 43:373-378, 1998.
27. Jacobs P: *Stereolithography and other RP&M technologies,* Dearborn, Mich, 1996, Society of Manufacturing Engineers.
28. Hull C: Apparatus for production of three-dimensional objects by stereolithography, (U.S. patent No. 4,575,330), March 11, 1986.
29. Choi JY, Choi JH, Kim NK et al: Analysis of errors in medical rapid prototyping models, *Int J Oral Maxillofac Surg* 31:23-32, 2002.
30. Joffe JM, McDermott PJ, Linney AD et al: Computer-generated titanium cranioplasty: report of a new technique for repairing skull defects, *Br J Neurosurg* 6:343-350, 1992.
31. Stoler A: Helical CT scanning for CAD/CAM subperiosteal implant construction, *J Oral Implantol* 22:247-257, 1996.
32. Santler G, Karcher H, Ruda C: Indications and limitations of three-dimensional models in cranio-maxillofacial surgery, *J Craniomaxillofac Surg* 26:11-16, 1998.
33. Heissler E, Fischer FS, Bolouri S et al: Custom-made cast titanium implants produced with CAD/CAM for the reconstruction of cranium defects, *Int J Oral Maxillofac Surg* 27:334-338, 1998.
34. Runte C, Dirksen D, Delere H et al: Optical data acquisition for computer-assisted design of facial prostheses, *Int J Prosthodont* 15:129-132, 2002.
35. Bianchi SD, Lojacono A, Nevins M et al: The use of replicate resin models in the treatment of maxillary sinus augmentation patients, *Int J Periodontics Restorative Dent* 17:348-357, 1997.
36. McAllister ML: Application of stereolithography to subperiosteal implant manufacture, *J Oral Implantol* 24:89-92, 1998.
37. Cranin AN, Klein M, Ley JP et al: An in vitro comparison of the computerized tomography/CAD-CAM and direct bone impression techniques for subperiosteal implant model generation, *J Oral Implantol* 24:74-79, 1998.
38. Amet EM: Computerized tomography with CT models for contemporary ramus frame implant planning and construction, *J Oral Implantol* 24:152-158, 1998.
39. Erickson DM, Chance D, Schmitt S et al: An opinion survey of reported benefits from the use of stereolithographic models, *J Oral Maxillofac Surg* 57:1040-1043, 1999.
40. Fortin T, Champleboux G, Lormee J et al: Precise dental implant placement in bone using surgical guides in conjunction with medical imaging techniques, *J Oral Implantol* 26:300-303, 2000.
41. Sarment DP, Al-Shammari K, Kazor CE: Stereolythographic surgical templates for placement of dental implants in complex cases, *Int J Periodontics Restorative Dent* 23: 287-295, 2003.
42. Sarment DP, Misch CE: Scannographic templates for novel pre-implant planning methods, *Int Mag Oral Implantol* 1:16-22, 2002.
43. Van Brussel K, Lauweryns P, Van der Sloten J et al: Medical image based drill guides can achieve sub-millimeter accuracy in pedicle screw placement: a clinical study. In *Fifth Conference of the European Society for Engineering and Medicine,* Barcelona, Spain, 1999.
44. Goffin J, Van Brussel K, Martens K, et al: Three-dimensional computed tomography-based, personalized drill guide for posterior cervical stabilization at C1-C2, *Spine* 26: 1343-1347, 2001.
45. Klein M, Cranin AN, Sirakian A: A computerized tomography (CT) scan appliance for optimal presurgical and preprosthetic planning of the implant patient, *Pract Periodontics Aesthet Dent* 5:33-39, 1993.
46. Klein M, Abrams M: Computer-guided surgery utilizing a computer-milled surgical template, *Pract Proced Aesthet Dent* 13:165-169, 2001.
47. Fortin T, Coudert JL, Champleboux G et al: Computer-assisted dental implant surgery using computed tomography, *J Image Guid Surg* 1:53-58, 1995.
48. Fortin T, Champleboux G, Bianchi S et al: Precision of transfer of preoperative planning for oral implants based on cone-beam CT-scan images through a robotic drilling machine, *Clin Oral Implants Res* 13:651-656, 2002.
49. Sarment DP, Sukovic P, Clinthorne N: Accuracy of implant placement with a stereolythographic surgical guide, *Int J Oral Maxillofac Implants* 18:571-577, 2003.

WWW.HIGHDENT.IR

50. van Steenberghe D, Malevez C, Van Cleynenbreugel J et al: Accuracy of drilling guides for transfer from three-dimensional CT-based planning to placement of zygoma implants in human cadavers, *Clin Oral Implants Res* 14:131-136, 2003.
51. Di Giacomo GA, Cury PR, de Araujo NS et al: Clinical application of stereolithographic surgical guides for implant placement: preliminary results, *J Periodontol* 76:503-7, 2005.
52. Tardieu PB, Vrielinck L: Implantologie assistée par ordinateur: le programme SimPlant/Surgicase et le SAFE System, *Implant* 9:15-28, 2003.
53. Eriksson AR, Albrektsson T: Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit, *J Prosthet Dent* 50:101-107, 1983.
54. Casap N, Tarazi E, Wexler A, et al: Intraoperative computerized navigation for flapless implant surgery and immediate loading in the edentulous mandible, *Int J Oral Maxillofac Implants* 20:92-98, 2005.
55. Solar P, Grampp S, Gsellmann B: A computer-assisted navigation for oral implant surgery using 3D-CT reconstruction and real time video-projection. In Farman AG, editor: *Computer assisted radiology—CAR*, Amsterdam, 1996, Elsevier.
56. Shapira L: Image-guided implantology: real-time guidance of dental implant surgery in the operative field using CT-scan image. In Vannier MW, Inamura K, Farman AG et al, editors: *Proceedings of the 16th International Congress, Computer-Assisted Radiology and Surgery*, Paris, France, 2002.
57. Gaggl A, Schultes G: Assessment of accuracy of navigated implant placement in the maxilla, *Int J Oral Maxillofac Implants* 17:263-270, 2002.
58. Casap N, Wexler A, Persky N et al: Navigation surgery for dental implants: Assessment of accuracy of the image guided implantology system, *Oral Maxillofac Surg* 62: 116-119, 2004.
59. Granger S, Pennec X, Roche A: Rigid point-surface registration using oriented points and an EM variant of ICP for computer guided oral implantology. In Viergever MA, Niessen WG, editors: *Proceedings of the 4th International Conference, Medical Image Computing and Computer-Assisted Intervention—Miccai 2001*, Utrecht, Netherlands, 2001.
60. Wanschitz F, Birkfellner W, Watzinger F et al: Evaluation of accuracy of computer-aided intraoperative positioning of endosseous oral implants in the edentulous mandible, *Clin Oral Implants Res* 13:59-64, 2002.
61. Wanschitz F, Birkfellner W, Figl M et al: Computer-enhanced stereoscopic vision in a head-mounted display for oral implant surgery, *Clin Oral Implants Res* 13:610-616, 2001.
62. Birkfellner W, Solar P, Gahleitner A et al: In-vitro assessment of a registration protocol for image guided implant dentistry, *Clin Oral Implants Res* 12:69-78, 2001.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 14

The Edentulous Mandible: An Organized Approach to Implant-Supported Overdentures

Carl E. Misch

The dental profession and the public are more aware of the problems associated with a complete mandibular denture than any other dental prosthesis. The placement of implants enhances the support, retention, and stability of an overdenture. As a result, patients are very willing to accept a treatment plan for a mandibular implant overdenture. There is greater flexibility in implant position or prosthesis fabrication with a mandibular implant overdenture, and, as a result, it is also an ideal treatment modality to begin a learning curve in implant dentistry. Therefore one of the most beneficial treatments rendered to patients is also one of the best introductions for a dentist into the discipline of implant dentistry. In comparison, maxillary implants for overdentures are not used as often and are also more difficult to place and restore.

The concept of mandibular implant–supported overdentures has been used for many years. Successful reports were published originally with mandibular subperiostal implants or with immediately loaded and stabilized root form implants in the anterior mandible.[1,2] An increased awareness from the profession and patients allows a variety of clinical situations, bone densities, biomechanics, and patients' desires to restore an ever-growing number of patients with implant-supported overdentures.[3-32] As the data gathered on this prosthetic modality grow, overdentures are gaining even greater acceptance and justifying their place in the armamentarium of the implant dentist.

From a bone volume conservation standpoint, complete edentulous patients should be treated with enough implants to support overdentures in the maxilla and mandible. The continued bone loss after tooth loss and associated compromises in esthetics, function, and health make all edentulous patients implant candidates. The average denture patient does not see a dentist regularly. In fact, 10 or more years usually separate dental appointments of edentulous patients. The more often a patient wears a denture, the greater the bone loss. Yet 80% of denture patients wear their dentures day and night, which accelerates the bone loss.

Because of the long hiatus between dental visits, the amount of resorption from initial denture delivery to the next professional interaction already has caused the destruction of the original alveolar process. The bone loss that occurs during the first year after tooth loss is 10 times greater than in following years. In the case of multiple extractions, this often means a 4-mm vertical bone loss within the first 6 months. As the bony ridge resorbs, the muscle attachments become level with the edentulous ridge.[33-35]

Rather than waiting until the patient has lost most of the residual bone, the dentist should inform and emphasize to the patient the benefits of implants and why they should be inserted before the bone is lost. Therefore the profession should treat bone loss from extraction in a similar fashion as bone loss from periodontal disease. Rather than waiting until the bone is resorbed or the patient complains, the dental professional should educate the patient about the bone loss process caused not only by periodontal disease, but also by the lack of stimulation and its consequences of bone resorption, and explain how implants are available to treat the condition. Therefore most completely edentulous patients should be informed of the necessity of dental implants to maintain bone volume, function, masticatory muscle activity, esthetics, and psychologic health. Ideally, patients who have unsalvageable teeth should be given the option to include implants to support the future prosthesis. The traditional complete denture may be presented as a temporary measure to provide cosmetic and oral function during implant treatment.

The next progression in the implant philosophy is to convert all mandibular implant and soft tissue–supported

WWW.HIGHDENT.IR

restorations to a completely implant-supported prosthesis. The majority of mandibular overdentures are supported by two implants anterior to the foraminae and soft tissue support in the posterior regions (Figure 14-1). Yet posterior bone loss occurs four times faster than anterior bone loss. In the completely edentulous patient, the eventual paresthesia and mandibular body fractures are primarily from posterior bone loss. The anterior implants allow improved anterior bone maintenance, and the prosthesis benefits from improved function, retention, and stability. However, the lack of posterior support in two- and three-implant overdentures may cause accelerated posterior bone loss. To the contrary, studies by Wright et al.[30] and Reddy et al.[36] found prostheses completely supported by implants in the edentulous mandible actually may increase the posterior bone volume (even though posterior implants are not inserted). As a result, complete implant-supported restorations should be the restoration of choice.

Financial considerations have been identified as the reason for the selection of a limited treatment, which may consist of two or three implants to support the overdenture.[37-39] These restorations may be used as transitional devices until the patient can afford to upgrade the restoration. When a partially edentulous patient cannot afford to replace four missing first molars, the dentist often will replace one at a time over many years. Likewise, the dental implant team can insert one or two additional implants every few years until finally a complete implant-supported prosthesis is delivered.

The ultimate goal may be designed in the beginning of treatment and may take many years to complete. However, the advantage of developing a treatment plan for long-term health, rather than short-term gain, is beneficial to the patient. As such, if finances are not an issue, the dentist should design a prosthesis that is completely supported, retained, and stabilized by implants. If cost is a factor, a transitional implant-retained restoration greatly improves the performance of a mandibular denture. Then the dentist may establish a strategy for the next one or two steps for the final restoration.

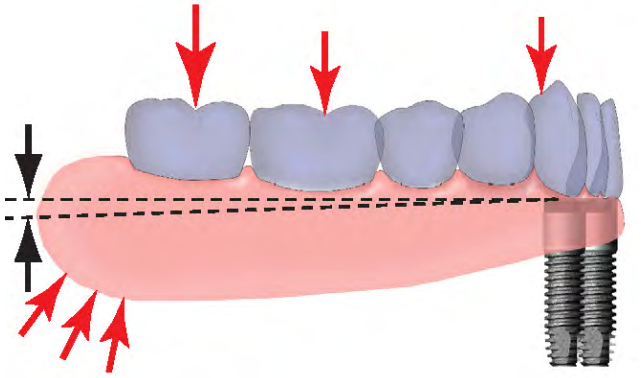


Figure 14-1 The majority of mandibular overdentures performed by dentists are with two implants, regardless of factors such as the remaining bone, the opposing arch, and patient complaints. Although this treatment is an improvement compared with a denture, the posterior bone loss continues and the anterior implants may experience greater problems than when additional implants are used.

REVIEW OF THE LITERATURE

In 1986 a multicenter study reported on 1739 implants placed in the mandibular symphysis of 484 patients. The implants were loaded immediately and restored with bars and overdentures with clips as retention. The overall success rate was 94%.[2] Engquist et al.[3] reported a 6% to 7% implant failure for mandibular implant–supported overdentures and a 19% to 35% failure for maxillary implant overdentures. Hyperplasia below the bar occurred in 25% of the patients. Prosthetic complications were not reported. Jemt et al.[4] reported on a 5-year prospective, multicenter study on 30 maxillae (117 Brånemark implants) and 103 mandibles with 393 implants. Survival rates in the mandible were 94.5% for implants and 100% for prostheses; in the maxilla, the survival rates were 72.4% for implants and 77.9% for prostheses. Higher failure rates in the maxilla were related directly to poor density and quantity of bone with a characteristic cluster failure pattern.

Wismeijer et al.[5] reported on 64 patients with 218 titanium plasma-sprayed implants, with a 97% survival with overdentures in a 6.5-year evaluation. Naert et al.[14] found 100% implant success at 5 years for overdentures with different anchorage systems. In Belgium, Naert at al. reported on 207 consecutively treated patients with 449 Brånemark implants and Dolder-bar overdentures. In this report, the cumulative implant failure rate was 3% at the 10-year benchmark.[9,10] In this long-term report, prosthetic complications related to overdenture relining and loose retention occurred 10% of the time. More care was needed if ball attachments were used rather than a Dolder bar.[9] In a 10-year study reporting on attachment methods, balls provided the greatest retention and magnets always scored the lowest.[10] Similarly, Hutton et al.[11] reported 97% survival rates for mandibular overdentures.

Misch[12] reported less than 1% implant failure and no prosthesis failure over a 7-year period with 147 patients when using the organized treatment options and prosthetic guidelines presented in this chapter. Kline et al.[13] reported on 266 implant-supported overdentures for 51 patients, with an implant survival rate of 99.6% and a prosthesis survival rate of 100%. Mericke-Stern et al.[16] reported 95% implant survival with two implant overdentures. In a randomized clinical report, Awad et al.[6] compared satisfaction and function in complete dentures (48 patients) versus two implant-supported overdentures (IODs) in 56 patients. There was significantly higher satisfaction, comfort, and chewing ability in the IOD group. A similar study in a senior population yielded similar results.[7] Thomason et al.,[8] in

WWW.HIGHDENT.IR

the United Kingdom, reported a 36% higher satisfaction for the implant IOD patients than the complete denture wearers in the criteria of comfort, stability, and chewing. In Canada, Attard and Zarb[40] followed IOD wearers for 20 years with a success rate of 84% and 87% for prosthesis and implants, respectively. In a 10-year study of IODs in Israel, with 285 implants and 69 implant over dentures, Schwartz-Arad et al. reported implant survival was 96.1% with higher success rates in the mandible.[41] Many reports have been published over the last two decades that conclude that implant-supported overdentures represent a valid beneficial option for denture wearers.[3-41] It should be noted that the majority of reports are for implant overdentures supported by only two implants.

ADVANTAGES OF IMPLANT OVERDENTURES

Traditional overdentures must rely on the remaining teeth to support the prosthesis. The location of these natural abutments is highly variable, and they often comprise past bone loss associated with periodontal disease. For a mandibular implant–supported overdenture, the implants may be placed in planned, specific sites, and their number may be determined by the restoring doctor and patient. In addition, the overdenture implant abutments are healthy and rigid and provide an excellent support system. As a result, the related benefits and risks of each treatment option may be predetermined.

The patient gains several advantages with an implant-supported prosthesis (Box 14-1). Minimal bone resorption of the anterior residual ridge occurs with the placement of implants. After the extraction of mandibular teeth, an average of 4-mm vertical bone loss occurs during the first year after treatment. This bone loss continues over the next 25 years, with the mandible experiencing a fourfold greater vertical bone loss than the maxilla.[35] The bone under an overdenture may resorb as little as 0.6 mm vertically over 5 years, and long-term resorption may remain at less than 0.05 mm per year.[4,40,42,43]

Box 14-1 Implant Overdenture Advantages

- Minimum anterior bone loss; prevents bone loss
- Improved esthetics
- Improved stability (reduces or eliminates prosthesis movement)
- Improved occlusion (reproducible centric relation occlusion)
- Decrease in soft tissue abrasions
- Improved chewing efficiency and force
- Increased occlusal efficiency
- Improved retention
- Improved support
- Improved speech
- Reduced prosthesis size (eliminates palate, flanges)
- Improved maxillofacial prostheses

Bone loss dictates the appearance of the inferior third of the face. A maxillary overdenture often provides improved support for the lips and soft tissues of the face compared with a fixed prosthesis because the prosthesis contour does not have to accommodate daily hygiene requirements. Denture teeth also provide an esthetic replacement for the natural dentition, which is more challenging for the technician to recreate with porcelain-fused-to-metal restorations. For the laboratory to create pink interdental papilla, as well as replace the soft tissue drape, is easier with an overdenture compared with porcelain-metal fixed restorations. In addition, the teeth can be positioned in the most esthetic position, without any restriction as to the relationship to the atrophied crest, because stability now is provided by the implant and does not depend on tooth position on the crest of the ridge.

Soft tissue abrasions and accelerated bone loss are more symptomatic of horizontal movement of the prosthesis under lateral forces. An implant-supported overdenture may limit lateral movements and direct more longitudinal forces. A mandibular denture may move 10 mm during function. Under these conditions, specific occlusal contacts and the control of masticatory forces are nearly impossible. An implant overdenture provides stability of the prosthesis, and the patient is able consistently to reproduce a determined centric occlusion.[44]

A study of chewing efficiency compared wearers of complete dentures with wearers of implant-supported overdentures. The complete denture group needed 1.5 to 3.6 times the number of chewing strokes compared with the overdenture group.[45] The chewing efficiency with an implant overdenture is improved by 20% compared with a traditional complete denture.[6,7,46,47]

Higher bite forces have been documented for mandibular overdentures on implants. The maximum occlusal force of a patient with dentures may improve 300% with an implant-supported prosthesis.[48] Mericke-Stern[49] and Mericke-Stern et al.[50] compared mastication between root overdentures and implant overdentures. The former was more discriminative, whereas the latter developed slightly harder chewing strokes and tended to masticate more vertically. Jemt et al.[51] showed a decrease in occlusal force when the bar connecting implants was removed, which they attributed to the loss of support, stability, and retention. If enough implant support is provided, the resulting prosthesis may be completely supported, retained, and stabilized by the implant (removable prosthesis type 4 [RP-4]).

The complete mandibular denture often moves during mandibular jaw movements during function

WWW.HIGHDENT.IR

and speech. The contraction of the mentalis, buccinator, or mylohyoid muscles may lift the denture off the soft tissue. As a consequence, the teeth may touch during speech and elicit clicking noises. The retentive implant overdenture remains in place during mandibular movement. The tongue and perioral musculature may resume a more normal position because they are not required to limit mandibular denture movement.

The implant overdenture may reduce the amount of soft tissue coverage and extension of the prosthesis. This is especially important for new denture wearers, patients with tori or exostoses, or patients with low gagging thresholds. Also, the existence of a labial flange in a conventional denture may result in exaggerated facial contours for the patient with recent extractions. Implant-supported prostheses do not require labial extensions or extended soft tissue coverage.

Soft and hard tissue defects from tumor excision or trauma do not permit the successful rehabilitation of the patient with traditional denture support. Hemimandibulectomy patients and other maxillofacial patients also may be restored successfully with an implant overdenture.[52,53]

The implant overdenture also provides many practical advantages over the implant-supported complete fixed partial denture (Box 14-2). Fewer implants are required, because soft tissue areas may provide additional support. Regions of inadequate bone for implant placement therefore may be eliminated from the treatment plan, rather than necessitating bone grafts or placing implants with poorer prognosis. Abutments do not require a specific mesiodistal placement because the prosthesis completely covers the implant abutments.

The esthetics for many edentulous patients with moderate to advanced bone loss are improved with an overdenture compared with a fixed restoration. Soft tissue support for facial appearance often is required for an implant patient because of advanced bone loss, especially in the maxilla. Interdental papilla and tooth size are easier to reproduce or control with an overdenture. Denture teeth easily reproduce contours and esthetics compared with time-consuming and technician-sensitive porcelain-metal fixed restorations. The labial flange may be designed for optimal appearance, not daily hygiene.

Hygiene conditions and home and professional care are improved with an overdenture compared with a fixed prosthesis. Peri-implant probing is diagnostic and easier around a bar than a fixed prosthesis because the crown often prevents straight line access along the abutment to the crest of the bone. The overdenture may be extended over the abutments to prevent food entrapment during function. Speech is not compromised because the denture may extend onto the soft tissues in the maxilla and prevent air and saliva from escaping.

An overdenture may be removed at bedtime to reduce the noxious effect of nocturnal parafunction, which increases stresses on the implant support system. The overdenture may provide stress relief between the superstructure and prosthesis, and the soft tissue may share a portion of the occlusal load. The prosthesis is usually easier to repair than a fixed restoration. Reduced laboratory fees and fewer implants allow the restoration of patients at reduced costs compared with a fixed prosthesis. In addition, long-term denture patients do not appear to have a psychologic problem associated with the ability to remove their implant prostheses.[54-62]

When cost is a factor, two implant-retained IODs may improve the patient's condition at a lower overall treatment cost than a fixed implant–supported prosthesis.[42,44,63] A survey by Carlsson et al.[64] in 10 countries indicated a wide range of treatment options. The proportion of implant overdentures selection versus fixed implant dentures was highest in the Netherlands (93%) and lowest in Sweden and Greece (12%). Cost was cited as the number one determining factor in the choice.

The primary indications for a mandibular implant overdenture are problems often found with lower dentures, such as lack of retention or stability, decrease in function, difficulties in speech, tissue sensitivity, and soft tissue abrasions. If an edentulous patient is willing to remain with a removable prosthesis, an overdenture is often the treatment of choice. In addition, if cost is a problem for the patient, the overdenture may serve as a transitional device until additional implants may be inserted and restored.

Box 14-2 Implant Overdenture Advantages versus Fixed Prosthesis

- Fewer implants (RP-5)
  - Less bone graft
  - Less specific placement
- Improved esthetics
  - Labial flange
  - Denture teeth
  - Soft tissue drape
- Soft tissue considerations
  - Improved peri-implant probing (follow-up)
  - Hygiene
- Reduced stress
  - Nocturnal parafunction (remove prosthesis, at night)
  - Stress-relief attachment
- Lower cost and laboratory cost (RP-5)
  - Fewer implants (RP-5)
  - Less bone grafting (RP-5)
  - Easy repair
  - Laboratory cost decrease (RP-5)
- Transitional device until fixed restoration guidelines are complete

WWW.HIGHDENT.IR

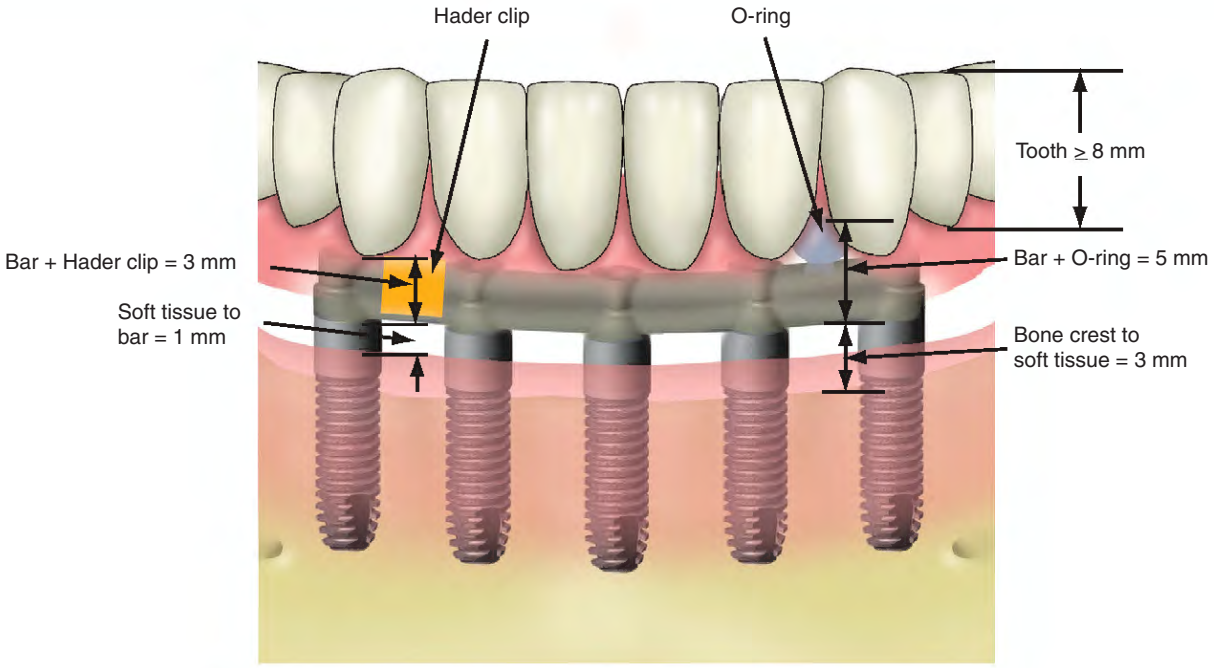


Figure 14-2 The mandibular overdenture requires at least 12 mm between the soft tissue and the occlusal plane to provide sufficient space (15 mm from bone level to occlusal plane) for the bar, attachments, and teeth.

DISADVANTAGES OF IMPLANT OVERDENTURES

The primary disadvantage of a mandibular overdenture is related to the patient's desire, primarily because they do not want to be able to remove the prosthesis. A fixed prosthesis often is perceived as an actual body part of the patient, and if a patient's primary request is not to remove the prosthesis, an implant-supported overdenture would not satisfy the psychological need of this patient.

The mandibular overdenture treatment plan requires more than 12 mm of space between crestal bone and the occlusal plane (Figure 14-2). When sufficient crown height space is lacking and the prosthesis is more prone to component fatigue and fracture, an overdenture is more difficult to fabricate than a porcelain-to-metal fixed prosthesis. The 12-mm minimum crown height space provides adequate bulk of acrylic to resist fracture, space to set denture teeth without modification, and room for attachments, bars, soft tissue, and hygiene. In the mandible, the soft tissue is often 1 to 3 mm thick above the bone, so the occlusal plane to soft tissue should be at least 9 to 11 mm in height. An osteoplasty to increase crown height space before implant placement or a fixed restoration is often indicated when abundant bone height and width are present.

Mandibular overdenture wearers often incur greater long-term expenses than those with fixed restorations. Attachments such as O-rings or clips wear and must be replaced regularly. Replacements appear more frequent during the first year, but remain a necessary maintenance step.[17,20,34,65-72] Denture teeth wear faster on an implant overdenture than with a traditional denture because bite force and masticatory dynamics are improved. A new overdenture often is required at 5- to 7-year increments because of denture tooth wear and soft tissue support changes. Therefore patient education of the long-term maintenance requirement should be outlined at the onset of implant therapy.[73]

A primary concern for RP-5 overdentures compared with RP-4 or fixed restorations is the continued bone loss in the posterior regions. The posterior bone resorbs faster than the anterior bone, and implant prostheses with posterior soft tissue support may accelerate posterior bone resorption two to three times faster than in a complete denture wearer.[73-75] By contrast, patients wearing fixed implant–supported prostheses showed no bone loss and usual occurrences of bone apposition.[76,77] Therefore the short-term benefit of decreased cost may be offset by the accelerated bone loss that is a primary consideration, especially in the younger edentulous patient. As previously discussed, all implant overdentures would benefit if they were completely implant supported, and the recommendation is to consider a RP-5 prosthesis as an interim device designed to enhance the function of the patient. These prostheses should not be considered as an end result for all patients. Instead, a regular evaluation of patients' performance paired with patient education should enable the transformation to a RP-4 restoration. In addition, reports indicate that mandibular implant overdentures may cause a combination-like syndrome, with increased looseness, subjective loss of fit, and midline fracture of the upper

WWW.HIGHDENT.IR

Box 14-3 Overdenture Disadvantages

- Psychological (need for nonremovable teeth)
- Greater abutment crown height space required
- Long-term maintenance
  - Attachments (change)
  - Relines (RP-5)
  - New prosthesis every 7 years
- Continued posterior bone loss
- Food impaction
- Movement (RP-5)

denture.[78-82] Although not yet established as a cause-and-effect situation, the condition also appears to be controlled by the choice of proper occlusal scheme.[80]

A side effect of a mandibular overdenture is food impaction. The flanges of the prosthesis do not extend to the floor of the mouth in the rest position (to eliminate sore spots caused by elevation of the floor of the mouth during swallowing). However, during eating, food particles migrate and become impacted under the prosthesis during swallowing. A similar condition is found with a traditional denture. However, because a lower denture "floats" during function, the food more readily goes under and out, whereas the implant overdenture traps the food debris against the implants, bars, and attachments (Box 14-3).

OVERDENTURE MOVEMENT

The most common complications found with mandibular implant overdentures are related to a lack of understanding of retention, support, and stability of the prosthesis. When a fixed restoration is fabricated on implants, it is rigid, and cantilevers or offset loads are clearly identified. Rarely will a practitioner place a full-arch fixed restoration on three implants, especially with excessive cantilevers because of implant positioning. However, three anterior implants with a connecting bar may support a fixed overdenture system solely because of attachment design or placement. The restoring doctor thinks the overdenture needs less support, but does not realize that an overdenture that does not move during function is actually a fixed restoration. Therefore an overdenture with no prosthesis movement should be supported by implants in number, position, and design similar to fixed restorations.

Many precision attachments with varying ranges of motion are used in implant overdentures. The motion may occur in one to six directions or planes: occlusal, gingival, facial, lingual, mesial, and distal. A type 2 attachment moves in two planes, a type 4 attachment in four planes. However, the resulting overdenture movement may be completely different from the one provided by independent attachments and may vary from one to six directions depending on the position and number of attachments, even when using the same type. Therefore attachment and prosthesis movement are independent from each other and should be evaluated as such.

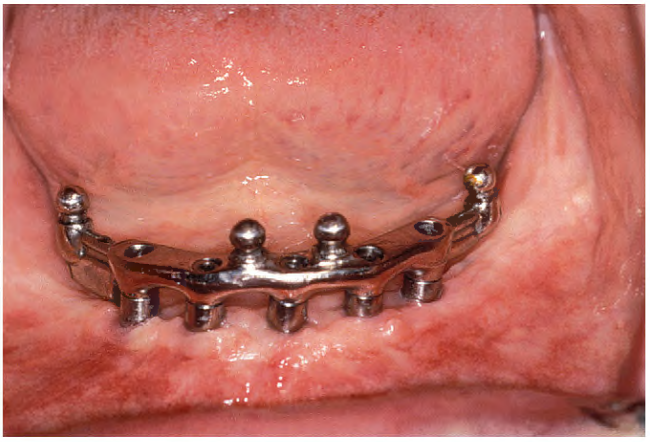


Figure 14-3 Prosthesis movement for overdentures is often different than "attachment" movement categories. In this illustration, O-rings (a Class 6 attachment movement) and Hader clip (a Class 2 attachment movement) support a rigid overdenture. The prosthesis movement is PM-0.

Classification of Prosthesis Movement

The classification system proposed by the author in 1985 evaluates the direction of movement of the implant-supported prosthesis, not the overall range of motion for the individual attachment; therefore the amount of prosthesis movement (PM) is the primary concern. An overdenture is by definition removable, but in function the prosthesis may not move. If the prosthesis does not have movement during function, it is designated PM-0 and requires implant support similar to a fixed prosthesis. A prosthesis with a hinge motion is PM-2, and a prosthesis with an apical and hinge motion is PM-3. A PM-4 allows movement in four directions, and the PM-6 has all ranges of prosthesis movement.

The dentist evaluates the prosthesis movement when seating the restoration. If the prosthesis is rigid when in place but can be removed, the prosthesis movement is labeled PM-0, regardless of the attachments used. For example, O-rings may provide motion in six different directions. But if four O-rings are placed along a complete arch bar, and the prosthesis rests on the bar, the situation may result in a PM-0 restoration (Figure 14-3). A hingelike prosthesis movement permits movement in two planes (PM-2) and most often uses a hingelike attachment. For example, the Dolder bar and clip without a spacer or Hader bar and clip are the most commonly used hingelike attachment. A Dolder bar is egg shaped in cross section, and a Hader bar is round. A clip attachment may rotate directly on the Dolder bar. A Hader bar is more flexible because round

WWW.HIGHDENT.IR

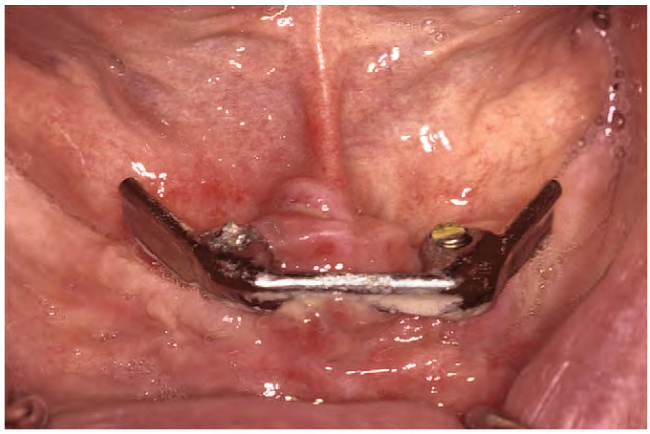


Figure 14-4 A Hader bar clip is a Class 2 attachment system. However, when the clips are placed parallel or at an angle to the desired prosthesis movement, the prosthesis is rigid. In this case, two implants are not enough to support a PM-0. Screw loosening, bone loss, and implant failure resulted.

bars flex to the power of 4 related to the distance and other bar shapes flex to the power of 3. As a result, an apron often is added to the tissue side of the Hader bar to limit metal flexure, which might contribute to unretained abutments or bar fracture. It should be noted that for these systems to function efficiently, the hinge attachment needs to be perpendicular to the axis of prosthesis rotation, so the prosthesis movement also will be in two planes (i.e., PM-2). If the Hader or Dolder bar is at an angle or parallel to the direction of desired rotation, the prosthesis is more rigid and may resemble a PM-0 system (Figure 14-4).

A cross section of the Hader bar and clip system reveals that the apron, by which the system gains strength compared with a round bar design, also limits the amplitude of rotation of the clip (and prosthesis) around the fulcrum to 20 degrees, thus transforming the prosthesis and bar into a more rigid assembly. Therefore the Hader bar and clip system may be used for a PM-2 when posterior ridge shapes are favorable and soft tissue is firm enough to limit prosthesis rotation.

A Hader bar-clip system is an ideal low-profile attachment for an RP-4 prosthesis, with PM-0. Usually, these clips are placed on the bar in different planes around the arch.

A Dolder bar and spacer is desirable when greater amplitude of movement is needed to account for poorer ridge anatomy. An attachment system that permits apical movement and a hinge motion is called a type 3 system. For example, a Dolder bar with spacer and clip is designed for three planes of movement.

A spacer can be added to the occlusal surface of the egg-shaped Dolder bar during the processing of the clip attachment. The retention clip does not engage the top of the bar when the spacer is removed. As a result, the removable prosthesis may move down toward the clip and then rotate around the bar. The bar and clip also must be perpendicular to the direction of prosthesis rotation to allow a PM-3 movement. When no clip is included to enhance prosthesis rotation, a more rigid system is created (i.e., PM-2).

A PM-4 restoration rarely is created with an overdenture system. The type 4 overdenture attachment systems allow a range of motion (i.e., mesial, distal, facial, and lingual directions), and the restoration may be removed. Magnets are the most common implant attachment system, allowing a PM-4. An advantage is that they exert practically no lateral force on the implants. However, the implants usually must be independent from each other for this range of motion to occur. If a superstructure connects the implants, the range of prosthesis motion decreases. Independent magnets provide excellent retention, but often poor stability of the prosthesis. Although great improvements in their size and retention characteristics have been achieved since their introduction into dentistry, magnets still often are plagued with long-term problems of corrosion.

An O-ring or extracoronal resilient attachment may correspond to six directions of motion. However, the implants usually need to remain independent (not connected with a bar) to permit a PM-6 range of movement. A superstructure bar limits the prosthesis movement, depending on its design. A greater interarch space is required and greater forces are applied against the support system of an O-ring because the vertical component of rotation for this attachment extends about 5 mm above the implant.

MANDIBULAR IMPLANT SITE SELECTION

Anterior retention and stability for an overdenture offer several advantages. The greatest available height of bone is located in the anterior mandible, between the mental foraminae. This region also usually presents optimal density of bone for implant support. In addition, overdentures with posterior movement (RP-5) gain better acceptance than removable restorations with anterior movement. An axiom in removable partial denture design is to gain rigid prosthetic support in the anterior region. When the prosthesis has poor anterior and good posterior support, it rocks back and forth. This rocking action applies torque to the abutments and increases stresses on the overdenture components and bone-implant interface. Therefore anterior forces should be resisted by implants or bars, whereas posterior forces may be directed on a soft tissue area such as the mandibular buccal shelf. Therefore the implant overdenture treatment options presented are designed for anterior implant placement.

The available bone in the anterior mandible is divided into five equal columns of bone serving as potential implant sites, labeled A, B, C, D, and E, starting from the patient's right side (Figure 14-5).[83,84] Regardless of

WWW.HIGHDENT.IR

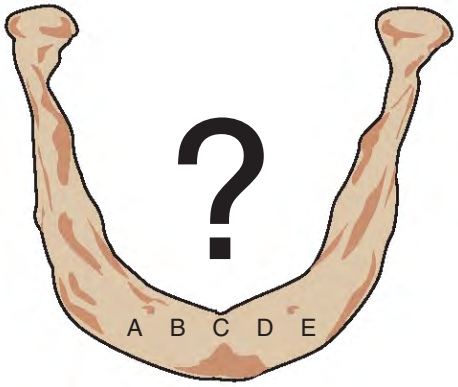


Figure 14-5 The anterior mandible is divided into five equal columns of bone between the mental foramens: A, B, C, D, and E.

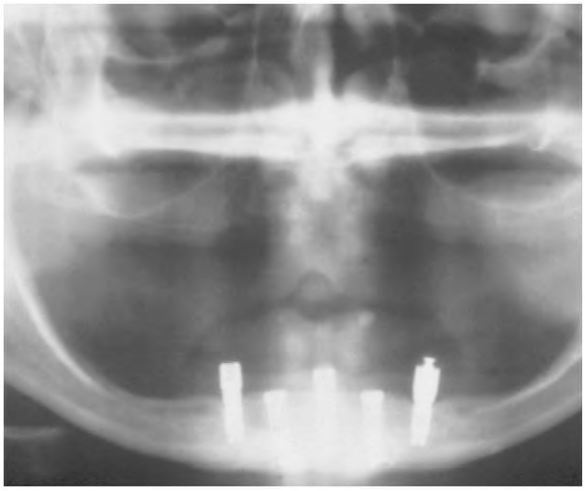


Figure 14-6 This patient wore a three-implant overdenture for several years. She then decided to improve the overdenture support, stability, and retention. The implant sites B and D could be added later because all five implant sites were planned initially.

the treatment option being executed, all five implant sites are mapped at the time of treatment planning and surgery. In this way, the patient always has the option to obtain additional implant support in the future. For example, a patient may receive adequate support for an implant overdenture with four implants. However, if the patient desires a fixed prosthesis in the future, these four implants may fall short of the new requirements. If the implant surgeon did not plan an additional implant site at the initial surgery but instead placed the four implants an equal distance apart, the additional space may not be available without removing one of the present implants. In addition, a patient may desire a completely implant-supported restoration as an RP-4 or fixed prosthesis but cannot afford the treatment all at once. Three implants in the A, C, and E positions and an overdenture may be provided now, and two implants may be added in the B and D locations later and a completely implant-supported overdenture or fixed restoration may be fabricated (Figures 14-6 and 14-7).

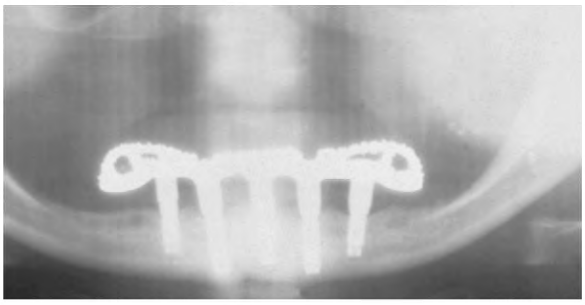


Figure 14-7 A hybrid fixed prosthesis was fabricated after the two additional implants were placed (same patient as Figure 14-6).

If an implant complication occurs, the preselected option sites permit corrective procedures. If implants were placed in the A, B, D, and E positions, and an implant fails to achieve rigid fixation, the failed implant may be removed and an additional implant placed in the C position at the same time. This saves an additional surgery and eliminates the time required for bone healing before another implant could be reinserted.

OVERDENTURE TREATMENT OPTIONS

In 1985 the author presented five organized treatment options for implant-supported mandibular overdentures in the completely edentulous patient.[12,83,84] The treatment options range from primarily soft tissue support and implant retention (RP-5) to a completely implant-supported prosthesis (RP-4) with rigid stability (Table 14-1). The options err on the side of safety to reduce the risk of failure or complications of bone loss and superstructure loosening. The initial treatment options are presented for completely edentulous patients with Division A (abundant) or B (sufficient) anterior bone, treated with Division A anterior root form implants of 4 mm or greater diameter. Modifications related to posterior ridge support and arch form also are discussed. Following these standardized conditions, anterior bone volume conditions of moderate atrophy (Division C minus height [C–h]) are presented.

The dentist evaluates the patient's existing dentures concerning support, retention, and stability. Support is related to the resistance to occlusal load. Retention describes the resistance of the prosthesis to movement away from the tissues. Stability is the lateral resistance criterion. The patient's complaints, anatomy, desires, and financial commitment determine the amount of implant support, retention, and stability required to address these conditions predictably. The amount of resistance provided in implant overdentures is related to the number and position of the implants (see Table 14-1). One should emphasize that most mandibular overdentures should be designed to eventually result in an RP-4 prosthesis, as previously discussed.

WWW.HIGHDENT.IR

Table 14-1 Mandibular Overdenture Treatment Options

OPTION	DESCRIPTION	REMOVABLE PROSTHESIS TYPE 5
OD-1	Implants in the B and D positions independent of each other	Ideal denture Ideal anterior and posterior ridge form Cost is a major factor Retention only PM-6
OD-2	Implants in the B and D positions, rigidly joined by a bar	Ideal posterior ridge form Ideal denture Cost is a major factor Retention and minor stability PM-3 to PM-6
OD-3A	Implants in the A, C, and E positions, ridgidly joined by a bar if posterior ridge form is good	Ideal posterior ridge form Ideal denture Retention and moderate stability PM-2 to PM-6 (two-legged chair)
OD-3B	Implants in the B, C, and D positions, joined by a rigid bar when posterior ridge form is poor	Division C–h anterior bone volume Poor posterior ridge form Retention and minor stability PM-3 to PM-6
OD-4	Implants in A, B, D, and E positions, rigidly joined by a bar cantilevered distally about 10 mm	Patient desires greater retention, major stability, and support PM-2 to PM-6 (three-legged chair)
OD-5	Implants in the A, B, C, D, and E positions, rigidly joined by a bar cantilevered distally about 15 mm	Patient has high demands or desires Retention, stability, and support PM-0 (four-legged chair)

From Misch CE: *Misch Implant Institute manual*, Dearborn, Mich, 1984, Misch International Implant Institute.
OD, Overdenture option; PM, prosthesis movement class; C–h, Division C minus height bone class.

Box 14-4 Patient Selection Criteria: OD-1

- Anatomical conditions are good to excellent.
- Posterior ridge form is an inverted U shape.
- Patient's needs and desires are minimal, primarily related to lack of retention.
- Cost is the primary factor.
- Hygiene is improved.
- Arch form is tapered.
- Additional implants will be inserted within 3 years.

Overdenture Option 1

The first treatment option for mandibular overdentures (OD-1) is indicated primarily when cost is the most significant patient factor. However, the patient's desires should also be minimal, and the bone volume should be abundant (Division A or B). The posterior ridge form should be an inverted U shape, with high parallel walls for good to excellent anatomical conditions for conventional denture, support, and stability (Box 14-4). The problem associated with the existing denture relates primarily to the amount of retention. Under these conditions, two implants may be inserted in the B and D positions. The implants remain independent of each other and are not connected with a superstructure. The most common type of attachment used in OD-1 is an O-ring design, and the prosthesis movement should be as much as is practical.

Positioning of the implants in the B and D position is a much better prosthetic option in OD-1 than positioning in the A and E regions (Figure 14-8). Kennedy Class 1 patients with bilateral distal extensions and anterior missing teeth often are restored with a fixed prosthesis anteriorly and a Class 1 removable partial denture. This eliminates the unfavorable rocking leverages that exist when replacement denture teeth are anterior to the fulcrum line. If only two natural canines remain, a cross-arch tissue bar can be placed to gain a favorable distribution of forces in the anterior region.[85] Likewise, independent implants in the A and E positions allow a greater amplitude of rocking of the restoration compared with implants in the B and D regions. When using B and D implants, the anterior movement of the prosthesis is reduced, and the prosthesis even may act as a splint for the two implants during anterior biting forces, thereby decreasing some of the stress to each implant. However, most situations do not allow the prosthesis to act as a true splint because a stress relief attachment permits movement in any plane. As a result, only one implant is loaded at a time in most situations. The stability and support of the prosthesis are gained primarily from the anatomy of the mandible and prosthesis design, which is similar to a complete denture. The implant support mechanism is poor because stress relief is permitted in any plane.

The patient's primary advantage with OD-1 is cost. The existing restoration often may be adapted with an intraoral rebase and pickup procedure around the implants and attachments. Additional indications are

WWW.HIGHDENT.IR

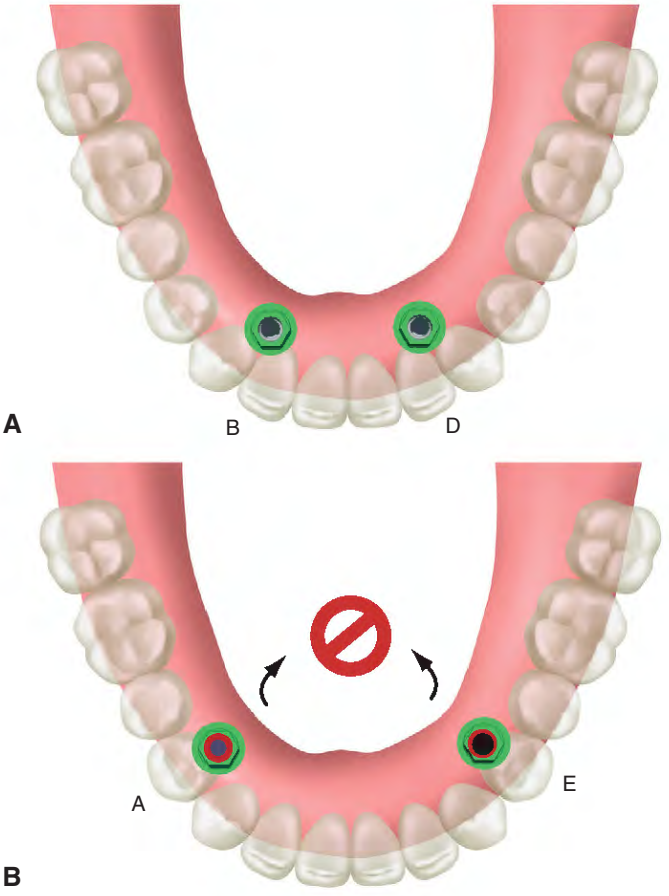


Figure 14-8 Overdenture option 1 consists of two independent implants. These are best placed in the B and D positions **(A)** to limit the forward rocking of the restoration during function. Independent implants in the A and E positions **(B)** allow a greater rocking of the restoration and place greater leverage forces against the implants.

when arch shape is considerably tapered such that a connecting bar would be cantilevered too far to the facial or would interfere with speech and mastication if too lingual. Hygiene procedures also are facilitated with independent ball attachments.

The disadvantages of the OD-1 relate to its relatively poor implant support and stability, compared with the other options (which have connecting bars), because of the independent nature of the implants. Jemt et al.[4] demonstrated a decrease in occlusal force when the bar connecting implants was removed from implant overdenture patients. In addition, bone loss in the edentulous regions of the mandible is not reduced significantly because only two anterior implants are inserted.

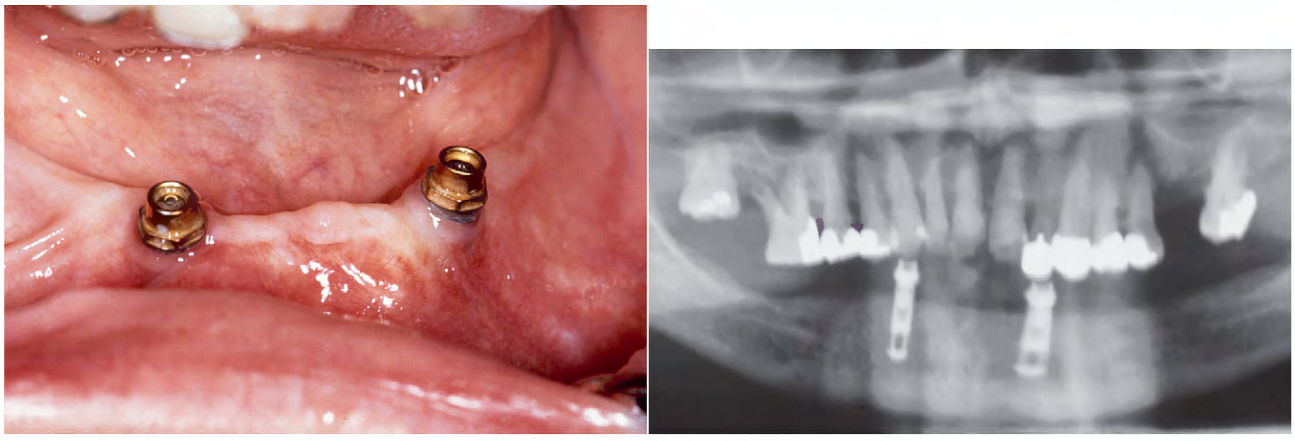
The other disadvantages of OD-1 relate to an increase in prosthetic maintenance appointments. For the restoration to be inserted and function ideally, the two implants should be parallel to each other, perpendicular to the occlusal plane, at the same horizontal height (parallel to the occlusal plane), and equal distance off the midline. If one implant is not parallel to the other, the prosthesis will wear one attachment faster because of the greater displacement during insertion and removal than the other. If the angulation difference is severe, the prosthesis may not engage one attachment at all. The implants also should be perpendicular to the occlusal plane. Because the goal is to allow the posterior regions of the overdenture to rock downward and load the soft tissue over the mandibular buccal shelves for support, the hinge rotation should be at 90 degrees to the rotation path. In addition, because only two implants sustain the occlusal load during function or parafunction, minimization of the forces to the implant components and crestal bone by placing them in the long axis of the implant body and perpendicular to the occlusal plane is ideal.

The two independent implants should be positioned at the same occlusal height, parallel to the occlusal plane. If one implant is higher than the other, the prosthesis will disengage from the lower implant during function and rotate primarily on the higher implant (Figure 14-9). This situation will accelerate the wear of the O-ring or attachment on the lower implant. In addition, because the higher implant receives the majority of the occlusal load, an increased risk of complications may occur, including abutment screw loosening, crestal bone loss, and implant failure.

The implants should be equal distance off the midline. If one implant is more distal (farther from the midline), it will serve as the primary rotation point or fulcrum when the patient occludes in the posterior segments. As such, the more medial implant attachment will wear faster, and the more distal implant will receive a greater occlusal load. As a consequence of additional maintenance risks, independent implants should be used less frequently than implants joined together with a bar. Attachments in a connection bar may be placed by the laboratory in similar horizontal, vertical, and axial planes much easier than the surgeon placing the implants.

The OD-1 is used as a treatment option when patients understand that additional implant support is beneficial, but financial constraints require a transition period of a few years before placing additional implants. The next goal in the treatment plan is to convert OD-1 patients to an RP-5 prosthesis with more support and stability before the loss of the posterior bone in the mandible behind the foraminae. As soon as the patient can afford two more implants, the implants should be placed in the A and E position, and all four ABDE implants should be connected with a bar that may be cantilevered to help reduce the posterior bone loss (OD-4). If bone height and width distal to one mental foramen are adequate, the additional implants may be positioned in one of the first molar regions. This plan will help maintain posterior bone, limit the cantilever to only one side, and greatly improve the anteroposterior distance (A-P spread) between implants and allow an RP-4 restoration.

WWW.HIGHDENT.IR



A B

Figure 14-9 **A,** When one implant is placed more anterior than the other (as shown), the more distal implant is the fulcrum when chewing in the posterior region and the more anterior implant is the fulcrum for movement when the patient incises food. This causes instability, wearing of the attachments, and loosening of the O-rings from the implant bodies. **B,** Two independent implants should be at the same height, equal distance off the midline, and parallel to each other. When the implants are positioned as in this radiograph, one implant (not two) becomes the dominant fulcrum and increases the risk of overload complications. This is especially important when force factors are higher than usual. The patient's occlusal plane also should be modified to allow a bilateral balanced occlusion on an RP-5 overdenture.

Box 14-5 Patient Selection Criteria: OD-2

- Anatomical conditions are good to excellent.
- Posterior ridge forms an inverted U shape.
- Patient's need and desires are minimal, primarily related to lack of retention.
- Patient can afford new prosthesis and connecting bar.
- Additional implants will not be inserted for more than 3 years.

Overdenture Option 2

The second treatment option for a mandibular overdenture (OD-2) is selected as the initial option more often than OD-1. The implants are positioned in locations B and D and splinted together with a superstructure bar without any distal cantilever (Figure 14-10). Reduced loading forces are exerted on two anterior implants when splinted with a bar compared with individual implants.[86-89] The bar is designed to position the attachments an equal distance off the midline, parallel to each other, at the same occlusal height, and in a similar angulation to provide added retention[27] (Box 14-5).

The two splinted implants should not be in the A and E positions (Figure 14-11). There are many reasons why two implants placed in the A and E positions should not be splinted together. Because these implants are placed just anterior to the mental foraminae, they are usually in the first premolar positions. This results in a curved arch form anterior to the implant sites and is too long a span relative to occlusal loading and flexibility of the metal bar.

The superstructure that follows the anterior curve of the arch results in an improved lingual contour of the restoration. However, the curve corresponds to an increased length and even greater flexibility of the superstructure. Because the bar is under the anterior teeth but anterior to the implants, a greater moment of force also is created. The prosthesis attachment system to the superstructure also may be compromised if clips are used for retention. The clips must be perpendicular to the path of rotation but a curved bar often places the clips closer to the implants and prevents rotation of the prosthesis. If the prosthesis rests against the sides of the curved bar, the prosthesis movement may be reduced to PM-0. This places a much greater lateral load on the implant system. Bars that course in a tangential direction do not permit friction-free rotation of the prosthesis around the fulcrum. Excess torsional loading is exerted on the implants and bar resulting in screw loosening or crestal bone loss.

The distance between A and E implants represents approximately a span of six teeth. The superstructure flexibility is related to the length. As a result, five times more flexure is observed than if the implants were in the B and D locations.[89] The increase in superstructure movement may result in loosening of the coping screws. After this occurs, the remaining attached implant receives a dramatic increase in moment of forces from the long lever arm of the superstructure. This increase in force may result in bone loss, mobility of the implant, and possible fracture of an implant component.[89] The ideal distance between the implants is in the 14- to 16-mm range or B and D positions. However, implants placed too close to each other will result in reduced prosthesis stability during function.

Another problem of joining A and E implants is the sagittal position of the superstructure. If the bar is straight and not bent to follow the arch, it occupies a lingual position relative to the arch. The lingual flange of the denture then extends as much as

WWW.HIGHDENT.IR

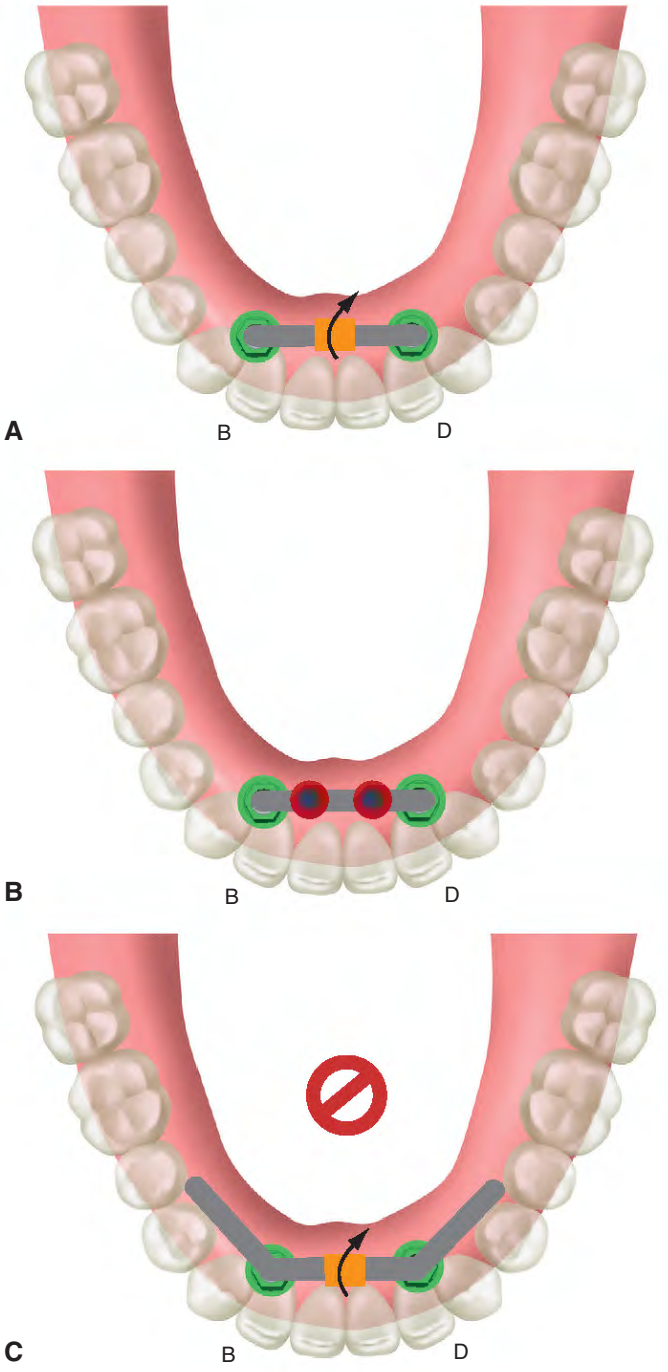


Figure 14-10 **A,** Treatment Option 2 has implants in the B and D positions and a bar joins the implants. The bar should not be cantilevered off the distal side of the implants. The prosthesis movement will be reduced and too much force on the bar and implants will increase complications. Attachments such as an O-ring **(B)** or a Hader clip **(C),** which allow movement of the prosthesis, can be added to the bar. The attachments are placed at the same height at equal distance off the midline and parallel to each other.

10 mm more lingually and 7 mm more vertically to accommodate the attachment, which is connected over the superstructure.

Because the teeth are set over or are anterior to the crest of the ridge, anterior to the superstructure bar, rotation and tipping of the restoration are more

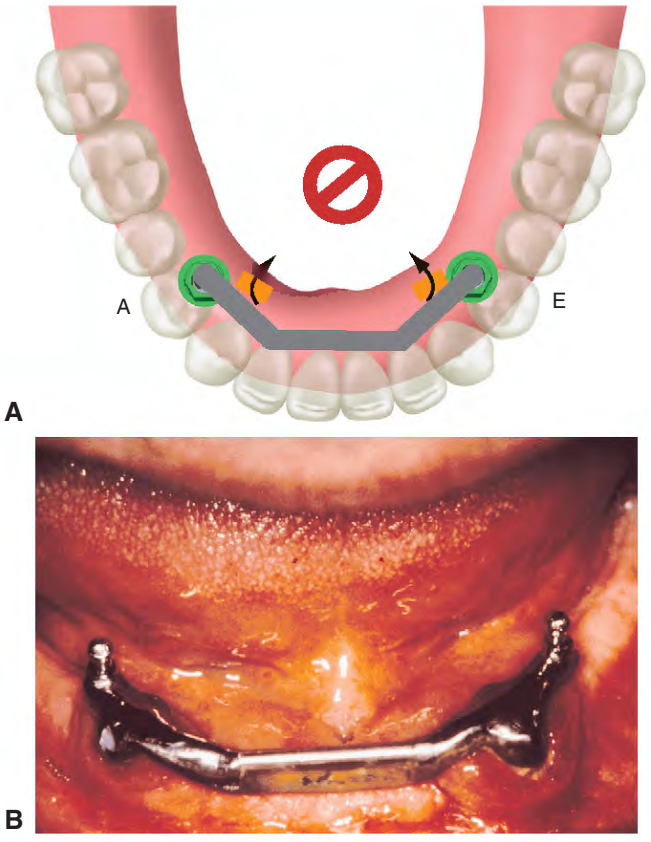


Figure 14-11 **A,** Implants in positions A and E should not be splinted. **B,** This patient had an overdenture constructed on the two first premolar teeth. The bar became uncemented in the patient's right side and then acted as a cantilever on the patient's left premolar. This situation is similar to implant positions A and E because the mental foramens are most often between the premolars or distal to the second premolar.

prevalent. The moment of force on a straight bar connecting implants in the A and E positions is twice that for implants in the B and D locations.[89]

Implants splinted in the A and E positions have greater potential load per surface area compared with implants in the B and D regions. The bite force increases toward the posterior aspects of the mouth. As a result, a greater vertical load is also present, with increased stresses when implants are placed in the A and E positions.

The A and E positions give more lateral stability to the prosthesis than the B and D positions. However, only two implants resist this lateral load. In contrast, the B and D positions increase lateral movement of the prosthesis, which is a patient disadvantage, but the positioning also decreases the lateral forces on the implants (Box 14-6). As a result of these many disadvantages, the placement of two implants in the A and E positions is strongly discouraged.

If the surgeon inadvertently inserts the implants in the A and E position, two options exist. The first is to place at least one additional implant, usually in the C position. The second is to leave the implants

WWW.HIGHDENT.IR

independent with O-ring attachments. With the second option, the anatomical ridge form should be good to excellent, and the overdenture should have excellent support and retention independent of the implants. The two implants should not be splinted to reduce complications because they are too far apart.

Two implant overdentures are not indicated in C–h or D bone and are not indicated when opposing natural teeth. The increase in crown height and the poorer posterior ridge form or the increase in bite forces place additional stresses on the implant system and increase complications. Additional implants should be used to decrease the implant and prosthetic risks. The length of the edentulous span, the position of the connecting bar, the flexure of the metal span, and the forces on the abutments create considerable risk in this treatment option. Instead, the B and D implant positions are closer to the canine positions and are much better suited for force and prosthetic guidelines.

Patient selection criteria for OD-2 treatments include the following (see Box 14-5):

- Anatomical conditions for a traditional denture are good to excellent.
- The posterior ridge form is an inverted U shape and provides good to excellent support and lateral stability.
- The patient's complaints are minimal and relate primarily to retention.
- The patient requires a new prosthesis and is willing to invest slightly more time and expense than the patient with the OD-1 option.
- When the patient is unable to insert additional implants within a short time frame (within 3 years), OD-2 is safer than an OD-1 independent implant approach. Some disadvantages of OD-2 treatments compared with OD-1 are possible tissue hyperplasia under the bar, more difficult hygiene under the bar, and a more expensive initial treatment option (because a bar and retentive elements are included).

Box 14-6 Disadvantages of A and E Splinted Implants (First Premolar to First Premolar)

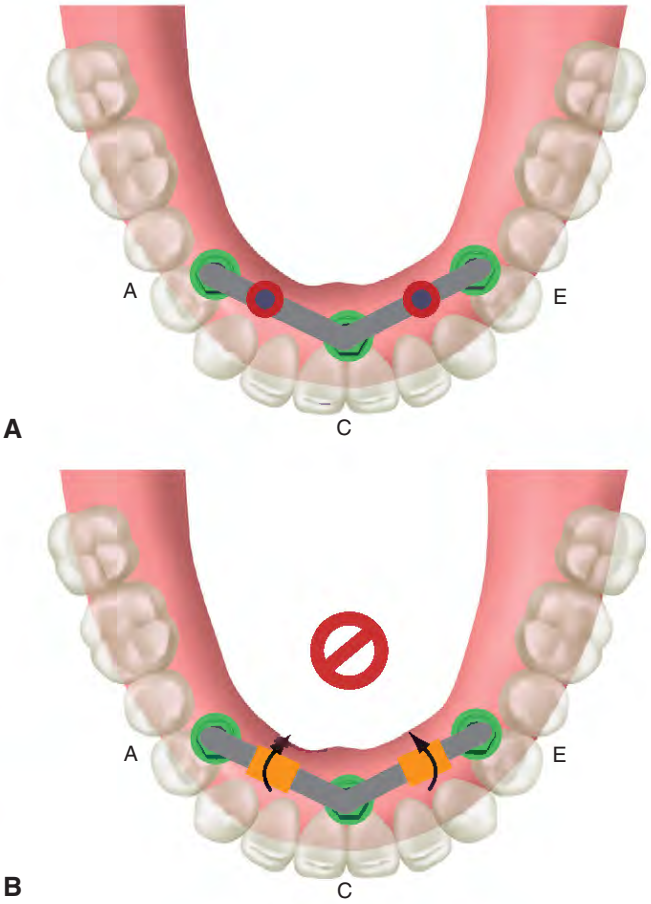
- Implants joined with straight bar are lingual to ridge.
  - Difficulty with speech
  - Anterior tipping of overdenture
  - Five times greater bar flexure than B and D positions
- Implants are joined with anterior curved bar.
  - Greater bar flexibility (nine times the B and D positions)
  - Increased screw loosening
  - Increased moment forces on anterior aspect of prosthesis
  - Attachment of curved bar may prevent prosthesis movement
- Bite force is higher than for B and D positions.
  - Greater lateral load from prosthesis to implants than B and D positions

Box 14-7 Patient Selection Criteria: OD-3

- Patient's needs and desires require improved retention, support, and stability
- Cost a moderate factor:
  - Anatomical conditions good to excellent
  - Posterior ridge forms inverted U shape

Overdenture Option 3

Three root form implants are placed in the A, C, and E positions for the third overdenture treatment option (OD-3) (Box 14-7). A superstructure bar connects the implants, but with no distal cantilever (Figure 14-12). The advantages of splinting A, C, and E implants compared with implants in the B and D positions are many (Box 14-8). The additional implant provides a sixfold reduction in superstructure flexure and limits



A

B

Figure 14-12 Overdenture option 3A corresponds to implants in the A, C, and E positions connected with a bar. The attachments should be positioned to allow movement of the distal section of the prosthesis **(A).** Two nonaligned Hader clips will not allow movement **(B).**

WWW.HIGHDENT.IR

Box 14-8 Advantages of Splinted A, C, and E Implants

- Six times less bar flexure compared with A and E positions
- Less screw loosening
  - Less metal flexure
  - Three implant abutments
- Less stress to each implant compared with A and E implants
  - Greater surface area
  - More implants
  - Greater A-P distance
- One-half moment force compared with A and E implants
- Less prosthesis movement
- One implant failure still provides adequate abutment support

A-P, Anteroposterior.

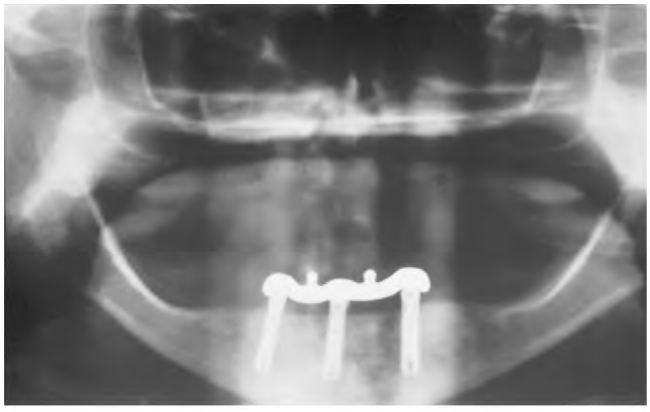


Figure 14-13 Panoramic radiograph of a mandible with Division A bone and overdenture option 3A, with implants in the A, C, and E positions.

the consequences previously discussed.[87] In addition, screw loosening occurs less frequently because three coping screws retain the superstructure rather than two. Implant reaction forces are reduced with a third implant as compared with two implants. The greater surface area of implant to bone allows better distribution of forces. The risk of abutment or coping screw loosening is reduced further because force factors are decreased. Three permucosal sites distribute stresses more efficiently and minimize crestal bone loss. Because the crestal bone is the first region of the bone to be affected, this represents a major advantage. The reduction in the maximum moment of force is twofold with a three-implant system compared with two implants in the A and E regions.[89]

The implants splinted in the A, C, and E positions should not form a straight line. The C implant is anterior to the more distal A and E implants and directly under the cingulum position of the denture teeth. The restoration benefits from direct occlusal load to the implant support in the anterior arch. When more than two implants are in the anterior mandible, a tripod support system may be established. To determine this benefit, the distal of the most posterior implants on each side are connected with a straight line. The distance from this line to the perpendicular position of the center implant is called the A-P spread.[89-91] The greater this dimension, the more biomechanically stable the implants when splinted together. The greater the A-P spread of the A, C, and E implants, the greater the biomechanical advantage of the bar to reduce stress on the implant and the better the lateral stability of the implant bar and overdenture system. Rotation of the prosthesis may also be more limited compared with OD-1 and OD-2. Therefore the third implant for OD-3 is a considerable advantage for the mandibular edentulous patient. This is usually the first treatment option for a patient with minimal complaints who is concerned primarily with retention and anterior stability when cost is a moderate factor. The posterior ridge form determines the posterior lingual flange extension of the denture, which limits lateral movement of the restoration. If the anterior and posterior ridge form is favorable (Divisions A or B), the implants are placed in the A, C, and E areas, and a wide range of attachments is available (Figure 14-13).[89]

If the posterior ridge form is poor (Division C–h), the lack of lateral stability places additional forces on the anterior implants. Implants then are best placed on the BCD position to allow greater freedom of movement of the prosthesis (Figure 14-14). The greater the stress to the system, the greater prosthesis movement/stress relief indicated. This increases the posterior movement of the restoration, but decreases the amount of stress placed on the implants and screw-retained bar.

The prosthesis movement for three implants with C–h posterior bone should be greater to minimize forces on the implants and bar retention system. If the patient with poor posterior ridge form requires more stability, more than three implants are indicated. In Division D posterior mandibles, five anterior implants are indicated to support the restoration.

When the patient can afford additional implants to those in the A, C, and E positions, the next implant placement is in the B and D positions when the posterior bone is inadequate for implants (C–h). When posterior bone permits, the two new implants are positioned with one in a molar region and the other inserted in the contralateral B or D position.

Overdenture Option 4

In the fourth mandibular overdenture option (OD-4), four implants are placed in the A, B, D, and E positions. These implants usually provide sufficient support to include a distal cantilever up to 10 mm on each side if the stress factors are low (Figure 14-15). The cantilevered superstructure is a feature of the four or more implant treatment option for three reasons: The first relates to

WWW.HIGHDENT.IR

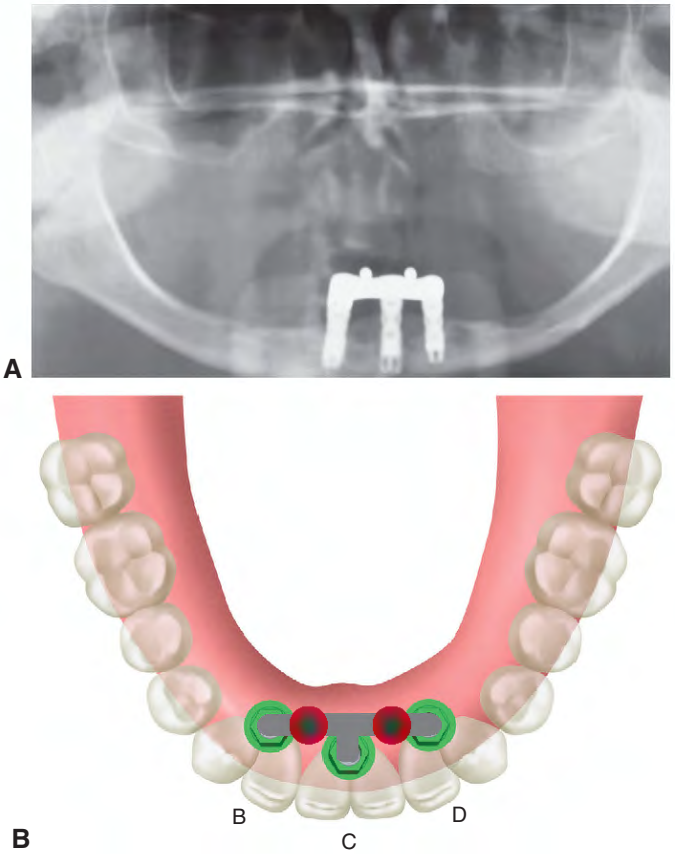


Figure 14-14 **A,** A panoramic radiograph of a Division C–h edentulous mandible. The patient should have more than three implants to protect the posterior bone loss. When the posterior ridge is poor and only three implants are placed, the implants are placed in the B, C, and D positions to decrease the risk of overload to the implants, screws, and bone and to allow the fabrication of an RP-5 prosthesis with distal movement of the prosthesis **(B).**

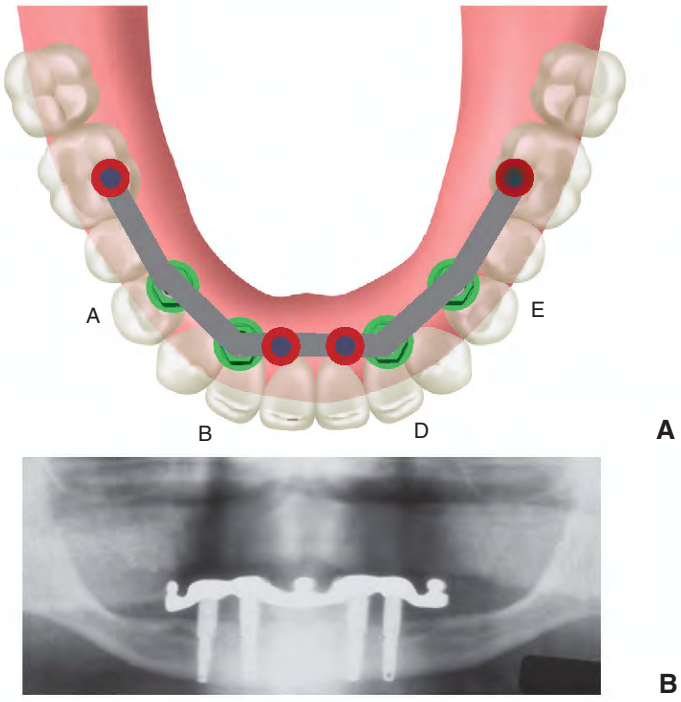


Figure 14-15 **A,** In overdenture option 4, four implants are placed in the A, B, D, and E positions. The implants provide sufficient support for a distal cantilever up to 10 mm. **B,** Panoramic radiograph of a mandibular overdenture option 4. In this option implants in the A, B, D, and E positions are connected with a cantilevered bar (up to 10 mm). The stress-breaking attachments are designed to allow some movement of the prosthesis during function.

the increase in implant support compared with OD-1 to OD-3. The second is that the biomechanical position of the splinted implants is improved in an ovoid or tapering arch form compared with OD-1 or OD-2. The third is related to the additional retention provided for the superstructure bar, which limits the risk of screw loosening and other related complications of cantilevered restorations.

In considering a distal cantilever for a mandibular overdenture bar, the implant position is the primary local determinant. Cantilevers may be compared with a class 1 lever in mechanics. The distalmost implant on each side acts as a fulcrum when occlusal forces are applied to the distal cantilever. Therefore the amount of the occlusal force is magnified by the length of the cantilever, which acts as a lever. For example, a 25-lb load to a 10-mm cantilever results in a 250-lb moment force.

This moment force is resisted by the length of the bar anterior to the fulcrum. Therefore if the two anterior implants are 10 mm from the fulcrum (distal implants), the effect of the posterior cantilever is countered. If the implants are 5 mm apart, the mechanical advantage of the lever is the 10-mm cantilever divided by the 5-mm A-P spread, which equals 2. A 25-lb distal force is magnified to 50 lb to the anterior implant and 75 lb (50 + 25 = 75) to the distal (fulcrum) implant.

The mandibular arch form may be square, tapering, or ovoid. Square arch forms limit the A-P spread between implants and may not be able to counter the effect of a distal cantilever. Therefore rarely are distal cantilevers designed for square arch forms. In a tapering arch form, the A-P spread between implants in the AE and DB positions is greater and therefore permits a longer distal cantilever. This A-P spread is often 10 mm and therefore often permits a cantilever up to 10 mm from the A and E positions. In an ovoid arch, which is most common, the A-P spread between AE and BD is usually 8 mm. Therefore the cantilever may be up to 8 mm long distally from the A and E implants.

The A-P spread is only one factor to determine the length of the cantilever. When stress factors such as occluding forces are greater, the cantilever is decreased. When the crown height is doubled, the moment forces are doubled. Therefore under ideal, low-force conditions (crown height less than 15 mm, no parafunction, older females, opposing maxillary denture), the cantilever may be up to 1.5 times the A-P spread for OD-4 overdentures.

WWW.HIGHDENT.IR

The patient's indications for this OD-4 include moderate to poor posterior anatomy that causes a lack of retention and stability, soft tissue abrasions, and difficulty with speech. The edentulous posterior mandible resorbs four times faster than the anterior mandible. In the C–h posterior mandible the external oblique and mylohyoid ridges are high and often correspond to the crest of the residual ridge. The muscle attachments therefore are at the crest of the ridge. The patient's complaints and desires are more demanding than for the previous treatment options (Box 14-9).

The OD-4 prosthesis is indicated to obtain greater stability and a more limited range of prosthesis motion. The overdenture attachments often are placed in the distal cantilevers with an O-ring attachment in the midline. The prosthesis is still RP-5, but with the least soft tissue support of all RP-5 designs. The anterior attachment must allow vertical movement for the distal aspect of the prosthesis to rotate toward the tissue. Clips, which permit rotation, are difficult to use on cantilevered superstructures. To allow movement, the clip must be placed perpendicular to the path of rotation, not along the cantilevered bar where its only function then is retention.

The patient benefits from the four implants because of greater occlusal load support and lateral prosthesis stability. The prosthesis only loads the soft tissue over the first and second molars and retromolar pad regions. Therefore the amount of occlusal force is reduced because the bar does not extend to the molar position, where the forces are greater. The amount of distal cantilever is related primarily to the force factors and to the arch form, which corresponds to the A-P spread from the center of the most anterior implants to the distal portions of the A and E implants.

The next treatment plan for the patient with a greater budget is to add an additional implant in one of the first molar positions (preferred) or the C position. Both these options increase the A-P spread to fabricate a prosthesis with enhanced implant support. The goal is to convert all patients to an RP-4 or fixed restoration (OD-5).

Box 14-9 Patient Selection Criteria: OD-4

- Moderate to severe problems with traditional dentures
- Needs or desires are demanding
- Need to decrease bulk of prosthesis
- Inability to wear traditional prostheses
- Desire to abate posterior bone loss
- Unfavorable anatomy for complete dentures
- Problems with function and stability
- Posterior sore spots

Box 14-10 Patient Selection Criteria: OD-5

- Moderate to severe problems with traditional dentures
- Needs or desires are demanding
- Need to decrease bulk of prosthesis
- Inability to wear traditional prostheses
- Desire to abate posterior bone loss
- Unfavorable anatomy for complete dentures
- Problems with function and stability
- Posterior sore spots
- Moderate to poor posterior anatomy
- Lack of retention and stability
- Soft tissue abrasion
- Speech difficulties
- More demanding patient type

Overdenture Option 5

The fifth mandibular overdenture option (OD-5) is designed for two types of patients. This is a minimum treatment option for patients with moderate to severe problems related to a traditional restoration. The needs and desires of the patient are often most demanding and may include limiting the bulk or amount of the prosthesis, major concerns regarding function or stability, posterior sore spots, and the inability to wear a mandibular denture (Box 14-10).

The second patient condition is for the treatment of continued bone loss in the posterior mandible. If no prosthetic load is on the posterior bone, the resorption process is delayed considerably and usually is reversed. Therefore even when no posterior implants are inserted, the cantilevered bar and overdenture avoid load to the residual ridge and often halt its resorption process. Recent evidence shows that completely implant-supported prostheses may increase the amount of posterior bone height, even when no posterior implants are inserted.[76-78] A better option to prevent this bone loss is the insertion of posterior implants before atrophy. This treatment option is more likely when the patient desires a fixed restoration or the arch form is square.

In the OD-5 treatment, five implants are inserted in the A, B, C, D, and E positions. The superstructure is cantilevered distally a maximum of 2.5 times the A-P spread (if all the stress factors are low) and averages 15 mm, which places it under the first molar area (Figures 14-16 and 14-17). If any stress factors are not favorable, the cantilever should be reduced. Stresses increase with the length of cantilever and should be planned carefully based on force factors and the existing anatomy.[90-98] In a study in which the failure criterion was the failure of the screw joint with arrangements of three, four, five, and six implants submitted to forces from 143 to 400 N, the transmitted forces to the prosthetic connection always exceeded the yield strength of the system. This study emphasizes the fact that the A-P spread is not the only factor to be considered for cantilever length determination.[92]

WWW.HIGHDENT.IR

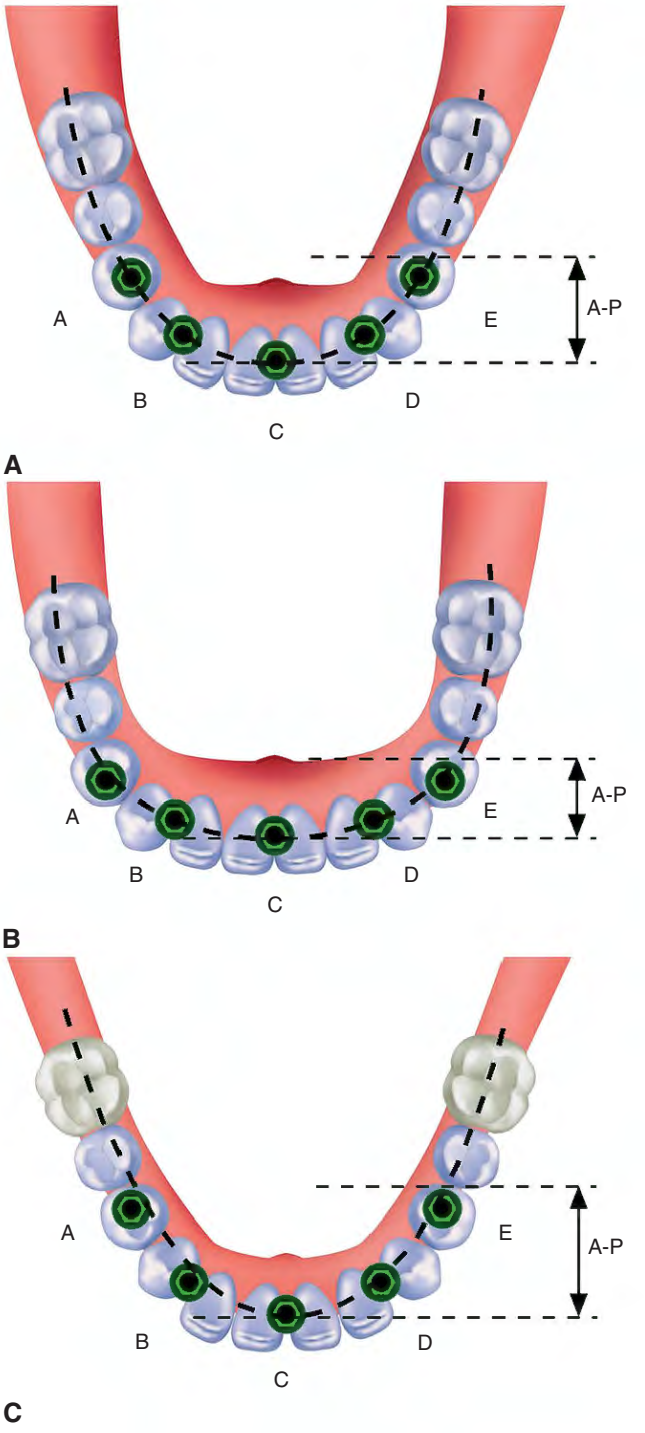


Figure 14-16 Arch shape affects the anteroposterior (A-P) distance. The ovoid arch form **(A)** often has an A-P distance of 6 to 8 mm. A square arch form **(B)** often has an A-P dimension of 2 to 5 mm. A tapered arch form **(C)** has the greatest A-P distance, larger than 8 mm.

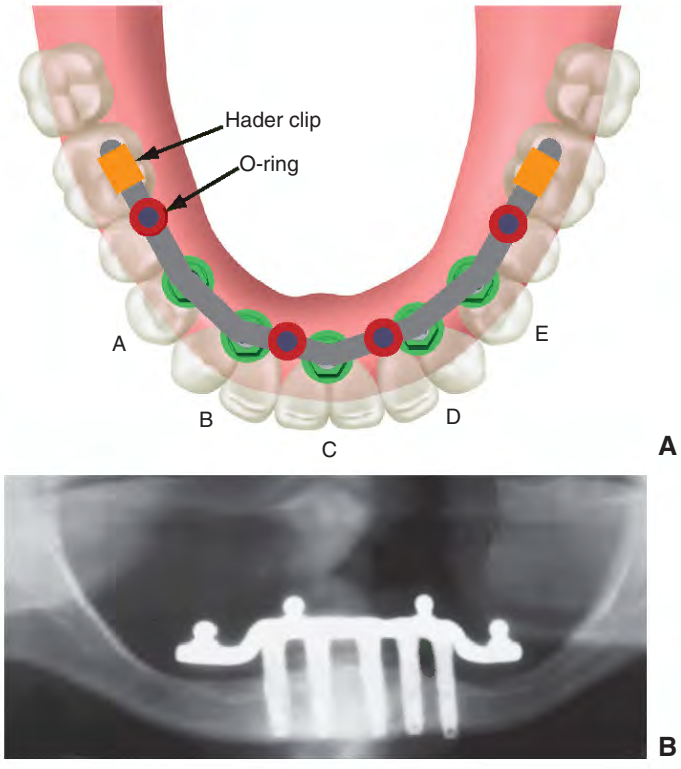


Figure 14-17 **A,** In the overdenture option 5, implants are placed in the A, B, C, D, and E positions. A bar splints the implants together and is cantilevered distally. The length of the cantilever depends on the anteroposterior distance and the force factors. **B,** A panoramic radiograph of an option 5 overdenture bar and five implants in the anterior mandible.

The Hidden Cantilever

The teeth on the final restoration usually do not extend beyond the first molar, so the last tooth does not extend beyond the bar. This helps prevent a hidden cantilever, which may extend beyond this position. The hidden cantilever is that portion of the cantilever that extends beyond the connecting bar. If the prosthesis does not rotate at the end of the bar to load the soft tissue, a hidden cantilever exists. For example, if the bar extends to the first molar but forces in the second molar of the restoration do not result in movement of the restoration down in the back and up in the front, the cantilever really is extended to the second molar position. Therefore the cantilever length is measured to the point of prosthesis movement, not to the end of the bar and attachment system. Under ideal conditions, the restoration is often an RP-4 completely implant-supported prosthesis.

When arch form or force factors do not permit an RP-4 restoration with only implants between the foramenae, placing an implant in one of the first molar positions should be considered. This increases the A-P spread and results with only one cantilever. Most often an RP-4 restoration is then acceptable with this implant position.

DIVISION C–H ANTERIOR MANDIBLES

The five treatment options proposed for mandibular implant–supported overdentures provide an organized

WWW.HIGHDENT.IR

approach to solving a patient's complaints or anatomical limitations.

The prosthesis support and range of motion should be part of the initial diagnosis. The treatment options initially proposed are designed for completely edentulous patients with Division A anterior bone in desire of an overdenture. These options are modified if the anterior bone is Division C–h. The increase in crown/implant ratio and decrease in implant surface area mandate modification of these initial options.

In the C–h anterior bone volume patient, one more implant is added to each option and OD-1 is eliminated completely. Therefore OD-2 has three implants (A, C, and E positions), OD-3 has four implants (A, B, D, and E regions), OD-4 has five implants (A, B, C, D, and E areas), and OD-5 has six implants. If six implants cannot be placed because of inadequate posterior bone, the cantilever length is reduced and a RP-5 restoration is fabricated.

DISCUSSION

The doctor and staff can explain the amount of support each treatment option can provide by comparing them with the support system of a chair. Treatment options OD-2 or OD-3 are related to a two-legged chair. The prosthesis provides some vertical support but can rock back and forth. Option OD-4 with four implants is compared with a three-legged chair. This system provides further support but can be rocked one way or the other under lateral forces. A four-legged chair provides the greatest support and is similar to OD-5, which is a stable, retentive prosthesis and RP-4 in design.

Overdenture bars may be cemented or retained with screws. Whether a cemented or screw-retained bar is used, the overdenture prosthesis retention is achieved by a similar combination of clips or O-ring attachments. Three advantages of a cemented bar are the insurance of a passive superstructure, reduced cost, and an easier bar impression technique.[25,99] Reports in the literature document similar success rates for both options, and the choice may be left to the discretion of the restoring doctor. However, stronger cements and a reduction of the cantilever length should be considered for OD-4 and OD-5 treatment plans because tensile forces may break the anterior cement seals and cause complications.[25,26]

SUMMARY

Implant overdentures borrow several principles from tooth-supported overdentures. The advantages of implant overdentures relate to the ability to place rigid, healthy abutments in the positions of choice. The number, location, superstructure design, and prosthetic range of motion can be predetermined and based on a patient's expressed needs and desires. Two implants placed just anterior to the mental foraminae rarely should be used. The overdenture should be designed to satisfy the patient's desires and anatomical limitations predictably.

The most common overdenture option used by the profession is the two-implant overdenture, with individual O-ring attachments. Yet the only benefit of this approach is a reduced initial cost. The bone loss accelerates in the posterior, and the maintenance of anterior bone is limited to the zone around each implant.

An ideal approach for the overall long-term health of the mandible is a complete implant–supported prosthesis. The bone volume is maintained in the anterior, and the posterior bone loss is significantly reduced. The stability of the prosthesis is maximal because it does not move on the connecting bar. The retention is excellent because it may have four to six attachments. The occlusal load support is on the implants, not the soft tissues.

The patient initially may not be able to afford an OD-5 option. However, an OD-3 may be converted to an OD-4 after several years and eventually to an OD-5 after several more years. If the transition from one option to another is in a short time frame (1 to 2 years), the implants may be independent and use an O-ring system short term. This reduces the fee for the transitional prosthesis because no bar is fabricated, and a rebase may be used to modify the prosthesis.

References

1. Perel ML: *Dental implantology and prostheses,* Philadelphia, 1980, JB Lippincott.
2. Babbush CA, Kent JN, Misiek DJ: Titanium plasma spray (TPS) Swiss screw implants for the reconstruction of the edentulous mandible, *J Oral Maxillofac Surg* 44:247-282, 1986.
3. Engquist B, Bergendal T, Kallus T et al: A retrospective multicenter evaluation of osseointegrated implants supporting overdentures, *Int J Oral Maxillofac Implants* 3:129-134, 1988.
4. Jemt T, Chai J, Harnett J: A 5-year prospective multicenter follow-up report on overdentures supported by osseointegrated implants, *Int J Oral Maxillofac Implants* 11: 291-298, 1996.
5. Wismeijer D, Van Waas MAJ, Vermeeren J: Overdenture supported by implants: a 6.5 year evaluation of patient satisfaction and prosthetic after care, *Int J Oral Maxillofac Implants* 10:744-749, 1995.
6. Awad MA, Lund JP, Dufresne E et al: Comparing the efficacy of mandibular implant-retained overdentures and conventional dentures among middle-aged edentulous patients: satisfaction and functional assessment, *Int J Prosthodont* 16:117-122,2003.
7. Awad MA, Lund JP, Shapiro SH et al: Oral health status and treatment satisfaction with mandibular implant overdentures and conventional dentures: a randomized clinical trial in a senior population, *Int J Prosthodont* 16:390-396, 2003.

WWW.HIGHDENT.IR

8. Thomason JM, Lund JP, Chehade A et al: Patient satisfaction with mandibular implant overdentures and conventional dentures 6 months after delivery, *Int J Prosthodont* 16:467-473, 2003.
9. Naert IE, Hooghe M, Quirynen M et al: The reliability of implant-retained hinging overdentures for the fully edentulous mandible: an up to 9-year longitudinal study, *Clin Oral Investig* 1:119-124, 1997.
10. Naert I, Alssaadi G, van Steenberghe D et al: A 10-year randomized clinical trial on the influence of splinted and unsplinted oral implants retaining mandibular overdentures: peri-implant outcome, *Int J Oral Maxillofac Implants* 19:695-702, 2004.

10a. Naert I, Alsaadi G, Quirynen M: Prosthetic aspects and patient satisfaction with two-implant-retained mandibular overdentures: a 10-year randomized clinical study, *Int J Prosthodont* 17:401-410, 2004.

11. Hutton JE, Heath MR, Chai JY et al: Factors related to success and failure rates at 3-year follow-up in a multicenter study of overdentures supported by Brånemark implants, *Int J Oral Maxillofac Implants* 10: 33-42, 1995.
12. Misch CE: Treatment options for mandibular implant overdentures: an organized approach. In Misch CE, editor: *Contemporary implant dentistry*, St Louis, 1993, Mosby.
13. Kline R, Hoar J, Beck GH et al: A prospective multicenter clinical investigation of a bone quality based dental implant system, *Implant Dent* 11:224-234, 2002.
14. Naert I, DeClercq M, Theuniers G et al: Overdentures supported by osseointegrated fixtures for the edentulous mandible: a 2.5 year report, *Int J Oral Maxillofac Implants* 3:191-196, 1988.
15. Mericke-Stern R: Clinical evaluation of overdenture restorations supported by osseointegrated titanium implants: a retrospective study, *Int J Oral Maxillofac Implants* 5:375-383, 1990.
16. Mericske-Stern R, Steinlin Schaffner T, Marti P et al: Peri-implant mucosal aspects of ITI implants supporting overdentures: a five-year longitudinal study, *Clin Oral Implants Res* 5:9-18, 1994.
17. Naert I, Gizani S, Vuylsteke M et al: A 5-year prospective randomized clinical trial on the influence of splinted and unsplinted oral implants retaining a mandibular overdenture: prosthetic aspects and patient satisfaction, *J Oral Rehabil* 26:195-202, 1999.
18. Batenburg RH, Meijer HH, Raghoebar GM et al: Treatment concept for mandibular overdentures supported by endosseous implants: a literature review, *Int J Oral Maxillofac Implants* 13:539-545, 1998.
19. Burns DR: Mandibular implant overdenture treatment: consensus and controversy, *J Prosthodont* 9:37-46, 2000.
20. Geertman ME, Boerrigter EM, Van Waas MA et al: Clinical aspects of multicenter clinical trial of implant-retained mandibular overdentures in patients with severely resorbed mandibles, *J Prosthet Dent* 75:194-204, 1996.
21. Hemmings KW, Schmitt A, Zarb GA: Complications and maintenance requirements for fixed prostheses and overdentures in the edentulous mandible: a 5-year report, *Int J Oral Maxillofac Implants* 9:191-196, 1984.
22. Davis DM, Rogers JO, Packer ME: The extent of maintenance required by implant retained mandibular overdentures: a 3-year report, *Int J Oral Maxillofac Implants* 11:767-774, 1996.
23. Jemt T, Book K, Linden B et al: Failures and complications in 92 consecutively inserted overdentures supported by Brånemark implants in severely resorbed maxillae: a study from prosthetic treatment to first annual check-up, *Int J Oral Maxillofac Implants* 7:162-166, 1992.
24. Jemt T, Lekholm U: Implant treatment in edentulous maxillae: a 5-year follow-up report on patients with different degrees of jaw resorption, *Int J Oral Maxillofac Implants* 10:303-311, 1995.
25. Judy KWM, Richter R: Implant supported overdenture prosthesis, *Pract Periodontics Aesthet Dent* 3:51-56, 1991.
26. Naert I, Quirynen M, Theuniers G et al: Prosthetic aspects of osseointegrated fixtures supporting overdentures: a 4-year report, *J Prosthet Dent* 65:671-680, 1991.
27. Naert I, Quirynen M, Hooghe M et al: A comparative prospective study of splinted and unsplinted Brånemark implants in mandibular overdenture therapy, *J Prosthet Dent* 71:486-492, 1994.
28. Chan MFW, Johnston C, Howell RA et al: Prosthetic management of the atrophic mandible using endosseous implants and overdentures: a 6-year review, *Br Dent J* 179:329-337, 1995.
29. Bergendal T, Engquist B: Implant supported overdentures: a longitudinal prospective study, *Int J Oral Maxillofac Implants* 13:253-262, 1998.
30. Wright PS, Watson RM: Effect of prefabricated bar design with implant-stabilized prostheses on ridge resorption: a clinical report, *Int J Oral Maxillofac Implants* 13:77-81, 1998.
31. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implant and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
32. Feine JS, Carlsson GS, Awad MA et al: The McGill consensus statement on overdentures, *Int J Prosthodont* 15:413-414, 2002.
33. Atwood DA, Coy WA: Clinical, cephalometric, and densitometric study of reduction of residual ridge, *J Prosthet Dent* 26:280-295, 1971.
34. Tallgren A: The continuing reduction of the residual alveolar ridges in complete denture wearers: a mixed-longitudinal study covering 25 years, *J Prosthet Dent* 27:120-132, 1972.
35. Tallgren A: The reduction in face height of edentulous and partially edentulous subjects during long-term denture wear: a longitudinal roentgenographic cephalometric study, *Acta Odontol Scand* 24:195-239, 1966.
36. Reddy SM, Geurs NC, Wang IC et al: Mandibular growth following implant restoration: does Wolff's law apply to residual ridge resorption? *Int J Period Rest Dent* 22: 315-321, 2002.
37. Carlsson GE, Kronstrom M, de Baat C et al: A survey of the use of mandibular implant overdentures in 10 countries, *Int J Prosthodont* 17:211-217, 2004.
38. Palmqvist S, Owall B, Schou S: A prospective randomized clinical study comparing implant-supported fixed prostheses and overdentures in the edentulous mandible: prosthodontic production time and costs, *Int J Prosthodont* 17:231-235, 2004.
39. Takanashi Y, Penrod JR, Lund JP et al: A cost comparison of mandibular two-implant overdenture and conventional denture treatment, *Int J Prosthodont* 17:181-618, 2004.
40. Attard NJ, Zarb GA: Long-term treatment outcomes in edentulous patients with implant overdentures: the Toronto study, *Int J Prosthodont* 17:425-33, 2004.
41. Schwartz-Arad D, Kidron N, Dolev E: A long-term study of implants supporting overdentures as a model for implant success, *J Periodontol* 76:1431-1435, 2005.

WWW.HIGHDENT.IR

42. Naert I, Gizani S, Vuylsteke M et al: A 5-year randomized clinical trial on the influence of splinted and unsplinted oral implants in the mandibular overdenture therapy. 1. Peri-implant outcome, *Clin Oral Implants Res* 9:70-177, 1998.
43. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 10:387-416, 1981.
44. Jemt T, Stalblad PA: The effect of chewing movements on changing mandibular complete dentures to osseo-integrated overdentures, *J Prosthet Dent* 55:357-361, 1986.
45. Geertman ME, Slagter AP, van Waas MA et al: Comminution of food with mandibular implant retained overdentures, *J Dent Res* 73:1858-1864, 1994.
46. Rissin L, House JE, Manly RS et al: Clinical comparison of masticatory performance and electromyographic activity of patients with complete dentures, overdentures and natural teeth, *J Prosthet Dent* 39:508-511, 1978.
47. Sposetti VJ, Gibbs CH, Alderson TH et al: Bite force and muscle activity in overdenture wearers before and after attachment placement, *J Prosthet Dent* 55:265-273, 1986.
48. Haraldson T, Jemt T, Stalblad PA et al: Oral function in subjects with overdentures supported by osseointegrated implants, *Scand J Dent Res* 96:235-242, 1988.
49. Mericke-Stern R: The forces on implant supporting overdentures: a preliminary study of morphologic and cephalometric considerations, *Int J Oral Maxillofac Implants* 8:256-263, 1993.
50. Mericke-Stern R, Hofman J, Wedig A et al: In vivo measurements of maximum occlusal force and minimal pressure threshold on overdentures supported by implants or natural roots: a comparative study, part I, *Int J Oral Maxillofac Implants* 8:641-649, 1993.
51. Jemt T, Book K, Karlsson S: Occlusal force and mandibular movements in patients with removable overdentures and fixed prostheses supported by implants in the maxilla, *Int J Oral Maxillofac Implants* 8:301-308, 1993.
52. Beumer J III, Roumanas E, Nishimura R: Advances in osseointegrated implants for dental facial rehabilitation following major head and neck surgery, *Semin Surg Oncol* 11:2000-2007, 1995.
53. Roumanas E, Nishimura RD, David BK et al: Clinical evaluation of implant retaining maxilla obturator prostheses, *J Prosthet Dent* 77:184-190, 1997.
54. Feine JS, de Grandmont P, Boudrias P et al: Within-subject comparisons of implant-supported mandibular prostheses: choice of prosthesis, *J Dent Res* 73:1105-1111, 1994.
55. de Grandmont P, Feine JS, Tache R et al: Within-subject comparisons of implant-supported mandibular prostheses: psychometric evaluation, *J Dent Res* 73: 1096-1104, 1994.
56. Burnes DR, Unger JW, Elswick RK Jr et al: Prospective clinical evaluation of mandibular implant overdentures. II. Patient satisfaction and preference, *J Prosthet Dent* 73:364-369, 1995.
57. Strietzel FP: Patients' informed consent prior to implant-prosthodontic treatement: a restrospective analysis of expert opinions, *Int J Oral Maxillofac Implants* 18: 433-439, 2003.
58. Boerrigter EM, Geertman ME, Van Oort RP et al: Patient satisfaction with implant-retained mandibular overdentures: a comparison with new complete dentures not retained by implants—a multicentre randomized clinical trial, *Br J Oral Maxillofac Surg* 33:282-288, 1995.
59. Humphris GM, Healey T, Howell RA et al: The psychological impact of implant-retained mandibular prostheses: a cross-sectional study, *Int J Oral Maxillofac Implants* 10:437-444, 1995.
60. Meijer HJ, Raghoebar GM, Van't Hof MA et al: Implant-retained mandibular overdentures compared with complete dentures: a 5 year follow up study of clinical aspects and patient satisfaction, *Clin Oral Implants Res* 10:238-244, 1999.
61. Harle TH, Anderson JD: Patient satisfaction with implant supported prostheses, *Int J Prosthodont* 6:153-162, 1993.
62. Wismeijer D, van Waas MA, Vermeeren JI et al: Patient satisfaction with implant-supported mandibular over-dentures: a comparison of three treatment strategies with ITI-dental implants, *Int J Oral Maxillofac Surg* 26: 263-267, 1997.
63. Attard N, Wei X, Laporte A et al: A cost minimization analysis of implant treatment in mandibular edentulous patients, *Int J Prosthodont* 16:271-276, 2003.
64. Carlsson GE, Kronstrom M, de Baat C et al: A survey of the use of mandibular implant overdentures in 10 countries, *Int J Prosthodont* 17:211-217, 2004.
65. Watson RM, Jemt T, Chai J et al: Prosthodontic treatment, patient response, and the need for maintenance of complete implant-supported overdentures: an appraisal of 5 years of prospective study, *Int J Prosthodont* 10: 345-354, 1997.
66. Walton JN: A randomized clinical trial comparing two mandibular implant overdenture designs: 3-year prosthetic outcomes using a six-field protocol, *Int J Prosthodont* 16:255-260, 2003.
67. Mac Entee MI, Walton JN, Glick N: A clinical trial of patient satisfaction and prosthodontic needs with ball and bar attachments for implant-retained complete overdentures: three-year results, *J Prosthet Dent* 93:28-37, 2005.
68. Johns RB, Jemt T, Heath MR et al: A multicenter study of overdentures supported by Brånemark implants, *Int J Oral Maxillofac Implants* 7:513-522, 1992.
69. Walton JN, McEntee MI: Problems with prostheses on implants: a retrospective study, *J Prosthet Dent* 71: 283-288, 1994.
70. Payne AG, Solomons YF: Mandibular implant-supported overdentures: a prospective evaluation of the burden of prosthodontic maintenance with 3 different attachment systems, *Int J Prosthodont* 13:246-253, 2000.
71. Watson RM, David DM: Follow up and maintenance of implant supported prostheses: a comparison of 20 complete mandibular fixed cantilever prostheses, *Br Dent J* 181:321-327, 1996.
72. Gotfredsen K, Holm B: Implant-supported mandibular overdentures retained with a ball or bar attachments: a randomized prospective 5-year study, *Int J Prosthodont* 13:125-162, 2000.
73. Kordatzis K, Wright PS, Meijer HJ: Posterior mandibular redidual ridge resorption in patients with conventional dentures and implant overdentures, *Int J Oral Maxillofac Implants* 18:447-452, 2003.
74. Blum IR, McCord JF: A clinical investigation of the morphological changes in the posterior mandible when implant-retained overdentures are used, *Clin Oral Implants Res* 15:700-708, 2004.

WWW.HIGHDENT.IR

75. Jacobs R, Schotte A, van Steenberghe D et al: Posterior jaw bone resorption in osseointegrated implant supported overdentures, *Clin Oral Implants Res* 3:63-70, 1992.
76. Davis WH, Lam PS, Marshall MW et al: Using restorations borne totally by anterior implants to preserve the edentulous mandible, *J Am Dent Assoc* 130:1183-1189, 1999.
77. Wright PS, Glantz PO, Randow K et al: The effects of fixed and removable implant-stabilized prostheses on posterior mandibular residual ridge resorption, *Clin Oral Implants Res* 13:169-174, 2002.
78. Jacobs R, van Steenberghe D, Nys M et al: Maxillary bone resorption in patients with mandibular implant supported overdentures or fixed prosthesis, *J Prosthet Dent* 70:135-140, 1993.
79. Barber HD, Scott RF, Maxson BB et al: Evaluation of anterior maxillary alveolar ridge resorption when opposed by the transmandibular implant, *J Oral Maxillofac Surg* 48:1283-1287, 1990.
80. Thiel CP, Evans DB, Burnett RR: Combination syndrome associated with a mandibular implant-supported overdenture: a clinical report, *J Prosthet Dent* 75:107-113, 1996.
81. Lechner SK, Mammen A: Combination syndrome in relation to osseointegrated implant-supported overdentures: a survey, *Int J Prosthodont* 9:58-64, 1996.
82. Kreisler M, Behneke N, Behneke A et al: Residual ridge resorption in the edentulous maxilla in patients with implant-supported mandibular overdentures: an 8-year retrospective study, *Int J Prosthodont* 16:295-300, 2003.
83. Misch CE: Implant overdentures relieve discomfort for the edentulous patient, *Dentist* 67:37-38, 1989.
84. Misch CE: Mandibular overdenture treatment options. In *Misch Implant Institute manual,* Dearborn, Mich, 1984, Misch International Implant Institute.
85. Renner RP, Boucher LJ: *Removable partial dentures,* Chicago, 1987, Quintessence.
86. Dolder E: The bar joint mandibular denture, *J Prosthet Dent* 11:689-707, 1961.
87. Jager K, Wirz EJ: *In vitro spannung analysen on implantaten fur zahnartzt und zahntechniker,* Berlin, 1992, Quintessenz.
88. Mericke-Stern R, Piotti M, Sirtes G: 3-D in vivo force measurements on mandibular implants supporting overdentures: a comparative study, *Clin Oral Implants Res* 7:387-396, 1996.
89. Bidez MW, Misch CE: The biomechanics of interimplant spacing. In *Proceedings of the Fourth International Congress of Implants and Biomaterials in Stomatology,* Charleston, SC, May 24-25, 1990.
90. English CE: Finite element analysis of two abutment bar designs, *Implant Dent* 2:107-114, 1993.
91. English CE: Bar patterns in implant prosthodontics, *Implant Dent* 3:217-229, 1994.
92. Van Zyl PP, Grundling NL, Jooste CH et al: Three dimensional finite element model of a human mandible incorporating osseointegrated implants for stress analysis of mandibular cantilever prostheses, *Int J Oral Maxillofac Implants* 10:51-57, 1995.
93. Clelland NL, Papazoglou E, Carr AB et al: Comparison of stress transferred to a bone simulant among overdenture bars with various levels of misfit, *J Prosthet Dent* 4: 243-250, 1995.
94. Bidez MW, McLoughlin SW, Chen Y et al: Finite element analysis (FEA) studies in 2.5 mm round bar design: the effects of bar length and material composition on bar failure, *J Oral Implantol* 18:122-128, 1992.
95. White S, Caputo AA, Anderkuist T: Effect of cantilever length on stress transfer by implant supported prostheses, *J Prosthet Dent* 71:493-499, 1994.
96. Osier JF: Biomechanical load analysis of cantilever implant systems, *J Oral Implantol* 17:40, 1991.
97. Hertel RC, Kalk W: Influence of the dimensions of implant superstructure on periimplant bone loss, *Int J Prosthodont* 6:18-24, 1993.
98. McAlarney ME, Stavropoulos DN: Determination of cantilever length: anterior posterior spread ratio assuming failure criteria to be the compromise of the prosthesis retaining screw prosthesis joint, *Int J Oral Maxillofac Implants* 11:331-339, 1995.
99. Rochade Carvalho W, Barboza E, Caula AL: Cement retained prostheses in implant dentistry: a clinical report, *J Prosthet Dent* 85:345-351, 2001.

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Chapter 15

The Completely Edentulous Mandible: Treatment Plans for Fixed Restorations

Carl E. Misch

Twenty million adults in the United States have completely edentulous mandibular arches.[1] Over the last 15 years, many of these patients have been treated with an implant overdenture, which is a vast improvement compared with a complete denture. Many of these same patients might have preferred a fixed prosthesis, but often for financial considerations they chose to have a removable prosthesis fabricated. Maxillary implant-supported removable prostheses may provide advantages over full-arch fixed restorations, such as upper lip support for esthetics and ease of daily maintenance of a removable prosthesis. However, unlike a maxillary denture, the labial flange of a mandibular overdenture rarely is required for esthetics. The laboratory and component costs for a hybrid fixed restoration are often similar to those for a fully implant-supported overdenture (RP-4). The chair time required to fabricate an overdenture and bar is similar to that for an implant-supported fixed prosthesis. Yet because dentures and partial dentures typically cost several times less than fixed restorations on teeth, the doctor often charges half the fee for an implant overdenture and bar compared with a fixed restoration. However, the laboratory fees, component fees, and chair time are similar for these two types of prostheses. If the fees for these two restorations were similar, many patients would opt for a fixed prosthesis (Figures 15-1 and 15-2).

COMPARING FIXED VERSUS REMOVABLE IMPLANT PROSTHESES

A fixed restoration provides the psychological advantage of acting and feeling similar to natural teeth, whereas an overdenture, even if fully implant supported, remains a removable prosthesis. In fact, a common remark heard from patients with fixed restorations is "these implant teeth are better than my own teeth," whereas comments related to implant overdentures are "these are better than my denture."

Removable implant overdentures require greater maintenance and exhibit more frequent prosthetic-related complications than fixed restorations. Walton and McEntee[2] noted that there were three times more maintenance and adjustments for removable prostheses compared with fixed restorations. Implant overdentures (IODs) often require attachments to be changed or modified every 6 months to 2 years, and denture teeth often wear, requiring a new prosthesis to be fabricated every 5 to 7 years.[2-7] In a review of literature by Goodacre et al., IODs have retention and adjustment problems 30% of the time, relines 19%, clip or attachment fracture 17%, and fracture of the prosthesis 12% of the time. Fixed prostheses need less repair and less maintenance and often last the life of the implant support. Although porcelain fractures with a fixed restoration may be costly to repair, over a lifetime, the implant-supported removable prosthesis may be more expensive.

A mandibular overdenture often traps food below its flanges, similar to a denture. Dentures are border-molded to the muscle attachment level to allow the floor of the mouth to raise during swallowing. As a consequence,

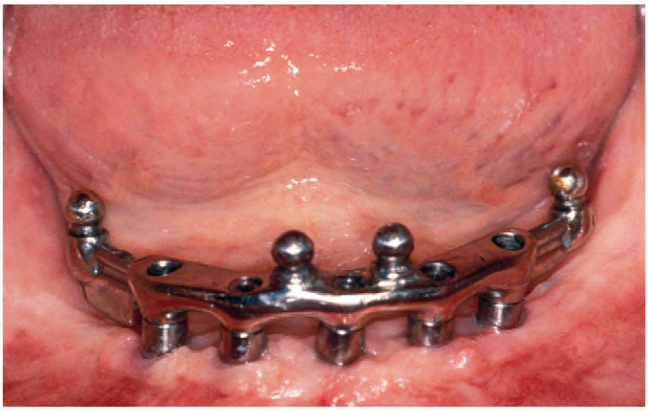


Figure 15-1 A mandibular implant overdenture may have complete implant support, retention, and stability from the implant support system (RP-4 prosthesis).

WWW.HIGHDENT.IR

Figure 15-2 A complete arch implant fixed prosthesis may be hybrid, with denture teeth and acrylic joined to a metal substructure.

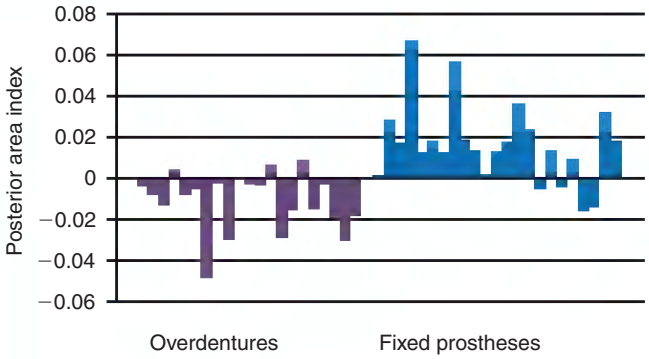


Figure 15-3 Implant overdentures with posterior soft tissue support lose bone in the posterior regions almost 75% of the time. Fixed prostheses cantilevered from anterior implants gain bone in the posterior regions more than 80% of the time (right side of graph).

Box 15-1 Advantages of a Fixed Partial Denture

- Psychological: "feels like teeth"
- Less prosthetic maintenance (e.g., attachments, relines, new overdenture)
- Less food entrapment
- Posterior mandibular bone gain

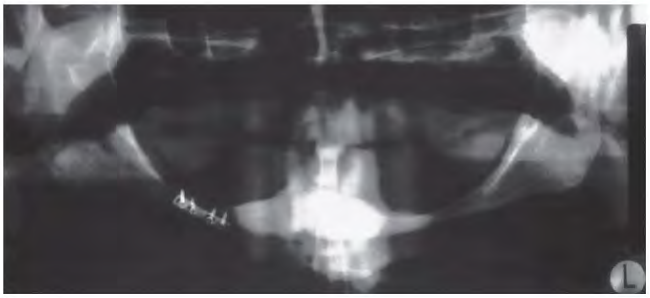


Figure 15-4 A panoramic radiograph of a severely resorbed mandible with a fracture and bone plate in the right body region. This patient has bilateral paresthesia of the lower lip.

food accumulates below the denture flange while the muscles are at rest and then is compressed under the restoration during deglutition. The contour of a fixed restoration is less prone to food entrapment (Box 15-1). The daily care for a bar implant overdenture (RP-4) is similar to that for a fixed mandibular restoration because ridge lap pontics are not required for esthetics or speech, as with some maxillary fixed prostheses.

A more recent clinical study by Wright et al. has evauated posterior mandibular bone loss in implant overdentures (RP-5) compared with cantilevered fixed prostheses from anterior implants.[8] The annual bone loss index observed in the RP-5 overdentures ranged from +0.02 to −0.05 with 14 of 20 patients losing bone in the posterior regions. On the other hand, the fixed prostheses group had a range from +0.07 to −0.015 with 18 of 22 patients gaining posterior bone area (Figure 15-3). Reddy et al. also found a similar clinical observation in 60 consecutively treated cantilevered fixed prostheses supported by five to six implants placed between the foarminae.[9] The mandibular body height was measured 5, 10, 15 and 20 mm distal to the last implant. The baseline measurements up to 4 years after function increased from 7.25 ± 0.25 mm to 8.18 ± 0.18 mm. Nearly all of the bone growth occurred during the first year of function. Therefore an important role for the presence of a complete implant-supported restoration is the maintenance and even regeneration of posterior bone in the mandible. This is especially important because posterior bone loss in this region may lead to paresthasia and even mandibular body fracture (Figure 15-4).

Too often, the dentist offers overdentures as the only option for edentulous patients, rather than including fixed treatment options. The advantages of a fixed restoration over an overdenture warrant most all edentulous patients to be given an option for a fixed prosthesis. This chapter discusses fixed treatment planning options for the completely edentulous mandibular arches.[10]

FORCE FACTORS

The amount of force transmitted to an implant-fixed prosthesis is similar to that of a completely implant-supported overdenture (RP-4). In one aspect then, the number of implants to support either prosthesis type should be similar. Mandibular overdentures may be removed at night to decrease the risk of nocturnal parafunctional overload. However, most mandibular edentulous patients also have an edentulous maxilla. Therefore if the patient is willing to remove the maxillary

WWW.HIGHDENT.IR

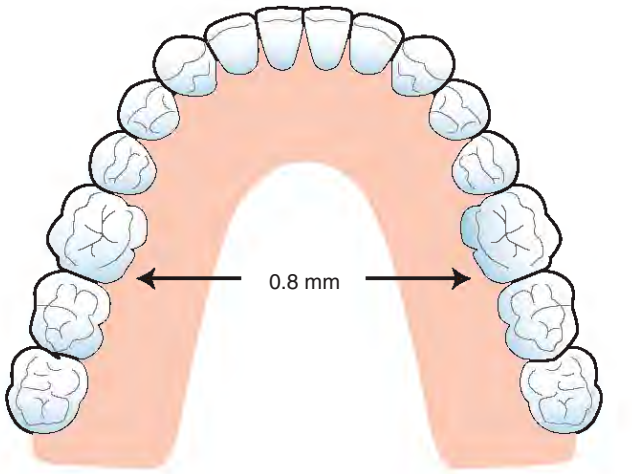


Figure 15-5 The flexure of the mandible during opening and protrusive movements occurs distal to the mental foramens. The amount of flexure depends on the amount of the bone volume and the sites in question. The medial movement from the first molar to the first molar region may be 800 µm.

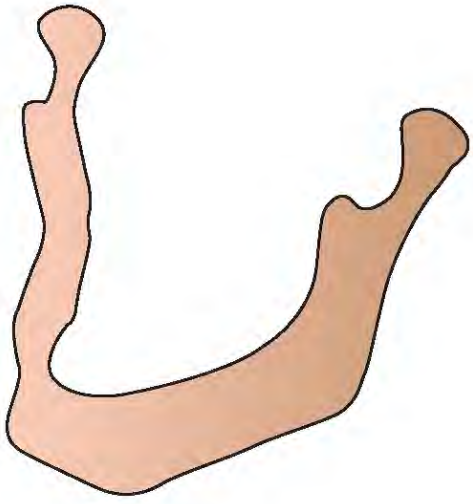


Figure 15-6 The mandible flexes toward the midline on opening or during protrusive movements as a result of the internal pterygoid muscle attachments on the ramus. The mandible also torques, with the inferior border rotating out and up and the crestal region rotating lingually. The movement is caused by the masseter muscles during forceful biting or parafunction.

denture at night, the risk of nocturnal parafunction also may be eliminated for patients with mandibular fixed prostheses. As a consequence, the number of implants required to restore a fixed prosthesis may be similar to a fully implant-supported overdenture.

When the patient has natural teeth and/or implants in the maxilla, more implants usually are indicated for the mandibular fixed prosthesis to reduce the risk of occlusal overload, or a reduction in cantilever length is necessary. Force factors such as parafunction, crown height, masticatory dynamics, and the bone density of the implanted regions also should modify the implant position, implant number, size, and design. Increased force factors contribute to uncemented restorations, screw loosening, component fracture, and crestal bone loss. As a result, the fixed prosthesis often may require an improved biomechanical position compared with an implant overdenture support system.

MANDIBULAR DYNAMICS

Medial Movement

Many reports have addressed the dimensional changes of the mandible during jaw activity as a result of masticatory muscle action.[11-19] Five different movements have been postulated.[18] Medial convergence is the one most commonly addressed.[19] The mandible between the mental foraminae is stable relative to flexure and torsion. However, distal to the foraminae, the mandible exhibits considerable movement toward the midline on opening.[15-16] This movement is caused primarily by the attachment of internal pterygoid muscles on the medial ramus of the mandible. The distortion of the mandible occurs early in the opening cycle, and the maximum changes may occur with as little as 28% opening, or about 12 mm of mandibular movement.[11] This flexure has also been observed during protrusive jaw movements.[20,21] The greater the active opening and protrusive movements, the greater the amplitude. The amount of movement varies among individuals and depends on the density and volume of bone and the location of the site in question. The amplitude of the mandibular body flexure toward the midline has been measured to be as much as 800 µm in the first molar to the first molar region to as much as 1500 µm in the ramus-to-ramus sites (Figure 15-5). In a study by Hobkirk on deformation of the mandible in subjects with fixed dental implant prostheses, medial covergence up to 41 µm was observed.[20]

Torsion

Torsion of the mandibular body distal to the foraminae has also been documented in animal and human studies.[22-26] Hylander evaluated larger members of the rhesus monkey family (macaque) and found the mandible twisted on the working side and bent in the parasagittal plane on the balancing side during the power stroke of mastication and unilateral molar biting.[25] Parasagittal bending of the human jaw during unilateral biting was confirmed by Marx,[26] who measured localized mandibular distortion in vivo in humans by using strain gauges on screws attached to cortical bone in the symphyseal and gonial regions. Hobkirk et al. confirmed the mandible of patients with implant prostheses measured up to 19 degrees of dorsoventral shear.[22] The torsion during parafunction is caused primarily by forceful contraction of the masseter muscle attachments (Figure 15-6). Therefore parafunctional bruxism and clenching may cause

WWW.HIGHDENT.IR

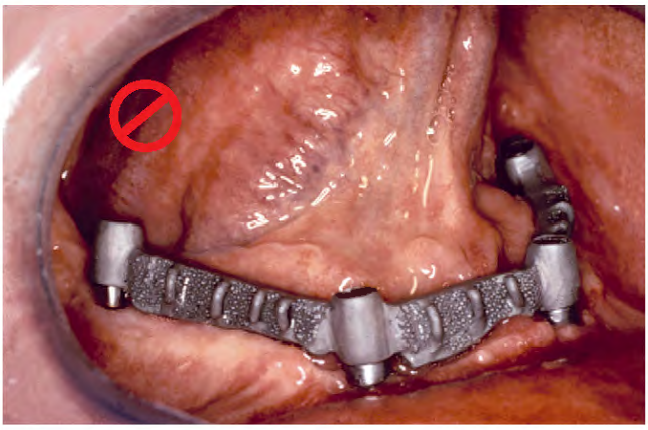


Figure 15-7 Some authors propose that the ideal implant positions to support a mandibular full-arch prosthesis are the bilateral molars and bilateral canines, splinted together with a rigid structure. These positions are not ideal because of the mandibular dynamics during opening and function.

problems in the implant support system and prosthesis when the mandibular teeth are splinted from molar to molar.

The posterior bone gain in edentulous patients restored with cantilevered prostheses from anterior implants may be a consequence of the mandibular flexure and torsion. Because the bite force may increase 300% with an implant prosthesis compared with a denture, the increased torsion may stimulate the posterior mandibular body to increase in size, as reported by Reddy et al.[9] and Wright et al.[8]

The most common position of the mental foramen is between the first and second premolar teeth; therefore when splinting teeth distal to the bilateral premolar position, mandibular dynamics should be considered.[27] A study by Miyamoto et al. identified jaw flexure as the primary cause of posterior implant loss in full-arch mandibular prostheses.[28] The more distal the rigid splint from one side to the other, the greater the risk that mandibular dynamics may influence the implants or prosthesis prognosis. In addition, the body of the mandible flexes more when the size of the bone decreases. As a result, the Division C minus height (C–h) or D mandible flexes or exhibits torsion more than the Division A mandible, all other factors being similar.

As a consequence, posterior rigid, fixated implants splinted to each other in a full-arch restoration are subject to a considerable buccolingual force on opening and during parafunction.[27-29] The difference in movement between an implant and a tooth has been addressed as a concern for dentists. The natural tooth movement ranges from 28 µm apically and 56 to 108 µm laterally. In contrast, the rigid implant has movement up to 5 µm apically and 10 to 60 µm laterally, yet the mandibular flexure and torsion may be more than 10 to 20 times the movement of a healthy tooth. Therefore the flexure and torsion of the mandibular body are more critical in the patient evaluation compared with whether an implant should be joined to the natural dentition.

Past authors have suggested four implants in the mandible with a full-arch splinted fixed restoration—two in the first molars and two in the canine regions[30] (Figure 15-7). Additional implants have been used with this full-arch splinted restorative option, with up to four other implants in the premolar and the incisor regions.[31] However, complete cross-arch splinting of posterior, molar rigid, fixated implants should be reconsidered in the mandible. The flexure of the mandible is thwarted by the prosthesis,[22,27,29,32-34] but this introduces lateral stresses to the implants. These implant positions place the molar implants, screws, and bone at increased risk because of the mandibular flexure and torsion previously addressed.

In complete mandibular subperiosteal implants, pain upon opening was noted in 25% of the patients at the suture removal appointment when a rigid bar connected molar-to-molar regions. When the connecting bar was cut into two sections between the foraminae, the pain upon opening was eliminated immediately. This clinical observation does not mean the other 75% of patients did not have flexure of the mandibular arch upon opening. The observation does demonstrate, however, that flexure may be relevant to postoperative complications. Consequences of a treatment plan with cross-arch connection of posterior mandibular implants may include bone loss around the implants, loss of implant fixation, material fracture (implant or prosthesis components), unretained restorations, and discomfort upon opening. Until clinical studies become available and state otherwise, full-arch splinted restorations joining bilateral molar implants in the mandible should not be a treatment of choice.

Implants placed in front of the foraminae and splinted together, or implants in one posterior quadrant joined to anterior implants, have not shown these complications related to the flexure or torsion of the mandible. Complete implant-supported fixed restorations can halt the posterior bone loss associated with edentulism, improve psychological health, and produce fewer prosthetic complications compared with removable restorations. Therefore all edentulous mandibular patients should be given the option of having a fixed prosthesis. There are five treatment options used to restore a complete edentulous mandible with a fixed prosthesis. These implant position options also may be considered for implant-supported overdentures.

When a mandibular overdenture is completely implant supported and retained and stabilized by a cantilevered bar, it acts similarly to a fixed prosthesis in function and bone maintenance. Therefore the five treatment options included in this chapter may be used for either a RP-4 overdenture or a fixed prosthesis (see Figures 15-1 and 15-2).

WWW.HIGHDENT.IR

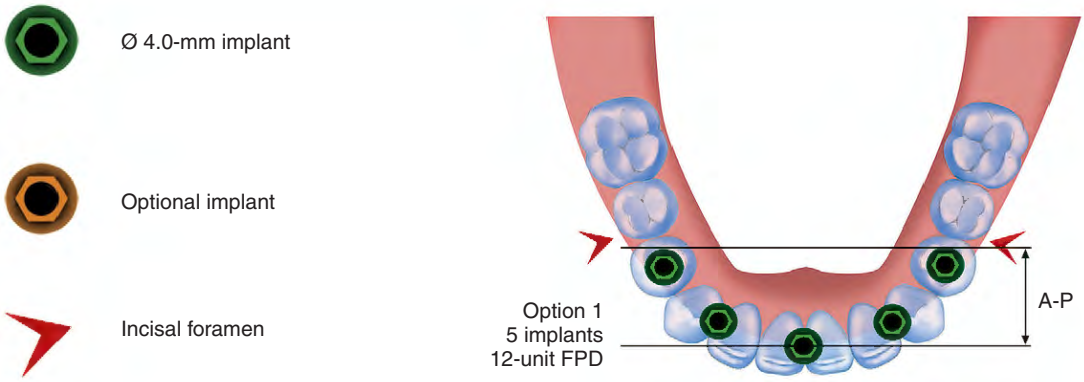


Figure 15-8 The most common number of implants between the foramens for option 1 is five. These implants provide as great an anteroposterior (A-P) distance as possible between the foraminae with sufficient interimplant spacing for treatment of complications. *FPD,* Fixed partial denture.

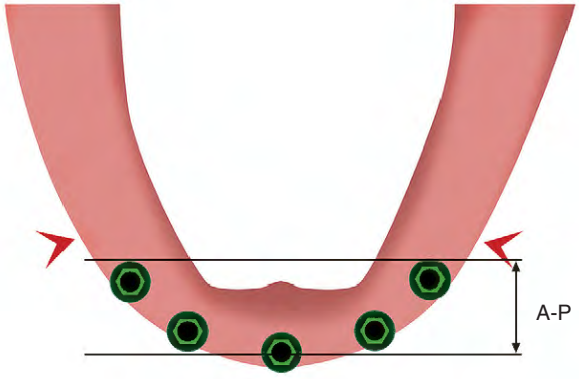


Figure 15-9 The anteroposterior (A-P) distance is determined by a line drawn from the distal portion of the distalmost implant on each side of the arch and another parallel line drawn through the center of the anteriormost implant from the cantilever.

Figure 15-10 A full-arch mandibular implant prosthesis with five implants placed between the mental foraminae and a hybrid fixed prosthesis cantilevered to the first molar region.

IMPLANT TREATMENT OPTIONS FOR FIXED RESTORATIONS

Treatment Option 1: The Brånemark Approach

The mandible does not flex or exhibit significant torsion between the mental foraminae. Therefore anterior implants may be splinted together without risk or compromise. The placement of four or six anterior root forms between the mental foraminae and a distal cantilever off each side to replace the posterior teeth was the treatment of choice for clinical reports from 1967 to 1981 with the Brånemark system[35] (Figure 15-8). This treatment approach resulted in an 80% to 90% implant survival for 5 to 12 years after the first year of loading. In a long-term, 18- to 23-year study, Attard and Zarb[36] reported an 84% success rate for prostheses and implants, respectively. The range of survival may be due to the broad application of the modality, regardless of crown height, opposing dentition, implant length, anteroposterior (A-P) position of implants, and parafunction.

The arch form and the position of the mental foraminae are important criteria when four to six implants are placed only in the anterior segment to replace the entire mandibular arch. The anterior arch form and foraminae position affects the position of the distalmost implants and the anterior arch form (square, oval, or tapering) is relative to the anteriormost implant position. The distance from the center of the most anterior implant to a line joining the distal aspect of the two most distal implants on each side is called the *A-P distance* or the *A-P spread*[37] (Figure 15-9). The greater the A-P spread, the further the distal cantilever may be extended to replace the missing posterior teeth.

The most common number of implants used today in the Brånemark treatment option is five (Figure 15-10). This number allows as great an A-P spread as six implants, with greater interimplant distance so that if bone loss occurs on one implant, the loss would not automatically affect the adjacent implant site. However, the A-P spread is only one of the force factors to be considered for the extent of the distal cantilever. As a general rule, when five anterior implants are placed in the anterior mandible between the foraminae to support a fixed prosthesis, the cantilever should not

WWW.HIGHDENT.IR

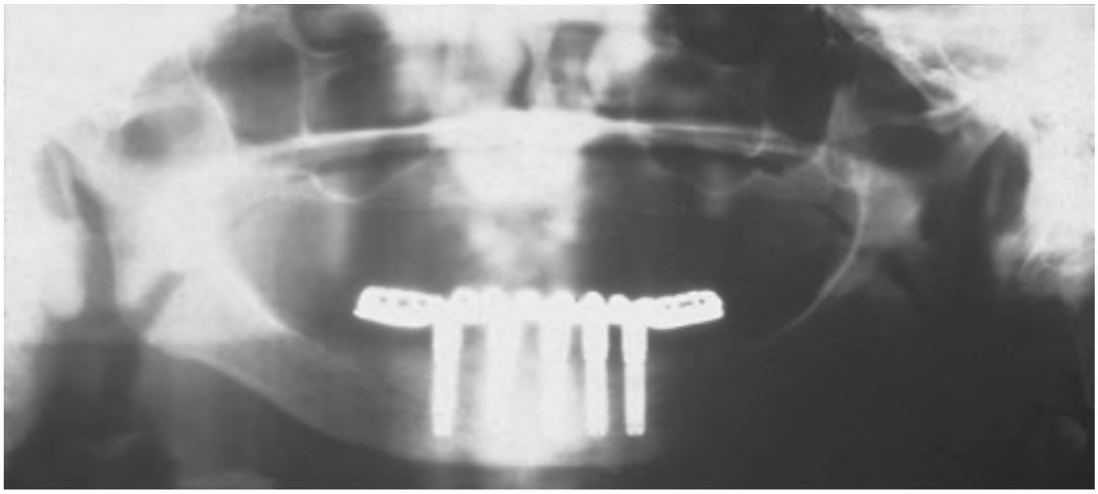


Figure 15-11 A panoramic radiograph of a hybrid full-arch fixed prosthesis cantilevered from five anterior endosteal implants positioned between the mental foraminae.

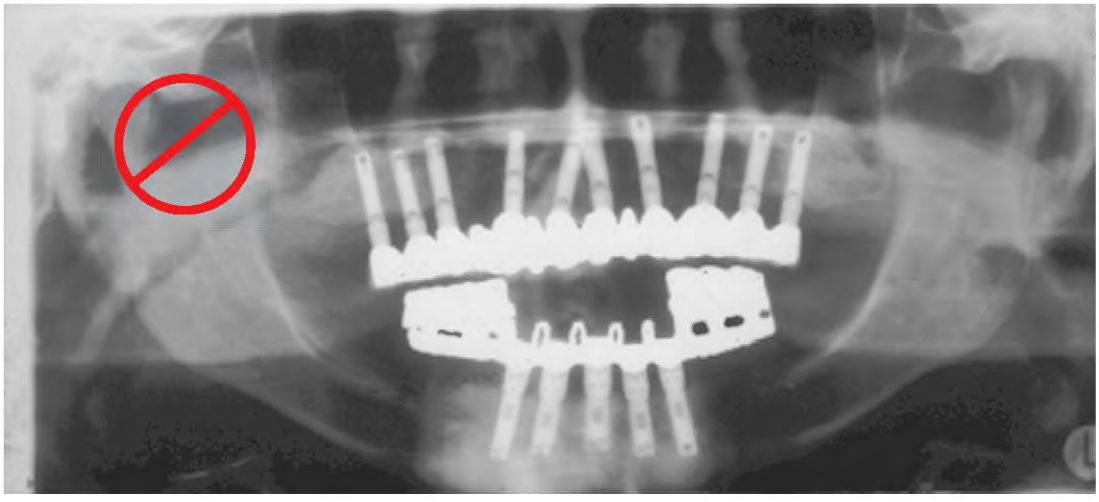


Figure 15-12 The length of the distal cantilever should be reduced when opposing an implant prosthesis or when the crown height space is more than 15 mm. A decrease in occlusal awareness causes high bite forces in implant prostheses, and crown height is a force magnifier in the presence of a cantilever. The cantilever in this radiograph should be reduced to decrease the risk of stress-related complications.

exceed 2.5 times the A-P spread, with all other stress factors being low (Figure 15-11). If the stress factors are high (e.g., parafunction, crown height, masticatory musculature dynamics, opposing arch), cantilevering of a prosthesis may be contraindicated. Therefore the length of the posterior cantilever depends on the specific force factors of the patient, of which A-P spread is only one (Figure 15-12).

Stress equals *force* divided by the *area* over which *force* is applied. The area over which the forces are applied from the prosthesis to the implants can be modified through the number, size, and design of the implants. A cantilever rarely is indicated on three implants, even with a similar A-P spread as five implants. The cantilever can be greater for five implants because of greater implant surface area and greater number of prosthetic components to decrease screw loosening. Often, narrow implants are not designed to support cantilevers, whereas wider implants can support a greater cantilever.

Treatment option 1 depends greatly on patient force factors, arch form, and the number, size, and design of the implants. As a result, the safest action is to reserve this option for patients with low force factors such as an older female wearing an upper denture, with abundant anterior bone, crown height inferior to 15 mm, with a tapered or ovoid mandibular arch, but with posterior segments of inadequate height for endosteal implant placement.

Treatment Option 2

Bidez and the University of Alabama at Birmingham, School of Engineering in the Department of Biomechanics, have evaluated dentate and edentulous mandibles and

WWW.HIGHDENT.IR

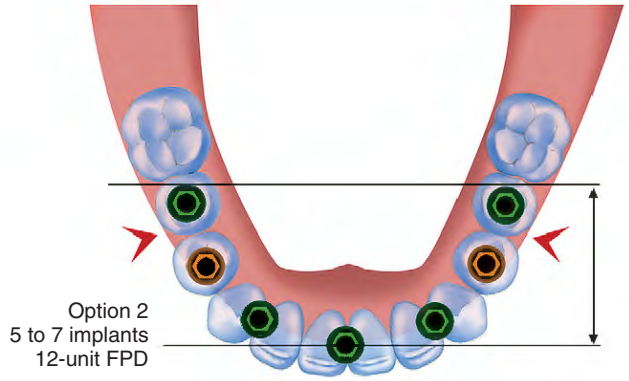


Figure 15-13 Treatment option 2 has five key implant positions: two implants placed over the mental foraminae, two implants in the canine positions, and one implant in the midline. Secondary implants may be positioned in the first premolar sites. *FPD,* Fixed partial denture.

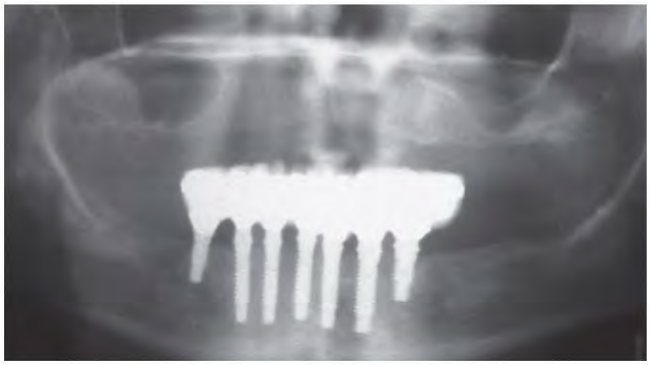


Figure 15-14 A panoramic radiograph of a full-arch fixed restoration with seven implants placed following treatment option 2: two implants over the foraminae, two implants in the first premolar sites, two implants in the canine positions, and one implant in the midline.

developed a bone strain model of flexure and torsion. As a consequence, a number of implant site options have become available.

A slight variation of the ad modum Brånemark protocol is to place additional implants above the mental foraminae, because the mandible flexes distal to the foramen (Figure 15-13). An implant above one or both foraminae presents several advantages. First, the number of implants may be increased to as many as seven (which increases implant surface area). Second, the A-P spread for implant placement is greatly increased, even when the total implant number is five. This implant position reduces the Class 1 lever forces generated from the distal cantilever. Third, the length of the cantilever is reduced dramatically because the distalmost implant is placed one tooth more distal (Figures 15-14 and 15-15).

A prerequisite for treatment option 2 is the presence of available bone in height and width over the foraminae. Because the foramen usually is located 12 mm above the inferior border of the mandible, available bone height is reduced in this location. When available, the foramen often requires implants of reduced height

Figure 15-15 An intraoral view of the full arch fixed prosthesis from Figure 15-14. A cantilever to the first molar site is reduced, because implants are positioned over the mental foraminae.

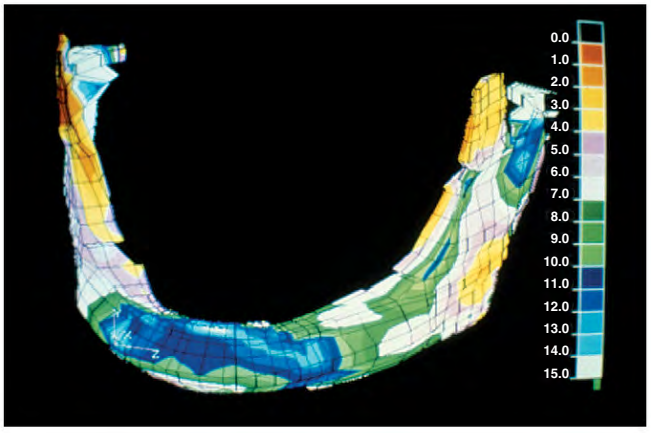


Figure 15-16 Studies at the Engineering school at the University of Alabama at Birmingham used a dentate and edentulous model to evaluate the bone strain conditions during flexure and torsion. A number of implant site options have become available as a consequence. *(Courtesy M.W. Bidez.)*

compared with the anterior implants. The most distal implant bears the greatest load when loads are placed on the cantilever (acts as fulcrum); therefore the greatest forces are generated on the shortest implants. A minimum recommended implant height of 9 mm and a greater diameter or an enhanced surface area design are recommended to compensate for the reduced length.

The key implant positions in this option are the second premolar positions, the canine positions, and the central incisor or midline position. The two optional implant sites are the first premolar sites.

Treatment Option 3

The Bidez strain model of an edentulous mandible indicated implants in one posterior section may be splinted to anterior implants without compromise (Figure 15-16). The author has evaluated full-arch fixed prostheses on implants with one posterior segment connected to the anterior region over the last decade

WWW.HIGHDENT.IR

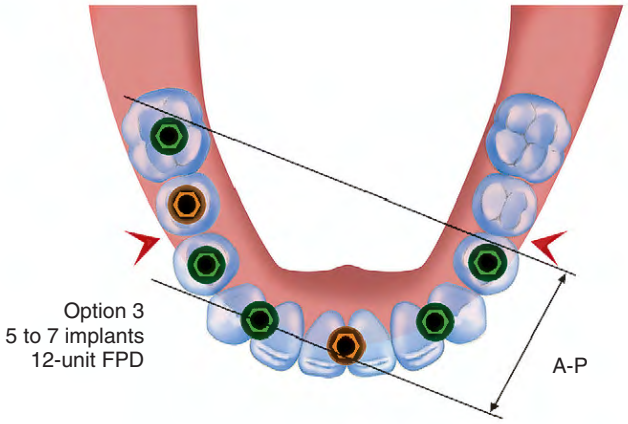


Figure 15-17 Treatment option 3 has key implant positions in one first molar site, bilateral first premolar positions, and two canine sites. Secondary implants may be used in the bilateral second premolar and midline position. The anteroposterior (A-P) distance is measured from the two distal most implants to the anteriormost implant from the cantilever. *FPD,* Fixed partial denture.

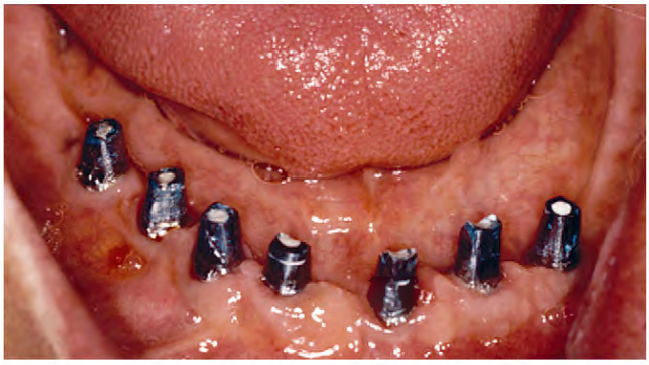


Figure 15-18 Five of the seven mandibular implants are positioned between the mental foramens, and two are placed on the patient's right side. The placement increases the anteroposterior distance and eliminates the prosthetic cantilever on the patient's left side.

and has found no additional complications experienced during this time frame compared with those with independent segments. Therefore another treatment plan option to support a fixed mandibular prosthesis consists of additional implants in the first molar or second premolar position, connected to four or five implants between the mental foraminae. Hence a total of five to seven implants usually are placed in this option.

The key implant positions for treatment option 3 are the first molar (on one side only), the bilateral premolar positions, and the bilateral canine sites. The secondary implant positions include the second premolar position on the same side as the molar implant and the central incisor (midline) position (Figure 15-17). On occasion, an additional site may include the position over the mental foramen on the side of the cantilever. A one-piece casting can be fabricated, and one cantilever to the opposite side of the molar implant would replace those posterior teeth. Although mandibular movement occurs, it has not been observed to cause complications.

Figure 15-19 A mandibular restoration in situ.

Figure 15-20 An intraoral view of the final maxillary and mandibular fixed implant-supported prostheses.

Treatment option 3 is a better option than anterior implants with bilateral cantilevers for several reasons. When one or two implants are placed distal to the foraminae on one side and are joined to anterior implants between the foraminae, a considerable biomechanical advantage is gained. Although the number of implants may be the same as option 1 or 2, the A-P spread is 1.5 to 2 times greater, because on one side the distal aspect of the last implant now corresponds to the distal aspect of the first molar (see Figure 15-17). In addition, only one cantilever is present, rather than bilateral cantilevers. When force factors are greater, six to seven implants may be used for this option. Five implants between the foraminae and one or two implants distal on one side compose the usual placement (Figures 15-18 to 15-21). Over the last 10 years, the author has fabricated more than 25 prostheses with five to seven implants in these locations. To date, no prosthesis has been replaced and no implants have failed. This approach is superior to treatment option 2 with bilateral cantilevers because the A-P spread is dramatically increased, more implants may be used if desired, and only one side has a cantilever. However, this option requires available

WWW.HIGHDENT.IR

Figure 15-21 A panoramic radiograph of the final maxillary and mandibular restorations (same patient as Figures 15-17 to 15-20). The restoration in the mandible has used treatment option 3 to support the full-arch prosthesis.

Figure 15-22 A lateral cephalometric radiograph 6 months after an iliac crest bone graft to the mandible has augmented the arch for bone height gains of 15 mm in the anterior regions and 20 mm in the posterior regions.

bone in at least one posterior region of the mandible (Figures 15-22 to 15-25).

Treatment Option 4

Treatment plan options for fixed full-arch prostheses also may include bilateral posterior implants as long as they are not splinted together in one prosthesis. This option is selected when force factors are great or the bone density is poor. Poor bone quality most often is observed in the posterior maxilla, but on occasion it is also found in the mandible. This option is also used when the body of the mandible is Division C–h and subperiosteal or disk-design implants are used for posterior implant support. Several options for fixed

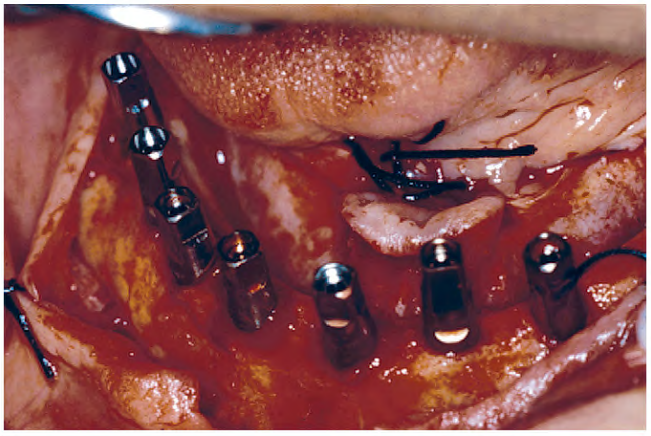


Figure 15-23 Two implants are positioned behind the foramen on the patient's right side, and one implant is added over the foramen on the patient's left side. The anteroposterior distance is increased more than four times compared with option 1, and the prosthetic cantilever has been decreased on the patient's left side.

Figure 15-24 The final mandibular restoration in situ opposing a maxillary full denture. The patient is in centric relation occlusion.

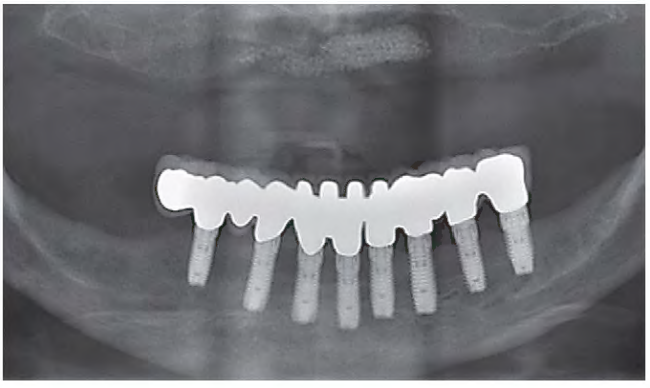


Figure 15-25 A panoramic radiograph 3 years after delivery of the mandibular fixed prosthesis. The iliac crest bone graft has matured, and the bone volume has been maintained.

restorations are available when bilateral posterior implants are included.

In treatment option 4, implants are placed in all three segments of the mandible (Figure 15-26). Key

WWW.HIGHDENT.IR

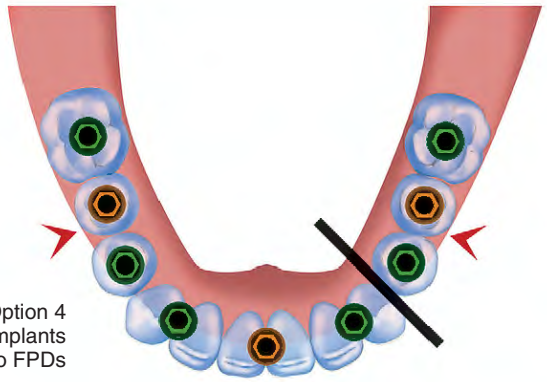


Figure 15-26 Treatment option 4 has implants in both molar sites. Other key positions include the two first premolar positions and the two canine sites. Secondary implants may also be positioned into second premolar locations and the midline. *FPD*, Fixed partial denture.

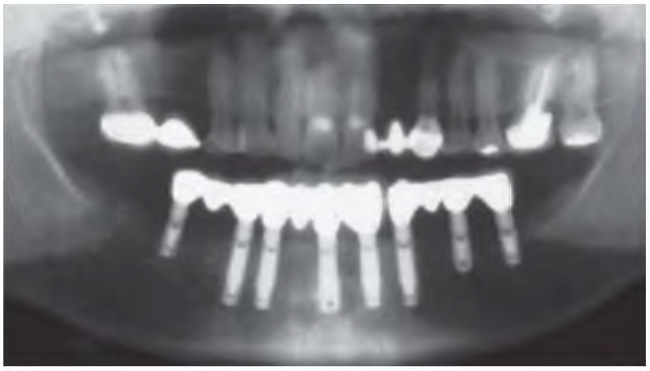


Figure 15-28 A panoramic radiograph of a full-arch fixed implant prosthesis in the mandible, with option 4 implant support. The prosthesis is separated in two sections between the first premolar and canine sites.

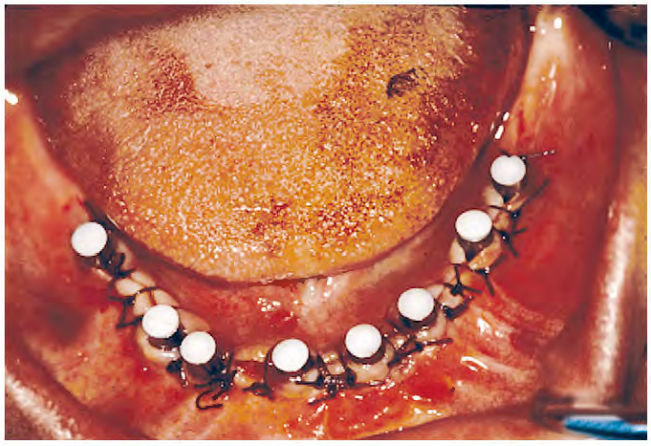


Figure 15-27 Eight endosteal implants at stage 2 uncovery with both posterior quadrants of sufficient bone height and width for endosteal implants.

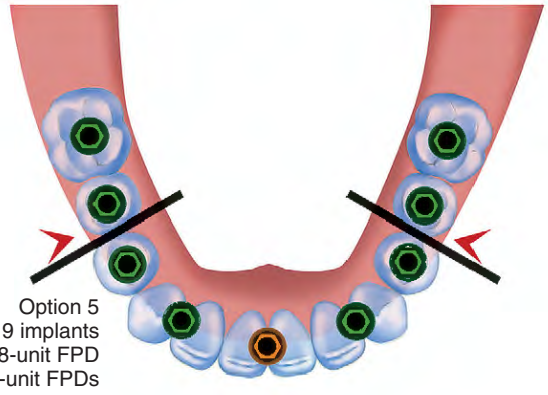


Figure 15-29 Treatment option 5 is designed to support three independent prostheses. The key implant positions are usually first molar sites, second premolar sites, first premolar sites, and canine positions. The posterior restorations are usually separated between two premolar sites. This option provides maximum flexure and torsion to the mandibular body during function and parafunction. *FPD*, Fixed partial denture.

implant positions for this treatment option include the two first molars, two first premolars and two canine sites. Secondary implants may be added in the second premolars and/or the incisor (midline) position. All implants in the anterior and one posterior side are splinted together for a nine-unit, fixed prosthesis. The other posterior segment is restored independently with an independent three-unit, fixed prosthesis supported by implants in the first premolar and first molar region as the key positions. The secondary sites are the second premolar positions (Figures 15-27 and 15-28). Three implants are used most often for the smaller segment to compensate for force factors and the alignment of the implants almost in a straight line. At least six implants typically are used in this option. Additional implants—as many as nine—may be inserted when force factors are greater.

The primary advantage of this treatment option is the elimination of cantilevers. As a result, risks of uncemented restorations and occlusal overload are reduced. Another advantage is that the prosthesis has two segments rather than one. Because no cantilever is present, weaker cements can be used to install the prosthesis. If the prosthesis requires repair, the affected segment may be removed more easily because only the segment requiring repair needs to be removed. Disadvantages include the need for abundant bone in both mandibular posterior regions and the additional costs incurred for one to four additional implants. The restoration should exhibit posterior disclusion in excursions to limit lateral loads, especially to the prosthesis supported by fewer implants.

Treatment Option 5

Another modification for the completely edentulous mandible is to fabricate three independent prostheses rather than one or two (Figure 15-29). The anterior region of the mandible may have four to five implants. The key implants are in the two first molar sites, the two first premolar, and two canine regions. Secondary positions are the two second premolar and central

WWW.HIGHDENT.IR

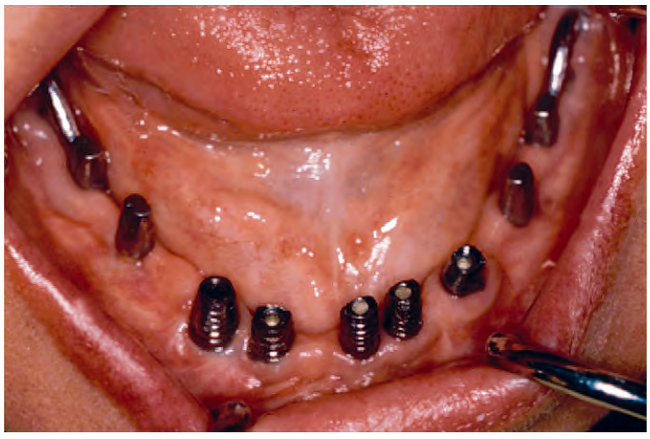


Figure 15-30 An intraoral view of five root form implants positioned between the mental foraminae. The posterior quadrants have a subperiosteal implant. This female patient desired maxillary and mandibular fixed prostheses, but declined an iliac crest bone graft to the posterior mandible.

Figure 15-31 The anterior fixed prosthesis is independent of the posterior restorations. This allows maximum flexure of the mandible. This fifth option is more indicated for Division C–h bone volume mandibles than Division A mandibles.

incisor (midline) sites. With this setup, the posterior restorations extend from first molar to first premolar and an anterior restoration replaces the six anterior teeth. However, these six implant sites are usually best used in treatment option 4. Therefore, when option 5 is indicated, it usually has two implants in first molars, second premolars, first premolars, and both canine positions. These eight implants may also have a secondary implant in the midline. The fixed anterior prosthesis extends from first premolar to first premolar. The posterior restorations are two independent implant prostheses, each with two units.

The advantages of this option are smaller segments for individual restorations in case one should fracture or become uncemented. In addition, if greater mandibular body movement is expected because of parafunction or a decrease in size of the body of the mandible, the independent restorations allow the most flexibility and torsion of the mandible. The primary disadvantage is

Figure 15-32 An intraoral view of the mandibular restorations. Two independent posterior restorations are supported by subperiosteal implants between the mental foraminae.

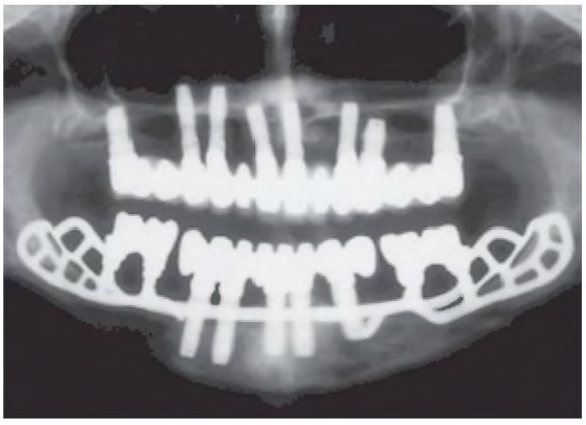


Figure 15-33 A panoramic radiograph of the final restorations and implants. The three independent mandibular prostheses permit flexure of the posterior mandible.

the greater number of implants required. In addition, the available bone needs are greatest with this treatment option. Rarely are more than nine implants required to replace the lower teeth, regardless of the bone density or force factors present. Option 5 is the treatment of choice when force factors are severe, but it is rarely used.

The most common scenario for option 5 is when the posterior mandible is C–h bone volume and a circumferential subperiosteal or disk-design implant is used as the second premolar and first molar implant abutment supports. The decrease in the bone volume of the posterior mandible increases the flexure and torsion. As a result, three independent prostheses are warranted (Figures 15-30 to 15-33).

LONG-TERM TREATMENT PLANNING

There is overwhelming evidence and agreement that a two-implant overdenture (RP-5) is a better option than a traditional denture, but the overdenture should not be considered a lifetime device. The posterior mandibular

WWW.HIGHDENT.IR

resorbs four times faster than the anterior mandible. An anterior implant-supported overdenture may accelerate posterior bone loss, because the bite force increases and the patient more likely is able to wear the mandibular prosthesis. When the doctor and patient consider a "lifetime strategy of treatment" rather than a 1-year treatment plan, the plan is then treated as a lifetime approach and both doctor and patient can benefit.

A mandibular overdenture may be upgraded from an RP-5 prosthesis to a completely implant-supported device (RP-4) and then to a fixed prosthesis (FP-3). If the cost involved to insert two or three implants can be invested every 5 years, within 4 to 20 years every patient may have a fixed restoration, provided available bone and force factors are compatible. As a result, the goal of the dentist should be to establish a lifetime strategy of health, with the patient's scheduled prosthesis upgrade. For example, if implants are placed in the canine positions at step 1 for an overdenture, the dentist may plan the placement of implants into the left first molar and right first premolar positions at step 2. At step 3, implants can be added to the left first premolar position to reach a goal of an RP-4 or FP-3 restoration.

SUMMARY

Many completely edentulous patients desire a fixed restoration rather than a removable prosthesis. Costs for a fixed implant prosthesis often have been a deterrent, but should be more similar to a completely implant-supported overdenture. The number and position of implants should be related to the amount of stress transmitted to the bone during occlusion and parafunction and the density of the bone. Other considerations include mandibular flexure and torsion. Five treatment options generally are available for this fixed complete mandibular implant-supported restoration. The primary advantage of treatment option 1 with five anterior implants is cost. Disadvantages include overload situations resulting from bilateral cantilevers. Treatment option 2 is improved, but bilateral cantilevers (which are shorter than option 1) are still a concern. Treatment option 3 is superior to the previous option but requires posterior bone in one quadrant to place endosteal, subperiosteal, or disk-design implants. The most ideal treatments are options 4 or 5 because they lack cantilevers and the dentist fabricates two or three separate restorations. These treatment options also accommodate the stronger mandibular bone dynamics without affecting the restoration. However, bone must be present or obtained through grafting for endosteal implants, or a circumferential subperiosteal or disk-design implant must be used for support in both posterior quadrants. In addition, the latter options increase costs because more implants are used for support of the fixed restoration.

References

1. Marcus SE, Drury JF, Brown LS et al: Tooth retention and tooth loss in the permanent dentition of adults: United States, 1988-1991, *J Dent Res* 75(special issue):684-695, 1996.
2. Walton JN, McEntee MI: Problems with prostheses on implants: a retrospective study, *J Prosthet Dent* 71:283-288, 1994.
3. Watson RM, Davis DM: Follow up and maintenance of implant supported prostheses: a comparison of 20 complete mandibular overdentures and 20 complete mandibular fixed cantilever prostheses, *Br Dent J* 181:321-327, 1996.
4. Payne AG, Solomons YF: Mandibular implant-supported overdentures: a prospective evaluation of the burden of prosthodontic maintenance with 3 different attachment systems, *Int J Prosthodont* 13:246-253, 2000.
5. Watson RM, Jemt T, Chai J et al: Prosthodontics treatment, patient response, and the need for maintenance of complete implant-supported overdentures: an appraisal of 5 years of prospective study, *Int J Prosthodont* 10: 345-354, 1997.
6. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implant and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
7. Dudic A, Mericske-Stern R: Retention mechanisms and prosthetic complications of implant-supported mandibular overdentures: long-term results, *Clin Implant Dent Relat Res* 4:212-219, 2002.
8. Wright PS, Glastz PO, Randow K et al: The effects of fixed and removable implant-stabilized prostheses on postrior mandibular residual ridge resorption, *Clin Oral Impl Res* 13:169-174, 2002.
9. Reddy MS, Geurs NC, Wang IC et al: Mandibular growth following implant restoration: does Wolff's Law apply to residual ridge resorption? *Int J Periodontics Restorative Dent* 22:315-321, 2002.
10. Misch CE: Treatment options for mandibular full arch implant-supported fixed prostheses, *Dent Today* 20:68-73, 2001.
11. De Marco TJ, Paine S: Mandibular dimensional change, *J Prosthet Dent* 31:482-485, 1974.
12. Fischman B: The rotational aspect of mandibular flexure, *J Prosthet Dent* 64:483-485, 1990.
13. Goodkind RJ, Heringlake CB: Mandibular flexure in opening and closing movement, *J Prosthet Dent* 30:134-138, 1973.
14. Grant AA: Some aspects of mandibular movement: acceleration and horizontal distortion, *Ann Acad Med Singapore* 15:305-310, 1986.
15. Osborne J, Tomlin HR: Medial convergence of the mandible, *Br Dent J* 117:112-114, 1964.
16. Regli CP, Kelly EK: The phenomenon of decreased mandibular arch width in opening movement, *J Prosthet Dent* 17:49-53, 1967.
17. Gates GN, Nicholls JI: Evaluation of mandibular arch width change, *J Prosthet Dent* 46:385-392, 1981.
18. Hylander WL: Stress and strain in the mandibular symphysis of primates: a test of competing hypotheses, *Am J Phys Anthropol* 64:1-46, 1984.
19. Hylander WL: The human mandible: lever or link? *Am J Phys Anthropol* 43:227-242, 1975.
20. McDowell JA, Regli CP: A quantitative analysis of the decrease in width of the mandibular arch during forced movements of the mandible, *J Dent Res* 40:1183-1185, 1961.
21. Burch JG: Patterns of change in human mandibular arch width during jaw excursion, *Arch Oral Biol* 17:623-631, 1972.

WWW.HIGHDENT.IR

22. Abdel-Latif HH, Hobkirk JA, Kelleway JP: Functional mandibular deformation in edentulous subjects treated with dental implants, *Int J Prosthodont* 13:513-519, 2000.
23. Omar R, Wise MD: Mandibular flexure associated with muscle force applied in the retruded axis position, *J Oral Rehabil* 8:209-221, 1981.
24. Picton DCA: Distortion of the jaws during biting, *Arch Oral Biol* 7:573-580, 1962.
25. Hylander WL: Mandibular function in Galago crassicaudatus and Macaca fascicularis: an in vivo approach to stress analysis of the mandible, *J Morphol* 159:253-296, 1979.
26. Marx H: Untersuchungen des funktionsbedingten elastischen Deformierung der menschlichen Mandibula, *Dtsch Zahnarztl Z* 21:937-938, 1966.
27. Hobkirk JA, Havthoulas TK: The influence of mandibular deformation, implant numbers, and loading position on detected forces in abutments supporting fixed implant superstructures, *J Prosthet Dent* 80:169-174, 1998.
28. Miyamoto Y, Fujisawa K, Takechi M et al: Effect of the additional installation of implants in the posterior region on the prognosis of treatment in the edentulous mandibular jaw, *Clin Oral Implants Res* 14:727-733, 2003.
29. Zarone F, Apicell A, Nicolais L et al: Mandibular flexure and stress build-up in mandibular full-arch fixed prostheses supported by osseointegrated implants, *Clin Oral Implants Res* 14:103-114, 2003.
30. Parel SM, Sullivan D: Full arch edentulous ceramometal restoration. In Parel SM, Sullivan D, editors: *Esthetics and osseointegration*, Dallas, Tex, 1989, Osseointegration Seminars.
31. Balshi TJ: Opportunity to prevent or resolve implant complications, *Implant Soc* 1:6-9, 1990.
32. Fishman BM: The influence of fixed splints on mandibular flexure, *J Prosthet Dent* 35:643-667, 1976.
33. de Oliveria RM, Emtiaz S: Mandibular flexure and dental implants: a case report, *Implant Dent* 9:90-95, 2000.
34. Paez CY, Barco T, Roushdy S et al: Split-frame implant prosthesis designed to compensate for mandibular flexure: a clinical report, *J Prosthet Dent* 89:341-343, 2003.
35. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 10:387-416, 1981.
36. Attard NJ, Zarb GA: Long-term treatment outcomes in edentulous patients with implant-fixed prostheses: the Toronto study, *Int J Prosthodont* 17:417-424, 2004.
37. English CE: The mandibular overdenture supported by implants in the anterior symphysis: a prescription for implant placement and bar prosthesis design, *Dent Implantol Update* 4:9-14, 1993.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 16

Single-Tooth Replacement: Treatment Options

Carl E. Misch

Seventy percent of the dentate population in the United States is missing at least one tooth. Single-tooth replacement will most likely comprise a larger percentage of prosthetic dentistry in the future, compared with past generations. In 1960 the average American over age 55 years had just seven original teeth. Today the average 65 year old has 18 original teeth; however, baby boomers (those born between 1946 and 1964) can expect to have at least 24 original teeth when they reach 65 years of age. The first adult teeth lost today are usually between the ages of 35 and 54 years. Almost 30% of the 50 to 59 year olds examined in a U.S. national survey exhibited either single or multiple posterior edentulous spaces bordered by natural teeth.[1] This segment of the population has the most disposable income and is the least dependent on insurance companies to pay for dental care. Treatment to replace single teeth in the posterior regions represents nearly 7% of the annual dental care reimbursement from insurance companies and totals more than 3.2 billion U.S. dollars each year.[2,3]

POSTERIOR MISSING TOOTH

The first molars are the first permanent teeth to erupt in the mouth and often play a pivotal role in the maintenance of the arch form and proper occlusal schemes. These teeth are often the first to decay, and the adult patient often has had one or more crowns fabricated to restore the integrity of the tooth and replace previous large restorations. Longevity reports of crowns have yielded very disparate results, with the mean life span at failure reported to be 10.3 years. The primary cause of failure of the crown is endodontic therapy, porcelain or tooth fracture (or both), or uncemented restoration. The tooth is at risk for extraction as a result of these complications and is a leading cause of single posterior tooth loss in the adult[1-9] (Figure 16-1).

POSTERIOR SINGLE-TOOTH REPLACEMENT OPTIONS

Evidence-based medicine is the conscientious, explicit, and judicious use of the best evidence in making decisions about the care of individual patients (Cochrane Center, Oxford, England).[10] Over the years, researchers have observed that external clinical evidence would both invalidate previously accepted treatment and allow replacement with new modalities that are more efficacious and safe.[10] An evidence-based approach may be applied to the replacement of a posterior single tooth.

Five alternative treatment options exist for the replacement of a posterior single missing tooth (Box 16-1). The interocclusal space must be assessed carefully, regardless of the treatment selected. Patients with insufficient vertical space may be contraindicated for any prosthesis without the prior correction of the occlusal plane and maxillomandibular relationships.

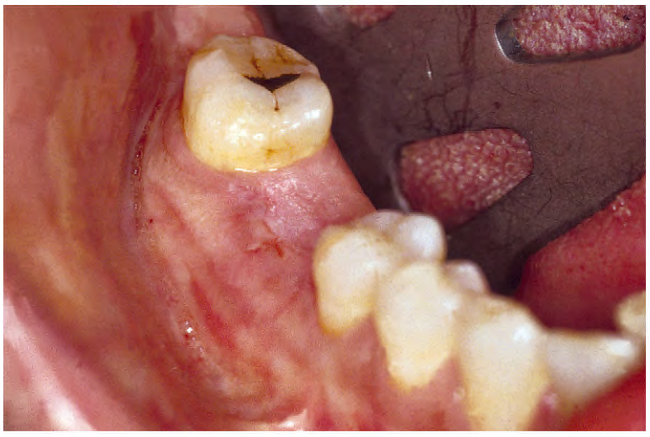


Figure 16-1 A mandibular first molar is often the first tooth lost in a permanent dentition.

WWW.HIGHDENT.IR

Box 16-1 Alternative Options: Single-Tooth Replacement

1. Removable partial denture (RPD)
2. Resin-bonded prosthesis
3. Space maintainer
4. Fixed partial denture (FPD)
5. Implant prosthesis

Box 16-2 Advantages of Removable Partial Dentures

1. Hygiene
2. Soft tissue replacement in esthetic areas
3. Maxillary lip support in gross defects
4. Minimum tooth preparation
5. Reduced cost

Removable Prosthesis

One option to replace a single missing tooth is a removable partial denture (RPD). A common axiom in restorative dentistry is to use a fixed prosthesis whenever possible.[8] RPDs are usually indicated to replace spans of three or more posterior teeth or a missing canine and two or more adjacent teeth. Rarely does a patient consent to a RPD as an acceptable definitive substitute for one posterior single tooth.

The advantages of the removable restoration for multiple tooth loss include the following: ease of daily care of the adjacent teeth, the ability to have a soft tissue replacement around the missing tooth in esthetic zones with gross defects, maxillary lip support in gross defects, minimal preparation of the abutment teeth, and reduced cost (Box 16-2). However, no reported advantages exist for an RPD replacing one posterior tooth.

Removable prostheses do not maintain bone. The maxillary posterior teeth are often in the esthetic zone (especially the maxillary premolars), and bone loss may compromise the esthetic result. Function is not improved with a removable prosthesis replacing one or two teeth, so esthetics and the fear of other teeth shifting in the arch are the two primary reasons for the patient to consent to wearing the restoration. Because of its bulk and the need for cross arch stabilization, an RPD promotes more food debris and plaque accumulation on the adjacent teeth than any other treatment option (Box 16-3). No clinical reports are available to assess the longevity survival rate, complications, or survival of adjacent teeth for a single-tooth RPD in the posterior regions of the mouth. From an evidence-based approach, this procedure is not indicated.

Recently, Shugars et al.[3] and Aquilino et al.[11] have reported on survival rates of teeth adjacent to treated and untreated posterior-bounded edentulous spaces. When RPDs were used to replace teeth, the survival of the posterior teeth adjacent to the edentulous space were poorer than with any other treatment option, with ranges from 17% to 44% abutment tooth loss at 4.2 to 13.5 years.[3,11-14] Patients electing not to wear the RPD may enjoy greater survival of the adjacent teeth than those wearing the removable prosthesis.

Box 16-3 Disadvantages of Single-Tooth Removable Partial Dentures

1. Bulk—need cross arch stabilization
2. Food debris, plaque
3. Movement
   a. Speech
   b. Function
4. No clinical studies
5. Bone loss
6. Highest loss of abutment teeth (up to 44% within 10 years)

Box 16-4 Advantages of Resin-Bonded Fixed Partial Dentures

1. Minimal preparation of teeth
2. Good on young patients (no need to crown, no risk of encroachment on pulp)
3. Good alternative when skeletal growth is not complete

Box 16-5 Disadvantages of Posterior Resin-Bonded Fixed Partial Dentures

1. High debond rates (50% within 3 years)
2. Inconvenience to patient and doctor
3. Risk of decay on abutment teeth

Resin-Bonded Fixed Partial Denture

A second option to restore a single missing tooth bordered by posterior natural teeth is a resin-bonded fixed partial prosthesis. The primary advantages of this restoration are the minimal preparation of the adjacent teeth and reduced cost (Box 16-4).

Failure rates reported in the literature are greatly disparate, but the majority of reports indicate a failure rate of at least 30% within 10 years and as high as 54% within 11 months.[6,15-17] It also appears that earlier perforated designs exhibited lower survival rates (Box 16-5).

The majority of resin-bonded fixed partial denture (FPD) failure occurs from cement failure, with different

WWW.HIGHDENT.IR

regions of the mouth exhibiting various retention rates. The highest survival rates occur in the maxillary anterior, followed by mandibular anterior, maxillary posterior, and mandibular posterior teeth respectively.[18] Therefore posterior tooth replacement is not as successful, compared with an anterior resin-bonded restoration. Debonding most often occurs during function; because eating is often a social experience, this may cause the patient embarrassment and insecurity. The selection of this option is usually driven by economics and the desire to maintain as much tooth structure as possible on the abutment teeth. This option is usually more accepted by the patient than the RPD, but it must be considered as a transitional restoration because of its high debonding rate.

Maintenance of the Posterior Space

A third treatment option to restore missing posterior teeth is to not replace the tooth but instead to maintain the missing space. A common doctrine has been to replace a missing tooth to prevent complications such as tipping, extrusion, increased plaque retention, caries, periodontal disease, and collapse of the integrity of the arch[8,14] (Figure 16-2). However, clinical studies evaluating the consequences of adjacent tooth loss indicate the loss of one or two teeth adjacent to a long-term edentulous space may range from 25% to less than 8% at 8 to 12 years.[3,13,14] For example, Aquilino et al.[11] reported an 18% 10-year tooth loss rate of adjacent teeth to a posterior missing tooth. The location of a missing posterior tooth often influences the prosthodontic treatment plan. In general, when third molars are missing, the author suggests not replacing a second mandibular molar[19] (Box 16-6). The mandibular second molar is not in the esthetic zone of the patient. Ninety percent of the masticatory efficiency is generated anterior to the mesial half of the mandibular first molar, so function is rarely a primary reason to replace the second molar. A 10% greater occlusal force is measured on the second molar compared with the first. This tooth is more likely to exhibit working or nonworking interfaces during mandibular excursions. The crown height space decreases as it proceeds posteriorly and represents a limited access for implant placement, component, and prosthesis screw, especially when opposing a natural dentition. As a result of the increased forces, occlusal interferences, limited abutment height, reduced retention, and cement surface area, a greater incidence of porcelain fracture, uncemented restorations, or both exists. In addition, the course of the mandibular canal anterior to the midfirst molar corresponds to the level of the mental foramen. However, in the region of the second molar, its course becomes highly variable with an elevated risk of paresthesia and neurovascular bundle damage during implant surgery and insertion. The bone quality in the second mandibular molar region is often inferior to other regions of the mandible, with increased risk of bone loss or implant failure.[20] The submandibular fossa topography mandates greater implant body angulation, with increased stresses at the crestal region of the implant, thereby increasing the risk of bone loss. Cheek biting is more common in this region because of the proximity of the buccinator muscle. The mandible exhibits increased flexure and torsion during opening

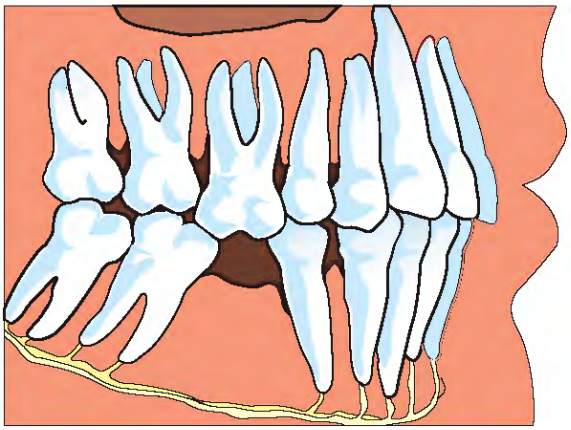


Figure 16-2 As many as 82 reasons exist for replacing a mandibular first molar after extraction. The most common reasons are tipping of adjacent teeth, extrusion of the opposing teeth, and increased plaque retention on the surrounding teeth.

Box 16-6 Disadvantages of Replacing a Mandibular Second Molar

1. Not in esthetic zone
2. Extruded maxillary second molar not esthetic or occlusal consequence
3. Less than 5% of total chewing efficiency
4. A 10% higher bite force (↑ bone loss risk, porcelain fracture risk, and abutment screw–loosening risk)
5. More often exhibits occlusal interferences during excursions
6. Higher and less predictable location of mandibular canal in that site
7. Less dense bone
8. Submandibular fossa depth greater; angulation of bone to occlusal plane greater
9. Limited to unfavorable crown height space for cement retention (increased risk of uncementation)
10. Limited access for occlusal screw placement
11. Limited access for correct implant body placement
12. Crossbite position—implant placed more buccal than maxillary tooth
13. Hygiene access more difficult
14. Cheek biting more common
15. More incision line opening after surgery
16. Greater mandibular flexure
17. Greater cost to patient
18. Mandibular third molar (when present) moves forward; intratooth space limited

WWW.HIGHDENT.IR

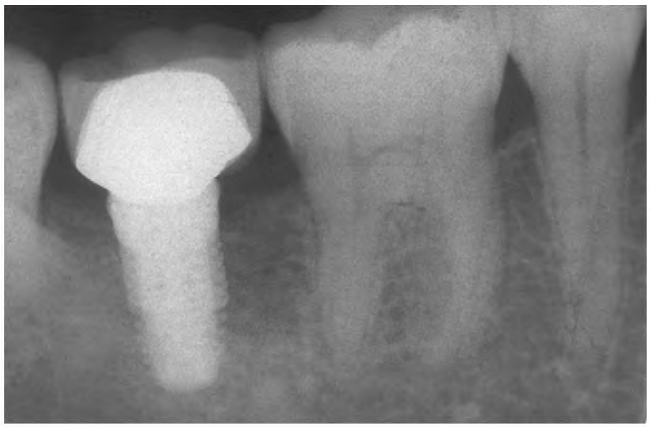


Figure 16-3 The second mandibular molar is usually replaced when the third molar is present and will remain in function.

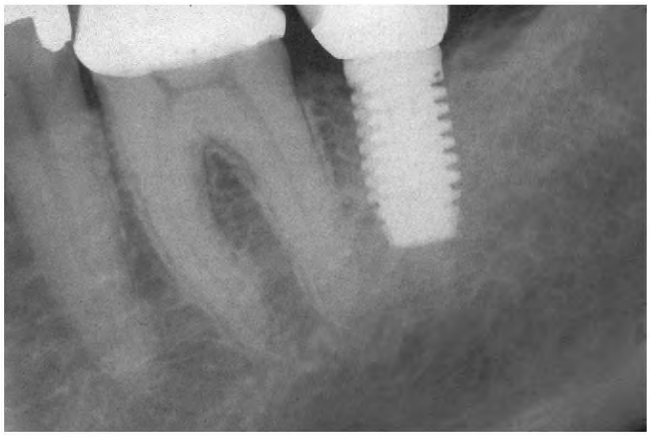


Figure 16-4 Some patients desire a second molar replacement despite the lack of need. When existing conditions are favorable, little disadvantage exists to placing an implant in this region. Most often a bicuspid-sized tooth and one implant are used in this region for restoration.

or heavy biting on one side at this second molar site, and masticatory dynamics are less favorable. Finally, the cost of an implant and/or fixed prosthesis to replace the second molar often does not warrant the benefits achieved. As a consequence, the mandibular second molar is often not replaced when the third molar and second molar are the only posterior mandibular teeth missing.

The primary disadvantage of electing not to replace a mandibular second molar tooth is the potential extrusion and loss of the maxillary second molar, or a loss of proper interproximal contact with the adjacent tooth, with increased risk of caries, periodontal disease, or both. If extrusion of the maxillary second molar is a concern for the patient or doctor, then a crown on the mandibular first molar may include an occlusal contact with the mesial marginal ridge of the maxillary second molar, or the maxillary second molar may be bonded to the maxillary first molar.

The mandibular second molar is usually replaced when the third molar is present and will remain in function (Figure 16-3). In addition, some patients desire an intact dentition and wish to have the tooth replaced (Figure 16-4). If the bone is abundant and no paresthesia risk is apparent, then the second molar may be replaced. However, this is usually the exception rather than the rule of treatment.

Another indication for not replacing a single missing posterior tooth is a small intratooth space. The existing occlusion may prevent the tipping of adjacent teeth and the extrusion of the opposing teeth. This condition is most often observed with a missing mandibular second premolar when a third molar is present. When the amount of intratooth space should be closed, orthodontics or overcontoured crowns on adjacent teeth may correct the condition.

Fixed Partial Denture

The treatment most commonly used for the replacement of a posterior single tooth is the three-unit fixed restoration. In 1990 more than 4 million FPDs were placed in the United States.[21] This type of restoration can be fabricated within 1 to 2 weeks and satisfies the criteria of normal contour, comfort, function, esthetics, speech, and health. Because of these benefits, the FPD has been the treatment of choice for the last 6 decades.[22,23] Few bone and soft tissue considerations exist in the missing tooth site. Every dentist is familiar with the procedure, and it is widely accepted by the profession, patients, and dental insurance companies (Box 16-7).

A three-unit FPD presents survival limitations to the restoration and to the abutment teeth.[7] In an evaluation of 42 reports since 1970, Creugers et al.[23] calculated a 74% survival rate for FPDs for 15 years. Walton et al.[24] and Schwartz et al.[25] reported mean life spans (50%) of 9.6 and 10.3 years, respectively. Scurria et al.[26] performed a metaanalysis of several reports at 10 to 15 years and found 30% to 50% failure within these time frames. However, reports are very inconsistent with as little as 3% loss over 23 years to 20% loss over 3 years.[4,5,23-26]

Box 16-7 Advantages of Fixed Partial Dentures

1. Most common treatment (doctor friendly)
2. Time (two appointments, 1 to 2 weeks apart)
3. Restores function, esthetics, and intraarch health
4. Few bone and soft tissue considerations
5. Proven long-term survival
6. Cost—dental insurance covers procedure (reduced patient cost)
7. Less than 6-mm mesiodistal space
8. Potential abutments have clinical mobility; will benefit from being splinted
9. Increases patient compliance and reduces fear
10. Few consequences if failure

WWW.HIGHDENT.IR

Box 16-8 Disadvantages of Fixed Partial Dentures

1. Mean life span often 10 to 15 years
2. Caries and endodontic failure of abutment teeth most common complication
3. Increased plaque retention of pontic increases caries and periodontal disease risk
4. Damage to healthy teeth
5. Failure of prosthesis related to loss of abutment teeth (8% to 18% within 10 years)
6. Fracture (porcelain, tooth)
7. Esthetics (anterior regions)
8. Uncemented restoration

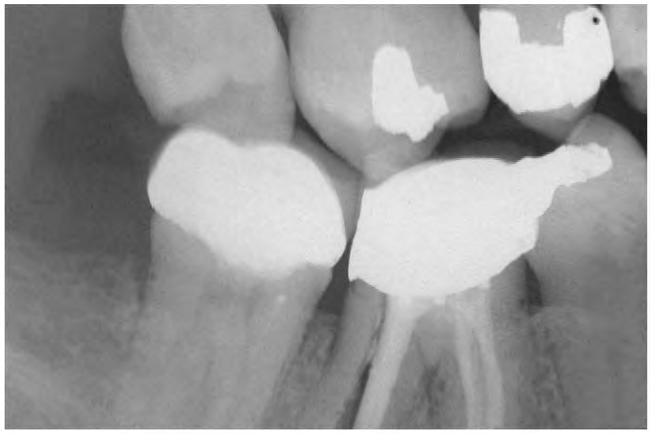


Figure 16-5 The most common cause of single-tooth loss is endodontic failure or fracture after endodontic therapy.

When a tooth is prepared for a crown, a 5.7% risk of irreversible pulpal injury and subsequent need for endodontic treatment exists.[27] In addition, the crown margin next to the pontic is more at risk of decay and the need for endodontics as a result. Caries and endodontic failure of the abutment teeth are the most common causes of prostheses failure.[22,24,26] Researchers have observed that abutment teeth for an FPD fail from endodontic complications more often than those with vital pulps.[28-30] Caries on the crown primarily occurs on the margin next to the pontic. A low percentage of patients floss on a regular basis, and those using a floss threader are even fewer.[30] As a result, the pontic acts as a large overhang next to the crown and a reservoir for plaque. Up to 15% of abutment teeth for a fixed restoration require endodontic therapy, compared with 3% of nonabutment teeth with crown preparations[31] (Box 16-8). The long-term periodontal health of the abutment teeth may also be at greater risk, including bone loss.

Unfavorable outcomes of FPD failure include not only the need to replace the failed prosthesis but also the loss of an abutment and the need for additional pontics and abutment teeth in the replacement bridge. Because

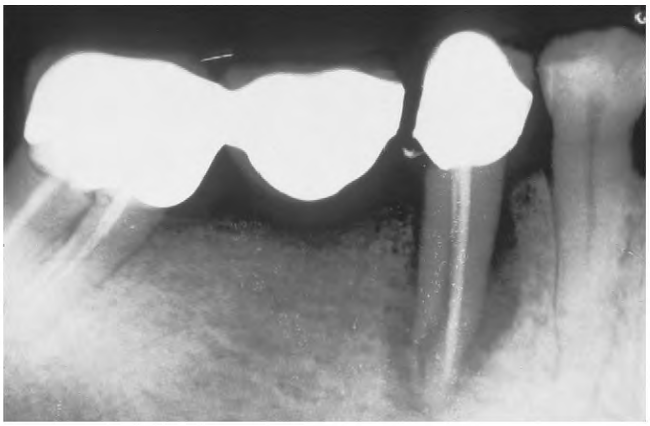


Figure 16-6 The most common reason for fixed partial denture (FPD) failure is caries on an abutment tooth resulting from increased plaque retention next to the pontic. Endodontic failure, fracture, and uncemented restorations often lead to abutment tooth loss.

Box 16-9 Contraindications for Use of Fixed Partial Dentures

1. Poor abutment teeth support
2. Inadequate hard or soft tissue (or both) in esthetic regions (pontic contour)
3. Patient will not allow preparation of adjacent teeth (patient desire)
4. Young patients with large pulp horns in clinical crowns

15% of FPD abutment teeth require endodontics, and root canal therapy is 90% successful at the 8-year mark, many abutment teeth may be lost (Figure 16-5).

The abutment teeth of an FPD may be lost at rates up to 30% for 8 to 14 years.[3,13,14] Recent reports indicate 8% to 18% of the abutment teeth holding an FPD are lost within 10 years (Figure 16-6). This is most disturbing, because 80% of abutments have no previous decay or are minimally restored before the fabrication of the FPD.[6] Some contraindications for a posterior fixed partial prosthesis are included in Box 16-9.

Single-Tooth Implants

The fifth treatment option to replace a posterior single missing tooth is a single-tooth implant. For years patients were advised to set their desires aside and accept the limitations of an FPD. However, many feel the most natural method to replace a tooth is to use an implant, rather than preparing adjacent teeth and joining them together with a prosthesis. The primary reasons for suggesting the FPD were its clinical ease and reduced treatment time. However, if this concept were expanded, then extractions would replace endodontics and dentures could even replace orthodontics. The primary reason to suggest or perform a treatment should not only be

WWW.HIGHDENT.IR

related to treatment time or difficulty to perform the procedure but also should reflect the best possible long-term solution for each individual (Figure 16-7).

Before 1990 few long-term studies focusing on single-tooth implant replacement with osteointegrated implants in any region of the mouth had been published. Early reports indicated single-tooth implant results were less predictable than they have become in the last 10 years. For example, in 1990, Jemt et al.[32] reported a 9% implant failure within 3 years of prosthesis completion on 23 implants with screw-retained restorations (21 in the maxilla, two in the mandible). In 1992, Andersson et al.[33] published a preliminary report of a prospective study of 37 implants restored with cemented single-tooth crowns in 34 patients. A 3-year follow-up included this "developmental group" and an additional 23 patients with 28 crowns. The cumulative success rate recorded was 93.7%, with 89% of the developmental group in function 3 to 4 years.[33]

From 1993 to the present time, single-tooth implants have become the most predictable method of tooth replacement. More refereed reports exist in the literature than for any other method of tooth replacement. Most all 5-year reports demonstrate a higher survival rate than for any other method of tooth replacement. For example, in 1993, Schmitt and Zarb[34] reported no failures for 40 implants placed in 32 patients (28 in the maxilla, 12 in the mandible, with 27 in the anterior region and 13 in the posterior). After a period of up to 6.6 years, all implants were in function. In 1994, Ekfeldt et al.[35] reported a 4- to 7-year retrospective study of 77 patients who received 93 implants. The restorations were cemented or screw retained. Two implants were lost, both within the first year of function. In the same year, Cordioli et al.[36] evaluated 67 endosteal implants for single-tooth replacement over a 5-year period and observed an implant loss of 5.6%. In 1995, Haas et al.[37] also reported on 76 single-tooth implants observed for 6 years with a 2.6% implant loss.

A series of reports in 1991, 1994, and 1996 reported on a multicenter prospective study consisting of 92 patients who received 107 implants with a cumulative survival rate of 97.2% at 3 and 5 years.[38-40] The mean marginal bone loss (measured from the first thread, which is 2 mm below the crestal bone) did not exceed 1.0 mm. Plaque and gingival indices were indicative of soft tissue health. The most frequent complication was loosening of the abutment fixation screw, and this complication was significantly reduced after the first year.

Becker et al.[41,42] reported more than 90% success in a study over 4 years and 282 molar implants. Simon[43] observed 70 molar implants over a period ranging from 6 months to 10 years, with a 97.1% success rate. Levin et al.[44] reported in 2006 on single-molar replacement with implants over a longer period, with a 93.6% success rate.

A multicenter prospective clinical study was initiated with the Maestro Dental Implant System from BioHorizons in 1996.[45] Thirty-eight implants were placed in the posterior regions of the jaws: 15 in the maxilla and 23 in the mandible. These included six second molars (four maxillary and two mandibular), 22 first molars (six maxillary and 16 mandibular), seven second premolars (three maxillary and four mandibular), and three first premolars (two maxillary and one mandibular). All crowns were cement retained. The implant survival rate was 100% at the 5-year follow-up. The mean bone loss from implant insertion to uncovering was 0.4 mm from the original crest of the ridge, the additional mean bone loss over the first 1 year of loading averaged less than 0.3 mm, and no bone loss over the following year was observed. No incidence of abutment screw loosening or fracture of any components was observed in this study. In 2000, Misch et al.[46] reported on 30 single-tooth implants with this same implant system in the posterior maxilla, with 100% survival rate over a 5-year period. In 2006, Misch et al.[47] found the 100% survival rate for single-tooth implants was maintained for as long as 10 years.[47]

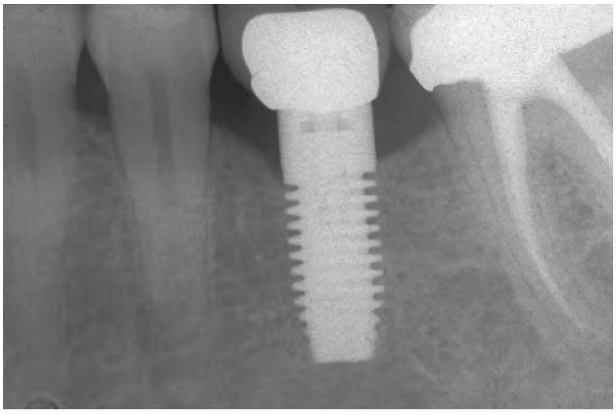


Figure 16-7 A single-tooth implant is usually the best treatment option to replace a posterior missing tooth.

A 10-year report by Priest[6] indicated the posterior single-tooth implant was more than 97% successful. Perhaps of more significance, no adjacent teeth loss from endodontic failure or caries occurred, and only one tooth required endodontic therapy after implant insertion. This report clearly identifies that the adjacent teeth are least at risk when the missing tooth is replaced with an implant.

Although posterior single-tooth replacement is a relatively new treatment alternative, more articles have been published than for any other treatment alternative. If early reports are excluded, then survival rates reported range from a low of 94.6% to a high of 100% for 1 to 10 years. A review of the literature by Goodacre et al.[48] from 1981 to 2003 found single-tooth replacement with an implant had the highest implant prosthesis survival rate and averaged 97% survival. The most common complication reported was abutment screw loosening, which did not cause the prosthesis or implant to fail.

WWW.HIGHDENT.IR

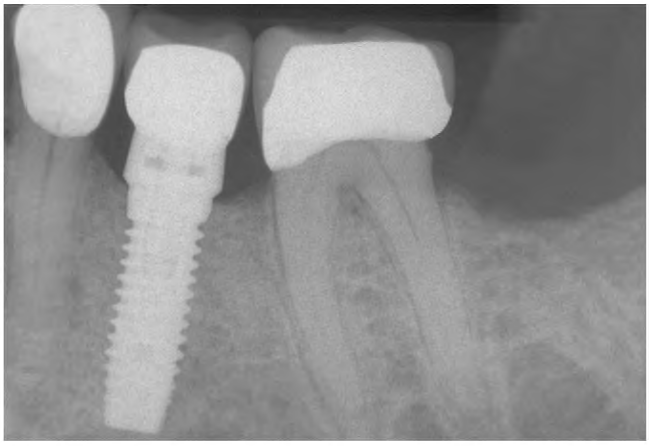


Figure 16-8 Because a single-tooth implant has the highest success rate of all the treatment options to replace a single-tooth, it is the treatment of choice, even when the adjacent teeth need crowns. (NOTE: A tooth with a crown has a 3% incidence of endodontic treatment versus a 15% risk for a tooth serving as an abutment of a fixed prosthesis.)

The single-tooth implant exhibits the highest survival rates of the five treatment options presented for single-tooth replacement. In addition, the adjacent teeth have the highest survival rate and the lowest complication rate, which is a considerable advantage (Figure 16-8). On the other hand, the longevity of the implant crown has not been adequately determined, because these reports do not extend as long as those of other treatment options. However, 10-year data clearly indicate an implant and its associated crown has greater survival than an FPD.

Despite some limitations and obvious clinical challenges, the posterior single-tooth implant represents a highly desirable and justified treatment option. In addition, cost comparison studies conclude that the implant restoration demonstrates a more favorable cost-effectiveness ratio.[6,48-49] When adjacent teeth are healthy or are able to be restored, or when the patient refuses their preparation for the fabrication of a traditional three-unit fixed partial restoration, a posterior single-tooth implant is an excellent solution. As a consequence of not preparing the adjacent teeth for crowns, many additional advantages are incurred. These advantages include the decreased risk of caries and endodontics treatment on the abutment teeth, the improved ability to clean the proximal surfaces of the adjacent teeth (which decreases the risk of decay and periodontal disease), a decreased risk of cold or root contact sensitivity with a brush or scaler on the abutment teeth, improved esthetics, maintenance of bone in the edentulous site, psychological advantages (especially with congenitally missing teeth or the loss of a tooth after a crown restoration), and the decreased risk of abutment tooth loss from endodontic failure or caries (Box 16-10). These advantages are so significant to the health and periodontal condition of the adjacent teeth and maintenance of the arch form that the single-tooth implant has become the treatment of choice in most situations.

Box 16-10 Advantages of Single-Tooth Implants

1. Adjacent teeth do not require splinted restorations
   a. Less risk of caries
   b. Less risk of endodontics
   c. Less risk of porcelain fracture
   d. Less risk of uncemented restoration
   e. Less fracture of tooth
2. Psychological need of patient addressed: patient does not desire two adjacent teeth (often virgin) prepared and splinted to restore missing tooth
3. Improved hygiene conditions
   a. Less decay risk
   b. Floss versus floss threader
   c. Less pontic "overhang"
4. Decreased cold or contact sensitivity
   a. Prepared teeth more temperature sensitive
   b. Cementum of tooth removed by tooth preparation; toothbrush or scaler sensitive
5. Improved esthetics: natural tooth versus crown esthetics
6. Maintains bone in site: 30% decreasing width within 3 years after extraction
7. Decreases adjacent tooth loss: 30% versus 0.05% risk at 10 years

The consequences of early failure may be greater for a single-tooth implant compared with a three-unit fixed prosthesis. Although surgical success is very high, the implant failure almost always results in bone loss. As a result, if the patient elects to repeat the procedure, then bone grafting may be required. This most often is at the expense of the doctor, because most patients believe early implant failure, at least in part, is the doctor's responsibility. Bone grafting is not as predictable as implant surgery; therefore if a graft is required (especially in height), then the procedure may not be successful. However, contrary to failure of a fixed prosthesis, implant failure does not compromise the adjacent teeth and does not increase the risk of their loss.

CONTRAINDICATIONS AND LIMITATIONS OF POSTERIOR SINGLE-TOOTH IMPLANTS

Local contraindications that are unique to posterior single-tooth implants (Table 16-1) and favor an FPD include inadequate bone volume, inadequate intratooth space, and observable mobility of the adjacent teeth. Grafting may modify inadequate bone volume, either in height or width. Bone grafting for additional height when the adjacent teeth have lost bone is not as predictable as implant insertion and healing, regardless of the technique used. Therefore an FPD may still be the treatment of choice.

WWW.HIGHDENT.IR

Table 16-1 Contraindications for a Posterior Single-Tooth Implant versus Indications for a Posterior Three-Unit FPD

LOCAL CONTRAINDICATIONS FOR A POSTERIOR SINGLE-TOOTH IMPLANT	INDICATIONS FOR A POSTERIOR THREE-UNIT FPD
Inadequate bone volume Faciopalatal <5 mm Mesiodistal <6.5 mm for a >3.5-mm-diameter implant	Indequate bone volume Inadequate intratooth space <6.5 mm (may also use two unsplinted crowns) Lack of intratooth bone height
Moderate to advanced mobility of two to four adjacent teeth	Adjacent teeth are mobile
Limited time for patient treatment	Reduced time of treatment

FPD, Fixed partial denture.

The mesiodistal posterior space should be at least 6.5 mm or greater. Smaller intratooth spaces should be restored with an FPD or two adjacent crowns that are overcontoured. Flossing is easier between unsplinted crowns than for a fixed prosthesis, and the cost is reduced. If the space is out of the esthetic zone, then the clinician may consider not replacing the tooth, if the adjacent teeth are not at risk of tipping or extrusion because of the occlusal relationship of the opposing teeth.

When the adjacent teeth have observable secondary mobility but all other periodontal indices are within normal limits, a three-unit fixed restoration is superior to the other treatment options. When the adjacent teeth have moderate to severe mobility, the occlusal adjustment of an implant crown may be difficult to perform, because it is the only rigid component in a span of three to five teeth. Posterior healthy teeth move vertically 28 µm and exhibit lateral movement of less than 75 µm. A heavy bite force occlusal adjustment allows the teeth to move within their physiologic range before the implant crown contacts in occlusion. However, when the surrounding teeth are mobile, an equilibration of force is not possible, because the implant crown will come into contact before the conclusion of the adjacent natural tooth movement. As a result, the implant bears the load of all the mobile teeth and therefore may be contraindicated when surrounded by teeth with advanced clinical mobility.

On rare occasions the length of time needed to replace the missing tooth constitutes the primary deterrent of the treatment. An FPD can be fabricated in less than 1 week and allows for the placement of a fixed transitional prosthesis.

To summarize, the primary indications for the selection of a three-unit FPD correspond to the limitations of single-implant tooth replacements: (1) limited time frame, (2) lack of available bone height with poor prognosis or impossibility to augment, (3) inadequate intratooth space, and (4) advanced clinical mobility of adjacent teeth.

Transitional Restorations

Few indications exist for a removable prosthesis as a definitive treatment when replacing a single posterior tooth. However, it is often used as a transitional restoration in esthetic regions during implant healing. A removable transitional restoration may load the soft tissue over a bone graft and compromise the result and volume of the augmentation. Less likely, the RPD may also cause bone loss, or perhaps even implant failure from the early loading around the implant during Stage I healing. The RPD transitional may also depress the interdental papillae of the adjacent teeth, resulting in an esthetic compromise. As a result, a resin-bonded fixed restoration may be fabricated when replacing teeth in the esthetic zone to provide an improved functional transitional prosthesis and protect the region. This is most often the primary option when bone grafting is necessary before or in conjunction with implant placement because of the bone graft's extreme vulnerability to movement and the extended healing time required.

Both a removable and a resin-bonded fixed prosthesis may be fabricated for the transitional restoration. The removable restoration (i.e., Essix appliance or flipper) is worn immediately after surgery to protect the suture line during the initial healing. Once the sutures are removed, the resin-bonded restoration (without tooth preparation) may be delivered. Because both resin-bonded and removable restorations are fabricated, the patient can insert the removable restoration if the bonded restoration becomes uncemented; this will eliminate the esthetic embarrassment until rebonding can be performed. However, this approach increases the overall cost of treatment. The posterior resin-bonded prosthesis may not be indicated in the case of short clinical crowns or unfavorable occlusal relationships.

An absence of transitional posterior tooth replacement is the most frequent situation during bone augmentation and implant healing in a nonesthetic region, such as the mandibular posterior aspect of the mouth. Although

WWW.HIGHDENT.IR

Table 16-2 Maxillary Teeth Dimensions*: Posterior Teeth

TOOTH	MESIODISTAL CROWN (mm)	MESIODISTAL CERVIX (mm)	MESIODISTAL CEMENT-ENAMEL JUNCTION	FACIOLINGUAL CROWN (mm)	FACIOLINGUAL CERVIX (mm)
First canine	7.1	4.8	4.2	9.2	8.2
Second canine	6.6	4.7	4.1	9.0	8.1
First molar	10.4	7.9	7.0	11.5	10.7

*Average measurements of maxillary teeth.

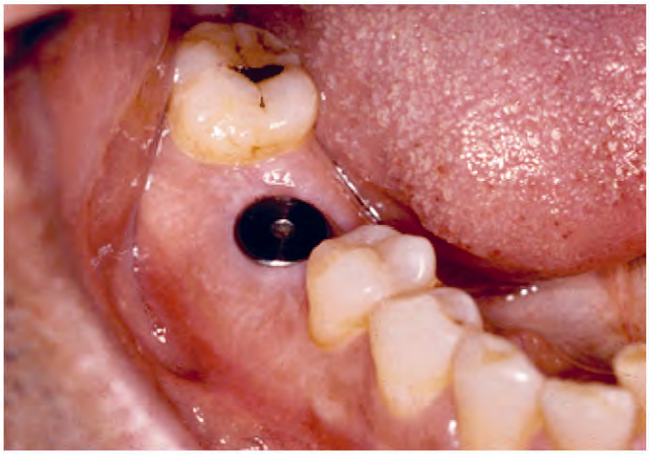


Figure 16-9 The most common option for tooth replacement during the initial healing period of the implant is no replacement.

the occlusion and adjacent teeth may change during the 4-month healing period, rarely is this a cause of further restoration in the region (Figure 16-9).

IMPLANT BODY SELECTION

The implant body for a posterior single-tooth implant should include specific features to reduce complications. The most common problem associated with a single tooth is abutment screw loosening.[35,38-42,47-55] Crest module and abutment connection designs that decrease forces to the abutment screw are therefore indicated. The implant must have an antirotational feature (i.e., external or internal hex). The greater the height or depth of the antirotational feature, the less force transmitted to the abutment screw. Accuracy of component fit and abutment screw design, as well as the number of threads, are other critical features.[56-58]

The implant body should be made of titanium alloy to reduce the risk of long-term fracture because it is four times more resistant to fracture than grade 1 titanium and twice as strong as grade 3 titanium. A threaded implant provides greater functional surface area than a cylinder, and a tapered implant provides less surface area than a parallel walled implant body. When implant bodies are internal hex designs, the dimension of the implant in the posterior regions should be at least 4 mm or more in diameter to increase the outer body wall thickness and reduce the risk of long-term body fracture.

The ideal diameter of a single-tooth implant is dependent on the mesiodistal dimension of the missing tooth and the buccolingual dimension of the implant site (Table 16-2). An angular defect may develop around the abutment-body connection measuring 1.0 to 1.4 mm in width. As a result, when the implant is placed closer than this dimension to an adjacent tooth, the vertical, angular defect dimension may cause bone loss on the tooth. When the implant has facial bone thickness less than 1.4 mm, bone loss may result and implant failures occur more frequently.[59] The horizontal bone loss around the implant will cause an increase in probing depths or an increased risk of soft tissue shrinkage. These may affect the bacterial flora or cervical esthetics of the soft tissue drape. This may be why gingival recession around wide-diameter implants have been noted to be greater than with a standard diameter.[60,61] As a consequence, the ideal implant diameter is 1.5 to 2.0 mm from an adjacent tooth and 1.5 mm from the lateral width of the ridge. Therefore the ideal implant diameter in the intratooth posterior region should be at least 3 mm less than the mesiodistal dimension of the missing tooth (from cement-enamel junction [CEJ] to CEJ) and 3 mm narrower than the buccolingual dimension of bone. As general rule, the molar implant should be larger in diameter than a premolar implant (Figure 16-10).

PREMOLAR IMPLANT REPLACEMENT

The most ideal posterior tooth to replace with an implant is the first premolar in either arch (Figure 16-11). When used as an abutment for a three-unit FPD, the canine is at an increased risk of material fracture or uncementation (because of the lateral forces applied) and is often more difficult to restore to its original appearance than are other anterior or posterior teeth.

WWW.HIGHDENT.IR

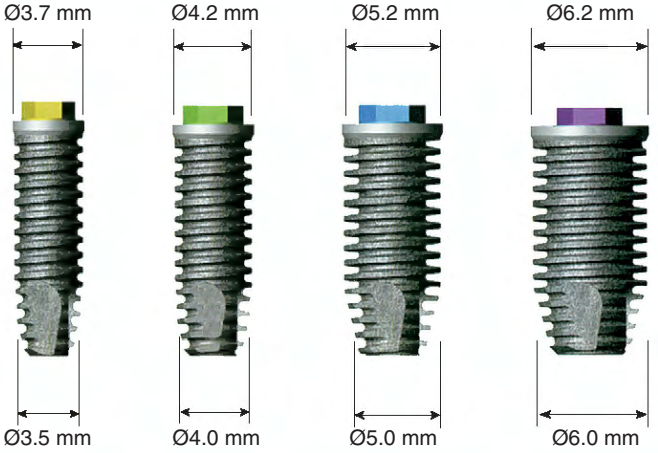


Figure 16-10 The single-tooth implant in the posterior region should range from 3.5 to 6 mm in diameter, 9 to 15 mm in length, be made of titanium alloy (for strength), and have a antirotational crest module design (i.e., external or internal hex).

Figure 16-12 The maxillary premolar implant should be positioned just lingual of the buccal cusp, similar to an implant in the anterior regions of the mouth, when the cervical region is within the esthetic zone. This position allows the facial emergence of the crown to be esthetic, yet it does not require a ridge lap or overcontouring.

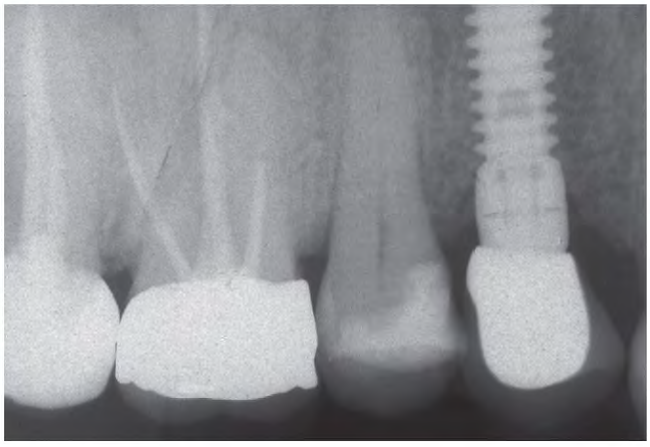


Figure 16-11 A missing first premolar (in either arch) is often indicated as an implant prosthesis, because a FPD with a canine abutment is more at risk and the vertical column of bone is usually in front of the maxillary sinus or mandibular mental foramen.

The vertical available bone is usually greater in the first premolar locations than in any other posterior tooth positions. In the maxilla, it is almost always anterior or below the maxillary sinus (or both), and the mandibular first premolar is almost always anterior to the mental foramen and associated mandibular neurovascular complex. The bone trajectory for implant insertion is more favorable in the mandibular first premolar than for any other tooth in the arch.

The maxillary premolars are often in the esthetic zone of patients with a high smile line. The need for bone grafting before maxillary first premolar implant placement is very common, because the extraction process of the thin buccal root often causes facial bone loss. Implant placement without bone grafting may result with a recessed emergence profile, which in the past was corrected with a facial ridge lap to the crown. However, the ridge lap contour does not allow proper hygiene or probing of the facial sulcular region of the crown and should not be used.

To ensure a proper esthetic result and to avoid the need for a crown with a ridge lap, the implant body is often positioned similar to an anterior implant, under the buccal cusp. The slight buccal implant placement improves the cervical emergence profile of the maxillary premolar crown (Figure 16-12). The natural premolar tooth root is 4.2 mm in diameter on average at a distance of 2 mm below the CEJ. As a consequence, the most common implant diameter is about 4 mm at the crest module. This also provides approximately 1.5 mm of bone on the proximal surfaces adjacent to the natural teeth when the mesiodistal space is 7 mm or greater. However, when the mesiodistal dimension is only 6.5 mm, a 3.5-mm implant is suggested.

The maxillary canine root is often angled 11 degrees distally and presents a distal curve 32% of the time, which may extend over the shorter root of the maxillary first premolar. The implant body is often longer than the natural tooth root. The surgeon may inadvertently place the implant parallel to the second premolar and, consequently, into the natural canine root. This may not only result in endodontic therapy of the canine but also may cause root fracture and loss of the tooth. Therefore in the maxillary first premolar region, care must be taken to evaluate the canine angulation and vertical height limitation. The first premolar implant may need to be placed parallel to the canine root, and a shorter implant than is considered ideal may be required. A tapered implant body at the apical one third may also be of benefit (Figure 16-13).

The second premolar apices may be located over the mandibular neurovascular canal or maxillary sinus. This results in a reduced height of bone, compared with the anterior region of the jaws. As a result a shorter implant is a common consequence in this region.

WWW.HIGHDENT.IR

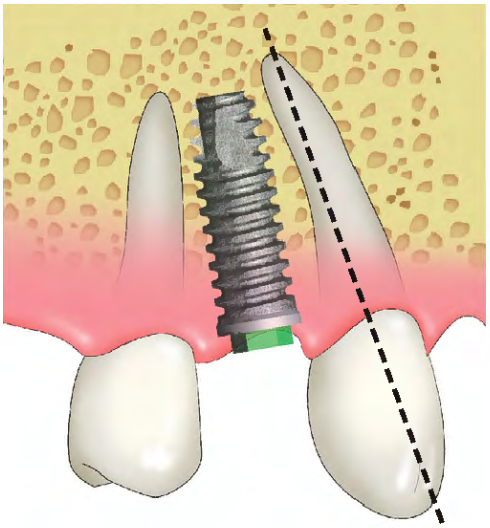


Figure 16-13 The maxillary first premolar implant often is angled distally to remain parallel to the maxillary canine root. A shorter implant or a tapered implant also may be of benefit.

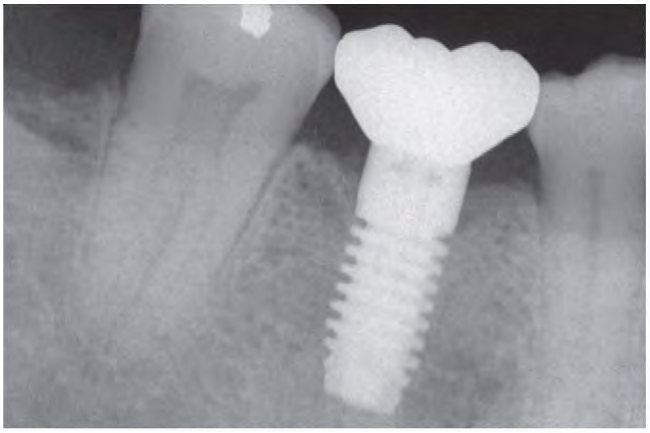


Figure 16-15 A 5-mm-diameter implant used to replace a mandibular first molar reduces the cantilever length on the marginal ridge, reduces stress to the abutment screw, and decreases the risk of crestal bone loss.

FIRST-MOLAR IMPLANT REPLACEMENT

The first molar is one of the teeth most frequently lost in a posterior segment. Its mesiodistal dimension usually ranges from 8 to 12 mm, depending on the original tooth size and the amount of mesial drift of the second molar before implant placement. When one 4-mm-diameter implant is placed to support a crown with a mesiodistal dimension of 12 mm, this may create a 4- to 5-mm cantilever on the marginal ridges of the crown (Figure 16-14). The magnified occlusal forces (especially important in parafunction) may cause bone loss, which may complicate home care, increase abutment screw loosening, and increase abutment or implant failure because of overload.[61,62] Sullivan[62] reported a 14% implant fracture rate for single molars fabricated on standard-diameter Nobel Biocare implants and concluded that this is not a viable treatment. Rangert et al.[63] reported overload-induced bone resorption appeared to precede implant fracture in a significant number of single-molar implant restorations. When possible, a larger-diameter implant should be inserted to enhance the mechanical properties of the implant system through increased surface area, stronger resistance to component fracture, increased abutment stability, and enhanced emergence profile for the crown[64-67] (Figure 16-15).

When the mesiodistal dimension of the missing tooth is 8 to 12 mm, with a buccolingual width greater than 7 mm, a 5- to 6-mm-diameter implant body is suggested (Figure 16-16). Langer et al.[64] also recommended the use of wide-diameter implants in bone of poor quality

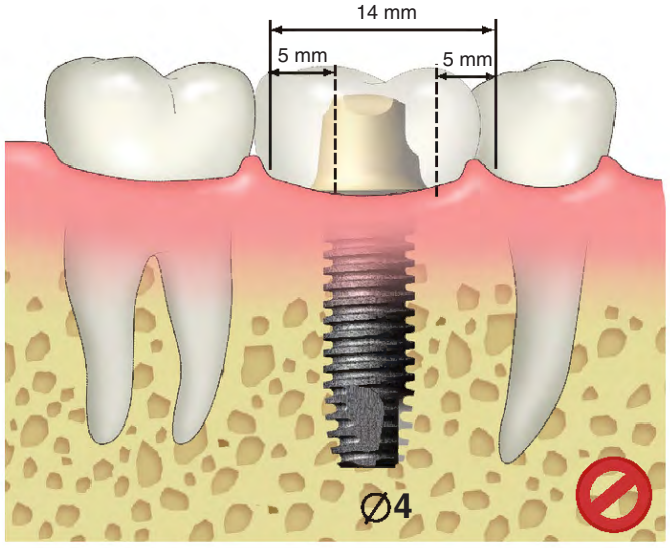


Figure 16-14 When one 4- or 5-mm-diameter implant is placed to replace a molar 14 mm in the mesiodistal dimension, as much as a 5-mm cantilever is created on both proximal aspects.

WWW.HIGHDENT.IR

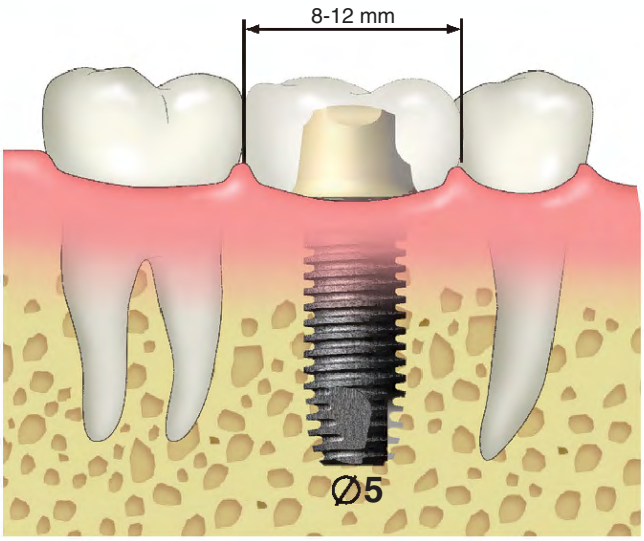


Figure 16-16 When the mesiodistal space is 8 to 12 mm and the buccolingual dimension permits, a larger-diameter implant is better suited to replace the missing tooth. The larger-diameter implant enhances the mechanical properties of the implant system (increased surface area, greater resistance to fracture, less screw loosening) and improves the emergence profile of the final restoration.

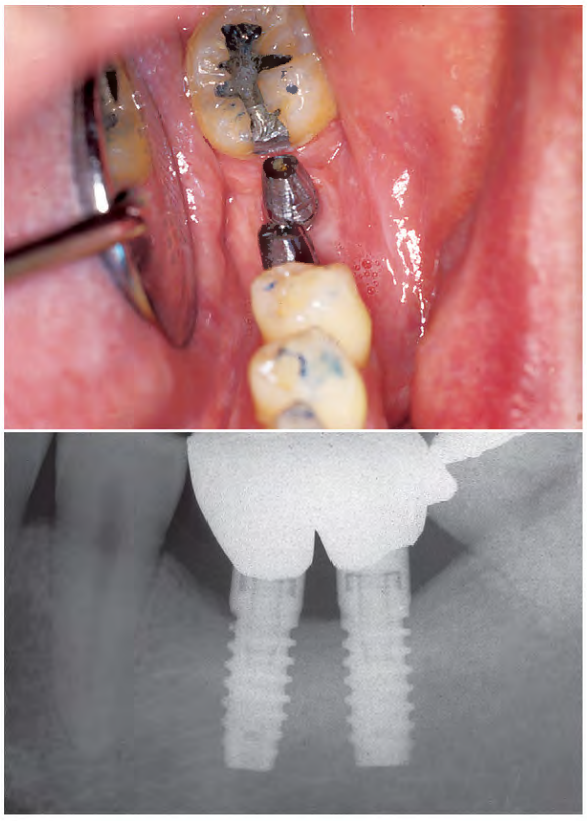


Figure 16-17 **A,** The mesiodistal space in the posterior first molar region is 14 mm. Two 4-mm-diameter implants were selected for the support of the prosthesis. **B,** A periapical radiograph of the two implants splinted together to restore the first molar site after 3 years in function.

or for the immediate replacement of failed implants. The larger-diameter implant does not require as long an implant, which is also a benefit because of the reduced posterior vertical bone height due to anatomical limitations and landmarks present, such as the maxillary sinus or mandibular canal.[51,64,68]

When the mesiodistal dimension is 14 mm or greater, two 4-mm-diameter implants should be considered to restore the region. When two implants replace the molar region, the mesiodistal offset loads to the prosthesis can be eliminated. The total surface area of support is greater for the two implants compared with the surface area provided by one larger-diameter implant. In addition, the two regular-size implants provide more stress reduction than just one larger-diameter implant, which in turn reduces the incidence of abutment screw loosening. In 1996, Bahat et al.[51] reported on the results of various implant numbers and size selection. The overall failure rate was 1.2%, with the two 5-mm implants having 100% success. In the same year, Balshi et al.[52,53] compared the use of one implant and two implants to replace a single molar. The 3-year cumulative success rate was 99%. Prosthesis mobility and screw loosening were the most common complications for the one-implant group (48%); this complication rate was reduced to 8% in the two-implant group. An in vitro study compared screw loosening of one wide-diameter versus two standard-diameter implants. Bakaeen et al.[69] also concluded that the one wide-diameter implant had greater screw loosening. In a finite element analysis of three different implant-supported molar crown design, Geramy and Morgano[70] showed a 50% decrease in mesiodistal and buccolingual stress between a 5-mm- and standard-diameter implants. The double implant design had the least stress of all. Therefore whenever possible, two implants should be used to replace a larger single-molar space to reduce cantilever loads and abutment screw loosening (Figure 16-17).

When the posterior space is 14 mm or greater, the largest implant diameter for the two implants may be calculated by subtracting 6 mm (1.5 mm from each tooth for soft tissue and surgical risk, and 3 mm between the implants) from the intratooth distance and dividing by 2, for a total of 5 mm for each implant (Figure 16-18).

$$\frac{16\,\text{mm} - 6\,\text{mm}}{2} = 5\,\text{mm}$$

The diameter is the crest module dimension, which is often 0.35 mm greater than the implant body dimension (i.e., Nobel Biocare, SteriOss, 3-I, LifeCore). The two implants should be 3 mm apart, because crestal bone loss around each implant may occur. The width of the crestal defect is usually less than 1.5 mm. Therefore the two adjacent implants 3 mm or more apart will not convert the angular defect to a horizontal defect that may increase sulcus depths and cause a loss of papilla

WWW.HIGHDENT.IR

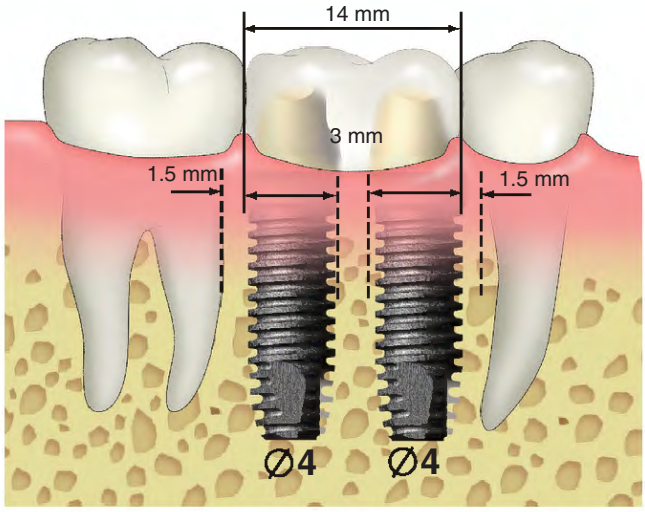


Figure 16-18 When the mesiodistal space between two teeth is 14 mm or greater, two implants should be used to restore the site. The implants are positioned at least 1.5 mm from each tooth and about 3 mm from each other.

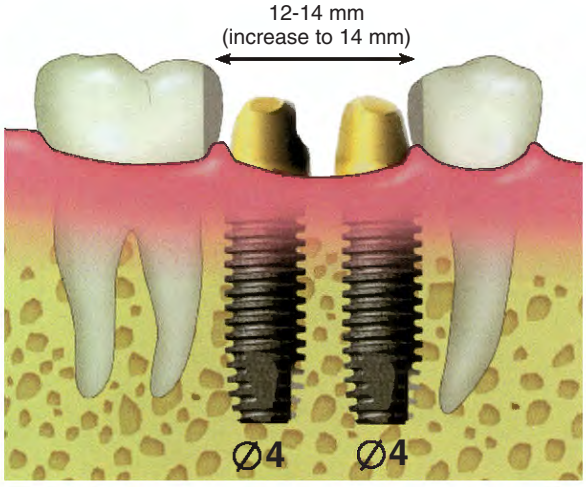


Figure 16-19 An enamoplasty can be used to increase the mesiodistal space slightly, especially on a tipped distal tooth. The minimum of 14 mm of mesiodistal space is measured from one cement-enamel junction (CEJ) to the other, not from one proximal contact to the other. When the interproximal contact is directly above the CEJ, further enamoplasty is not indicated because it will not increase the space required for the implants.

height.[71] When possible, two regular-size or one regular- and one larger-diameter implant are suggested when replacing molars. The natural molars have 200% more root surface area compared with premolars; therefore the implant support in a molar region should be greater than in the premolar position.

When the mesiodistal space is 12 to 14 mm, the treatment plan of choice is less obvious. A 5-mm-diameter implant may result in cantilevers up to 5 mm on each marginal ridge of the crown. However, two implants present a greater surgical, prosthetic, and hygiene risk. The primary goal is to obtain at least 14 mm of space,

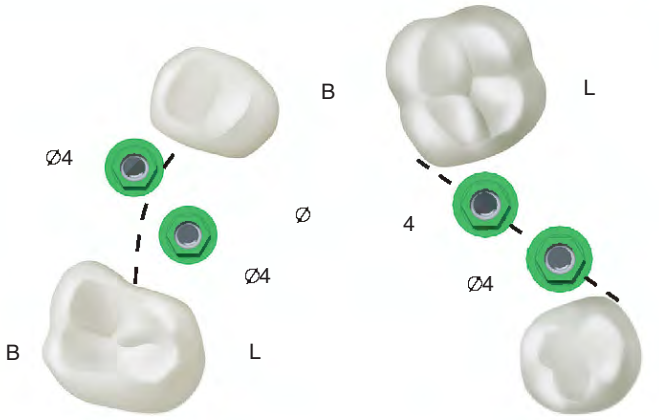


Figure 16-20 When the site is wide enough, up to 1 mm of additional mesiodistal space may be obtained when the implants are placed on a diagonal rather than aligned midcrestal. In the mandible the distal implant is positioned more buccally, and the mesial implant is placed more lingually. A floss threader has better access from the buccal for hygiene between the splinted crowns. In the maxilla the mesial implant is more buccal because it is often within the esthetic high-smile zone. The distal implant is positioned more palatal. Home care is not compromised because a palatal approach to the intraimplant space in the maxilla is more practical.

instead of 12 to 14 mm. Additional space may be gained in several ways:

- Enamoplasty of the adjacent teeth's proximal contours may be performed to increase the mesiodistal dimension of the missing tooth. It is not unusual that the distal natural tooth has tipped toward the edentulous space. An enamoplasty may be even more effective in these cases to increase space (Figure 16-19).
- Orthodontics may be the treatment of choice to upright a tilted second molar or increase the intratooth space. One anterior implant may be placed and an orthodontic spring incorporated in the transitional crown. The spring pushes the distal tooth more distal; after orthodontic movement, the second implant may be inserted with less risk and improved hygiene between each implant. Another option is to orthodontically reduce the space and place only one implant and crown.
- The implants may not be centered in the crestal width of bone. Instead, one implant is placed buccal and the other on a diagonal toward the lingual (Figure 16-20). The diagonal dimension increases the mesiodistal space by 0.5 to 1.0 mm. When implants are placed in such a way, consideration is given to oral hygiene and occlusion. In the mandible, the most anterior implant is placed to the lingual aspect of the midcrest, and the more distal implant is placed to the facial aspect to facilitate access of a floss threader from the vestibule into the intraimplant space. The occlusal contacts are also slightly modified on the buccal aspect of the mesial implant to occlude over the central fossa (Figure 16-21). In the maxilla, the anterior implant is placed to the buccal aspect and

WWW.HIGHDENT.IR

Figure 16-21 The mandibular first molar site is restored with two implants splinted together. The mesial implant is placed more lingual, and the distal implant is placed more buccal.

Table 16-3 Single-Molar Replacement Options

MESIODISTAL DIMENSION (mm)	IMPLANT DIAMETER
7	4 mm
8 to 12	5 mm
12 to 14	Gain additional space, then place 2 × 4 mm
≥14	For 14 mm, 2 × 4 mm For 15 mm, 1 × 5 mm, 1 × 4 mm For 16 mm, 2 × 5 mm

the distal implant to the palatal region, to improve the esthetics of the more visible half of the tooth. The distal occlusal contact is placed over the lingual cusp, and the mesial occlusal contact is located in the central fossa position. The cervical esthetics of the maxillary molar is compromised on the distal half of the tooth to the benefit of greater intratooth distance and easier access for home care. This maxillary implant placement requires the intraimplant furcation to be approached from the palate, rather than the buccal approach as in the mandible (Table 16-3).

ESTHETIC MAXILLARY ANTERIOR TOOTH REPLACEMENT

Contrary to missing posterior teeth, nearly all patients have an emotional response regarding a maxillary anterior missing tooth. No question exists regarding the need to replace the tooth, and financial considerations are less important. When posterior teeth are extracted, little resistance to the preparation of adjacent teeth may be given to the dentist. However, when anterior, normal-looking teeth must be prepared to serve as FPD abutments, the patient is more anxious and often looks for an alternative. In the patient's perspective, anterior FPD restorations are never as esthetic as natural teeth. In part, this is because patients are able to distinguish between good and poor esthetic results. Because patients are only able to notice the restorations that are not natural in appearance, they think anterior FPDs are not esthetic. In younger patients with congenitally missing maxillary lateral incisors or with trauma to the maxillary central incisor (which resulted in its failure, often after endodontic therapy), the parents are eager to provide the best possible replacement option. They often perceive this option to be a single-tooth implant. As a consequence of these psychological factors, a common site for a single-tooth implant in a restorative practice is the maxillary central or lateral incisor.

In 1994 the number of implants used in the United States averaged fewer than two per patient. This was a consequence of most dentists using implant treatments for a single-tooth implant,[72] small-span FPDs with two implants, and two implant overdentures. From 1990 to 1999, a threefold increase occurred in the number of single-tooth implants placed by a practitioner, as well as an increase in the overall implant market.[73] Single-tooth implants are now one of the most common implant procedures performed in the United States. In the nonesthetic posterior region, the single-tooth implant is one of the simplest procedures in implant surgery and prosthetics. However, the maxillary central incisor single-tooth replacement is often the most difficult procedure to perform in all of implant dentistry.

The highly esthetic zone of the premaxilla often requires both hard (bone and teeth) and soft tissue restoration. The soft tissue drape is often the most difficult aspect of treatment. As a consequence, maxillary anterior single-tooth replacement is often a challenge, regardless of the experience and skill of the dentist.

The most common causes of maxillary anterior single-tooth loss are included in Box 16-11. Endodontic failure is less likely in the maxillary anterior region compared with posterior teeth, but the cause of pulp necrosis may more often lead to root resorption, compared with the posterior regions.

Box 16-11 Causes of Maxillary Anterior Single-Tooth Loss

1. Agenesia
2. Trauma
3. Endodontic failure
4. Fracture
5. Resorption
6. Caries

WWW.HIGHDENT.IR

Box 16-12 Maxillary Anterior Single-Tooth Replacement Options

1. Traditional fixed prosthetic device (i.e., FPD)
2. Cantilever FPD (for missing lateral incisor)
3. Removable prosthetic device
4. Acid-etched, resin-bonded FPD
5. Implant-supported crown

FPD, Fixed partial denture.

ALTERNATE TREATMENT OPTIONS FOR ANTERIOR SINGLE-TOOTH REPLACEMENT

The treatment plan for a single missing tooth in the maxillary anterior region is more complex than in other regions of the mouth. The alternatives to restore a single, maxillary anterior missing tooth are listed in Box 16-12.

Factors that contribute to the selection of a specific treatment option include the expected longevity of the device, the most common complications, and the successful treatment of the complications. An evidence-based evaluation of alternative options for single-tooth replacement is warranted.

Fixed Partial Denture

The three-unit fixed restoration can be fabricated in shorter time, is more predictable in the short term, and often satisfies the criteria of normal contour, comfort, function, esthetics, speech, and health. However, 7- to 9-year survival estimates for a three-unit FPD are often less than 75%, with disparate longevity studies reporting failures ranging from only 3% over 23 years to 20% over 3 years.[6,8,26,11] In a study of 42 reports since 1970, Creugers et al.[23] calculated an estimated survival rate for FPDs under ideal conditions of 74% at 15 years. Scurria et al.[26] performed a meta-analysis of FPDs at 10 and 15 years and found a 30% and 50% failure rate.

The most common complications associated with FPD failure include caries, endodontic complications (including fractures), and uncemented restorations (leading to decay).[5,9,24] When a tooth is prepared for an FPD abutment, a 15% risk of endodontic treatment exists, compared with a 3% to 5% risk for a single crown.[7,27] This may be related to the additional tooth preparation for parallelism of the abutments, the repreparation of teeth after prosthesis failure, or the increased risk of decay on the abutment teeth. The success rate of endodontic treatment at 8 years is 90% in a review of the literature.[74-76] A 10% endodontic failure increases the risk of tooth loss and is the most common cause of single-tooth loss at 8 to 10 years.[6] Endodontically treated teeth are also at increased risk of fracture, which is the second most common cause of single-tooth loss.[6] The increased plaque retention of an FPD pontic increases the risk of caries and periodontial disease that may lead to the loss of an abutment tooth.[5,7,9,24] Fracture of porcelain with an FPD is also a contributing complication at increased risk compared with a single crown (16% versus 2%). In addition, esthetic failures are more frequent in the anterior than in the posterior regions of mouth (see Box 16-8). Goodacre et al.[7] determined the incidence of complications associated with FPDs to be 27%, with an average length of service of about 8 years, compared with a complication incidence of 11% at 6 years for a single crown.

An unfavorable outcome or FPD failure may not only require the replacement of the failed prosthesis but also may include the replacement of an abutment tooth. The abutment tooth of an FPD is extracted 8% to 12% of the time within 10 years and as often as 30% within 14 years. This is particularly disturbing, because the abutment teeth maybe unrestored or have minimum restoration before the FPD almost 80% of the time.[77] In this respect, a single-tooth implant places the adjacent teeth at a lesser long-term risk compared with a fixed prosthesis.

In light of the present technology, the primary reasons for suggesting an FPD are its clinical ease and reduced time and cost. If this concept was expanded, then extractions would replace endodontics and RPDs would replace FPDs. Dentures could even replace orthodontics, because the denture teeth are almost always straight. The primary reason to suggest or perform a treatment is often not related to the cost, time, or difficulty to perform the procedure; instead it lays in the best possible long-term solution for each individual patient. However, considerations exist for the maxillary anterior missing tooth that favor an FPD over other treatment options (see Box 16-7).

Advantages of a Fixed Partial Denture

Patient Compliance and Patient Fear

Patients with desires for an ideal result often must be willing to follow through with many steps of treatment in conjunction with an implant restoration. Orthodontics to improve the adjacent teeth position, bone height, or soft tissue may be necessary. Soft tissue surgeries, bone graft surgery, implant surgery, and several prosthetic steps most likely are required during an extended treatment and healing process. If the patient is unwilling to comply with these many steps emotionally, financially, and physically, then the result is often less than ideal and falls short of what the patient envisioned. An FPD may have less compromise of treatment in conditions of inadequate bone, soft tissue, or space considerations.

The fear of surgery may be a deterrent for treatment. Repeated surgeries for soft tissue, bone grafting, implant insertion, and uncovery may be too much for some patients to handle. As a result, nonsurgical procedures may be warranted. The patient's desires, compliance,

WWW.HIGHDENT.IR

or fears may be a contraindication to the implant procedures just as much as other factors.

Time of Treatment

Limitations of treatment are also related to the time of treatment. The time required for a rigidly fixated implant to heal and be restored is approximately 3 to 6 months, when adequate bone volume is present. If bone grafting and soft tissue rehabilation are required, then the treatment may extend more than 1 year. In contrast, a traditional three-unit fixed prosthesis often may be completed in less than 3 weeks.

In some carefully selected cases, grafting with immediate implant placement may be performed simultaneously and shorten the length of treatment. However, this is limited to extraction sites compatible with the requirements of the technique, such as a nonsalvageable tooth as a result of endodontic failure, fracture, extensive root caries, or some type of root resorption. This technique is not recommended in cases of an active inflammatory process or periodontal disease in which the pathogenic flora would impair the healing of the site.

Consequence of Failure

The consequences of bone graft, implant, or prosthetic short-term failure are greater for a single-tooth implant, compared with a three-unit fixed prosthesis.[78] The implant failure may result in bone loss (especially when it occurs in the anterior regions) and is not only limited to the implant site but also may include the support system of the adjacent teeth. This consequence of failure is, in general, not limited to only the hard tissues, because any vertical bone loss is usually associated with a soft tissue recession with devastating effects on the esthetics of the entire edentulous site and adjacent teeth. As a result, bone grafting may be required not only before implant placement but also after a bone graft or implant failure. Additional soft tissue reconstruction is also often needed in the process.

These additional procedures are most often at the expense of the doctor, because the patient believes the early implant failure, at least in part, is the doctor's responsibility. One single-tooth implant failure can eliminate the profits of more than five successful restorations, depending on the time and techniques required to bone graft and replace the failed implant. These potential complications affect the overall overhead of the implant crown procedure and the cost to the patient for a maxillary anterior single-tooth implant.

The most common contraindication for a traditional fixed prosthesis and indication for a single-tooth implant in the anterior regions of the mouth is the patient's desire. Typically, an anterior tooth is damaged by decay or trauma and is restored with a full crown. The tooth later requires endodontic therapy. Subsequent endodontic failure leads to an apicoectomy. A vertical fracture is discovered, and the tooth is extracted. Because this whole cascade of events started with an anterior single crown, the patient may not accept a three-unit conventional FPD. In addition, patients are more concerned regarding the appearance of anterior teeth and wish to keep adjacent teeth intact. Their point of view is that a single-tooth replacement is superior to and more esthetic than a three-tooth replacement.

Patient desires always affect the treatment options. The expected end results must be clearly explained along with risks and limitations. Care is taken to discuss the soft tissue drape, especially when a high lip line is present during smiling. When the patient's expectations are very high, all conditions before implant restoration must be ideal. Bone volume, location, implant position, angulation, size and depth, as well as soft tissue health, contour, and color all must be ideal to obtain the ideal result. In addition, crown contour, color, and all restorative aspects are to be considered.

Cost to Patient

The laboratory fee to the doctor for three crowns is usually $400 to $500. The implant body, abutment, analog, and final crown fee usually is more expensive. The scheduled operating room time for setup, preparation, impression, temporization, and insertion for a three-unit prosthesis is less than 2 hours. The time allotted for an implant setup, surgery, transitional restoration, uncovery, abutment selection, preparation, impression, transitional Stage II restoration, and crown insertion for an anterior single-tooth implant and crown is often longer. The training to perform a three-unit fixed prosthesis is acquired in dental school; the training for an anterior single-tooth implant is acquired in continuing education programs after graduation or through several trial-and-error experiences. The equipment to prepare and deliver an FPD is present in any general dentist office. The electric motor, hand pieces, and inventory to place a single-tooth implant are additional expenses, which all in turn affect the cost of the procedure. In addition, the financial consequences of a bone graft or implant short-term failure are greater than a traditional restoration. Initially an anterior single-tooth implant and crown most often cost more than a traditional three-unit fixed prosthesis.

Although the initial cost of treatment may be higher for an implant single crown, in a study evaluating the cumulative expense to the patient for replacement of an FPD over a 30-year period, Priest and Priest[79] showed that despite an initial higher cost for the implant option, the implant becomes a better alternative economically after the break-even point at 7 years. From a practice management point of view, Bragger et al.[49] also concluded that implant reconstruction was a better financial option in the long term.

Adjacent Tooth Mobility

When an anterior tooth is lost because of periodontal disease, the longer clinical crown on an implant abutment is not the only concern. Ideally, the adjacent teeth of the anterior implant site should exhibit minimum mobility.

WWW.HIGHDENT.IR

Figure 16-22 An orthodontist moved the lateral incisors together to replace the central incisors. The lateral incisor edentulous sites are too narrow for independent implants and crowns. Two three-unit fixed partial dentures (FPDs) would increase the central incisor crowns and decrease the canine crowns to provide improved size for the lateral incisor pontics.

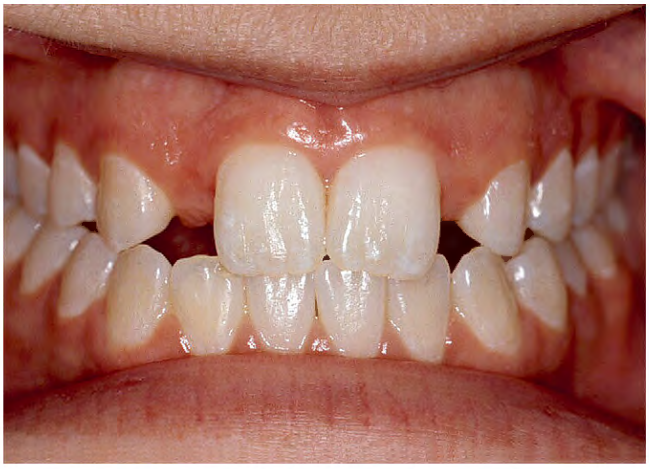


Figure 16-23 Inadequate bone in height, width, or intratooth space is a contraindication to an implant. A cantilevered fixed partial denture (FPD) from the canine position should be considered when the lateral incisor space is less than 5 mm.

However, this may not be the case because of a history of past periodontal disease. As a consequence, the slightly mobile adjacent teeth move out of occlusion during excursions, and the load of several teeth may be exerted solely on the rigid implant crown. When the adjacent teeth are mobile at a class 2 Miller index, the rigid implant most likely will bear the brunt of all occlusal load in centric occlusion and all mandibular excursions to this site. Therefore single-tooth implants surrounded by anterior mobile teeth are not indicated, unless the occlusion on the implant crown can be relieved to distribute occlusal forces to the natural teeth.

In cases of moderate to severe parafunction, movement of the adjacent teeth is a considerable risk. Natural tooth longevity is not related to mobility if all other periodontal indices are normal. Therefore a traditional FPD may be the treatment of choice under conditions of mobile adjacent teeth to the implant site. Splinting additional teeth to decrease the abutment mobility may be in order, especially if the adjacent teeth already require a restoration.

Unfavorable Tooth Size and Position

On occasion, the maxillary anterior central incisors may be misplaced, angled, rotated, or smaller than ideal (Figure 16-22). An FPD replacing a lateral incisor may improve the position and size of the central incisor. The canine may be made slightly narrower to make the lateral incisor similar in size to the contralateral incisor, because only the mesial half of a canine is visible when the rest of the anterior dentition is in view. Under these conditions, a conventional three-unit fixed prosthesis to replace the anterior missing tooth has several cosmetic advantages. This is especially noted when the lateral edentulous sites are smaller than 5 mm in width.

Contraindications for a Fixed Partial Denture

Contraindications for an FPD include poor abutment teeth support, inadequate bone and soft tissue in the edentulous site for proper pontic contour, anterior diastemae that the patient desires to maintain, adjacent teeth the patient will not allow to be prepared, or a young patient with large pulp horns in the clinical crowns (see Box 16-9).

Cantilevered Fixed Partial Denture

A cantilevered FPD has a worse prognosis than a traditional FPD.[8] The genesis of failure is usually an uncemented restoration, because it transfers tensile and shear forces to cements, and cements are more than 10 to 20 times weaker with this load type. However, a cantilever may be indicated in the anterior region (where forces are less) when the cantilever is short, when limited occlusion on the pontic exists, and when limited mesiodistal space exists.

When a missing lateral incisor space is less than 5 mm, consideration is given to a cantilevered restoration, most often using only the canine for abutment support because of its larger surface area and unique root shape (Figure 16-23). If needed, the canine crown may also be restored to a narrower mesiodistal dimension—to distribute more size to the lateral incisor pontic in order to make the anterior restoration more harmonious to the contralateral side.[4] As such, the cantilever FPD may eliminate the risks of implant placement in minimum intratooth space and eliminate the need for orthodontic treatment to gain additional space.

Removable Partial Denture

A transitional option to replace the single maxillary anterior tooth is an RPD. A common axiom in restorative dentistry is to use a fixed prosthesis whenever possible.[8] No short- or long-term clinical studies exist in the literature for single anterior tooth replacement with an RPD. The term *flipper* is often associated with this

WWW.HIGHDENT.IR

Figure 16-24 **A,** A resin-bonded restoration replacing a lateral incisor. **B,** The lingual support structure is bonded to the adjacent teeth, without preparation of the enamel. Therefore the restoration is retained below the occlusal contacts.

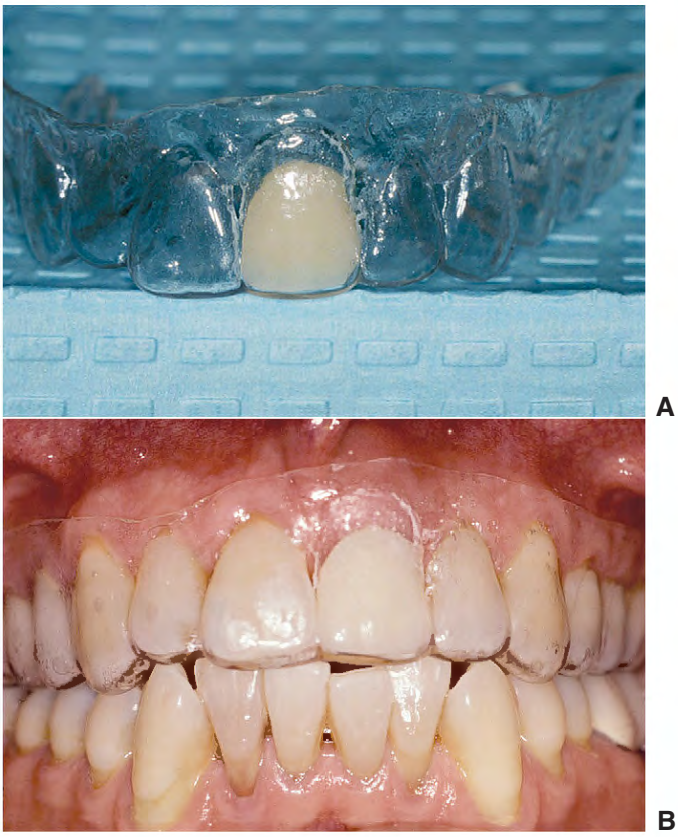


Figure 16-25 **A,** An Essix transitional restoration often is fabricated as the initial temporary device. If the patient is unhappy with the appearance, then a resin-bonded restoration is fabricated. **B,** The Essix appliance in place. If the resin-bonded restoration becomes unretained because of the reduced surface area design, then the Essix transitional restoration may be used until the patient returns to the office.

device and conveys the instability of the restoration during both speech and function. The usual indication for the removable option is economics. Rarely does a patient desire or accept a removable partial prosthesis as a definitive substitute for a single maxillary anterior tooth. However, this device represents the easiest interim treatment modality during submerged implant healing.

Loading of a bone graft with an RPD during initial healing will increase the risk of micromovement and decrease the success rate of a bone augmentation. Therefore if a bone augmentation is indicated and an RPD is used, it should have a cast framework with occlusal rests to prevent rotation and loading of the soft tissue during function.

Resin-Bonded Restoration

A resin-bonded restoration has a higher survival rate in the maxillary anterior region than any other location in the mouth. Despite this, a 30% uncementation rate is often reported.[15,18] Because the debonding is related to function, and eating is often a social event, the risk of embarrassment is high. Ideally, dental "emergencies" should be restricted to pain, rather than recementing a definitive restoration.

The primary indication for a resin-bonded prosthesis is as a transitional restoration during bone grafting and soft tissue grafts before implant placement. Soft tissue–borne interim prostheses impair bone graft healing. As such, a resin-bonded transitional restoration with two modifications may be used. First, no enamel preparation exists on the abutment teeth for occlusal clearance, and the metal substructure design is extended in areas of enamel that are gingival to the occlusal contact zones (Figure 16-24). As a result, the abutment teeth do not require restoration after treatment. However, this approach decreases retention of the device. The second modification of treatment is a modified Essix appliance (acrylic removable overlay prosthesis), or an acrylic RPD (flipper) is fabricated (Figure 16-25). The patient carries the RPD in case the resin-bonded restoration fails; as a result, cosmetic emergency appointments are eliminated. The secondary RPD is only intended to reduce cosmetic inconveniences to the patient. The bonded restoration may then be reinserted during regular scheduled office hours.

WWW.HIGHDENT.IR

Box 16-13 Contraindications for Resin-Retained Fixed Partial Dentures

1. Thin enamel on abutment teeth
2. Mobile abutment or abutments
3. Vertical overlap
4. Bruxism
5. Anterior diastemata
6. Short clinical crown
7. Poor occlusal relationship

Several limitations exist for a resin-bonded restoration. Thin enamel of adjacent teeth may result in a darkening or graying of teeth once the prosthesis is in place. Mobile teeth (even slight mobility differences in the abutments) often cause the debond rate to increase. A deep vertical overbite places considerable force on the prosthesis, especially in the presence of parafunction. Bruxism, in general, places this restoration at greater risk than a traditional prosthesis. The resin-bonded bridge may also be contraindicated in the case of wide interproximal spaces, short clinical crowns, and unfavorable occlusal relationships (Box 16-13).

Single-Tooth Implant

Maxillary single-tooth replacement is one of the most challenging restorations in dentistry. However, in light of all the advantages of single-implant longevity, bone maintenance, reduced abutment teeth complications, and increased survival of abutment teeth, single-tooth implants have become the treatment of choice.

Literature Review

Before 1990 few long-term studies of anterior single-tooth implant replacement with osteointegrated implants were conducted.[32] In 1996, Malevez et al.[61] presented a retrospective study of 75 patients treated with 84 screw-shaped implants (71% of the implants were in the anterior region), with a cumulative failure rate of 2.4% at 5 years. In 1997, Gomez-Roman et al.[54] published a 5-year report on 696 implants in 376 patients. Of the 696 implants, 42% were maxillary single-tooth implants, with a 96% overall success rate.

In 2005, Misch et al.[80] reported on 276 anterior maxillary single implants to restore missing teeth from agenesia. In 255 adolescent patients, the implants were monitored for a range of 2 to 16 years, with a 98.6% implant and prosthesis survival rate. In the same year, Wennstrom et al.[81] reported on a 5-year prospective study with 45 single-tooth implants, with a 97.7% implant survival rate with minimal bone loss. In 2006, Zarone et al.[82] reported on lateral maxillary agenesis replacement with 34 implants, with a 97% survival rate at 39 months.

More clinical studies have been conducted for a maxillary anterior single-tooth replacement with an implant than any other treatment option. Retrospective reports are available as with other modalities; however, of more importance is the fact that many prospective clinical studies confirm the data of previous reports.

The maxillary anterior single-tooth implant has the highest success rate compared with any other treatment option to replace missing teeth with an implant restoration (i.e., overdentures, short-span FPD, full-arch FPD, or single-tooth implant).[48,79-84] In a systematic review of single-implant restorations in all regions of the mouth, Creugers et al.[83] reported a cumulative rate of 97% success at 4 years, with 83% reporting uncomplicated maintenance. Lindhe et al.[84] published a meta-analysis of implants with nine studies on single implants, with a total of 570 single crowns with a follow-up range of 1 to 8 years and a 97.5% survival rate. A review of the literature by Goodacre et al.[48] found single-tooth implant studies had the highest survival rate of any prosthesis type and averaged 97%.

More recently, a trend toward single-stage and immediate-extraction implants has emerged. This appears especially attractive in the maxillary anterior region, where the soft tissue drape is ideal before the extraction and patients are more anxious to have a fixed replacement. In a prospective study of 102 single-tooth implants in the anterior maxilla, Kemppainen et al.[85] reported a 99% success rate using one-stage and two-stage implants. Other studies have recommended one stage and immediate load with some success in specific situations.[86-88]

As important as implant versus prosthesis survival rates is the fact that the adjacent teeth prognosis is improved with single-tooth implants compared with any other option. In a 10-year report, Priest[6] indicated adjacent teeth next to implants have less decay, endodontic risk, less sensitivity, less plaque retention, and less evidence of adjacent tooth loss over 10 years. Studies by Krenmair et al.[89] and Misch et al.[80] also resulted in a similar conclusion. As such, the maxillary anterior single-tooth implant has become the treatment of choice when bone and space parameters are sufficient or may be created.

Limiting Factors Influencing Treatment

One of the most common procedures performed in implant dentistry is a single-tooth replacement. Although implant success rates are high in the maxillary regions, high patient expectations, high esthetic requirements, and sensitive soft and hard tissue management compound the complexity of the anterior teeth restoration. A single maxillary central crown on a natural tooth is often a very difficult challenge for the restoring dentist. This

WWW.HIGHDENT.IR

Box 16-14 Maxillary Anterior Single-Tooth Replacement Influencing Factors of Treatment

1. Patient compliance and patient fear
2. Patient desires
3. Treatment time
4. Consequence of failure—potential damage to adjacent teeth
5. Cost
6. Patient's age
7. Esthetics
8. Adjacent tooth mobility
9. Crown height and occlusal relationship
10. Mesiodistal space at crown and bone level
11. Available bone height
12. Available bone width (buccolingual)
13. Soft tissue drape type—surrounding gingival tissues
14. Transitional prosthesis

challenge is significantly compounded when an implant serves as the prosthetic support. As a consequence, implants to replace a maxillary anterior single tooth remain one of the more difficult treatments to perform in implant dentistry.[90,91]

The limiting factors that may influence the treatment plan for maxillary anterior single-tooth replacement should be included in the presurgery preprosthetic evaluation. They are either related to the patient in general or are specific soft and hard tissue considerations for the area of treatment. The first five considerations (addressed earlier in the chapter) and other concerns are listed in Box 16-14.

Age Limitations

The minimum age of the implant patient is more often a concern for maxillary anterior tooth replacement, especially for congenitally missing teeth. Fixed partial dentures to replace teeth in children increase the risk of pulp necrosis of the adjacent teeth, because of the size of the pulp horns. Resin-bonded prostheses often become unretained in the younger patient. If bone is available, then the dentist wants to place the implant before bone loss in the near future. However, growth and development in the region may be affected by the implant, because it may act as an ankylosed tooth.

Researchers have documented that implants do not erupt along with adjacent teeth or become secondarily displaced in space as do ankylosed teeth during growth of the jaws.[92,93] As a result, many implants placed in adolescents with residual growth may be in infraposition after 10 years.[94,95] A new crown may correct the esthetic problem, but the bone position creates a greater soft tissue pocket around the implant, which may lead to shrinkage, peri-implant conditions, or both (Figure 16-26). In an 8-year study of single maxillary incisor implants in adolescents, Thilander et al.[96] concluded that a fixed chronological age is not an adequate guideline because of a slight continuous eruption of the adjacent teeth after adolescence. Instead, proper orthodontic treatment must be performed before implant placement to achieve proper space, avoid tooth intrusion movement, establish good incisor stability, and ensure proper stabilization (using a retainer) to avoid relapse.

The growth of the maxilla occurs in three distinct planes: (1) transverse (width), (2) sagittal (length), and (3) vertical. The transverse growth of the anterior maxilla is completed before adolescence, and the intercanine distance changes very little after age 10. The sagittal growth is the result of growth at the suture and bone apposition in the maxillary tuberosity region. The most variable growth of concern is the sagittal growth, because the premaxilla may advance downward and forward or primarily downward. As much as 25% of this displacement is lost as the result of resorption at the anterior region that can lead to facial bone resorption of the maxillary implants placed before completion of growth. This growth is a result of passive displacement, which comprises one third of the growth at age 7, whereas the additional two thirds of growth occurs later because of enlargement. However, this enlargement has been shown to be extremely variable among individuals.[97] This is the region where the growth should be completed before implant placement. Multiple implants should not be splinted across the midline in the adolescent when cessation of growth and development is undetermined.

Additionally during this growth, teeth shift mesially. The posterior segment (canine to molar) moves an average of 5 mm mesially between the ages of 10 and 21, and the anterior segment moves an average of 2.5 mm. Therefore an implant placed too early in the growth period could impede the mesial shift, thus resulting in an asymmetrical arch.

Figure 16-26 An implant and crown in a developing child will become "submerged," similar to an ankylosed deciduous tooth. The implant captures the bone in time and space. The soft tissue may recede and create a peri-implant pocket next to the implant.

WWW.HIGHDENT.IR

The vertical growth of the maxilla occurs via displacement and drift by growth of the orbits, nasal cavity, maxillary sinuses, and by deposition of bone on the palatal and alveolar surfaces. The vertical growth continues well after transverse and sagittal growth. The maxillary incisors have been shown to move approximately 6 mm downward and 2.5 mm facial between the ages of 9 and 25.[98] The eruption velocity is approximately 1.5 mm per year during active growth and 0.1 to 0.2 mm per year up to the age of 18. Clinical reports have shown that implants in the anterior maxilla at the age of 7 may be located up to 10 mm apically compared with the neighboring teeth 9 years later, and solitary implants placed at the age of 12 will be in infraocclusion 5 to 7 mm 4 years later.[99]

As a general rule, the lateral incisor may be inserted at a younger age than a central incisor or canine. It is less obvious to the eye when lateral incisors are at different height positions, compared with central incisors. It is not unusual for a lateral incisor to be shorter than the adjacent teeth, whereas a shorter central incisor becomes a more obvious esthetic problem when growth and development brings the adjacent natural tooth more incisal.

Over the last decade, Misch et al.[80] have created four guidelines for implants placed in younger patients. The first guideline is the chronological age of the patient. The chronological age of growth cessation for girls ranges from 9 to 15 years and 11 to 17 years for boys. It is logical to wait until skeletal and dental growth have been completed. As a general rule, implant insertion in the anterior maxilla is delayed for female patients until at least 15 years and male patients until 18 years of age. However, this guideline is too variable to be used alone; ideally, age is related to the patient's biological age more than his or her chronological age.[97]

Other biological factors indicative of completed growth should be assessed before implant insertion. The second criterion for implant insertion relative to children is endocrine changes. The female patient should be able to menstruate, and the male patient should have body hair, voice changes, and most often need to shave (if his father shaves daily).

The size of the child is also very relevant for implant insertion and is the third criterion. The prospective implant patient should have greater height than their same-sex parent. The size of the patient is more important than the age of the patient.

The fourth criterion for implant insertion is that the patient has not grown in the last 6-month period. Thilander et al.[96] has noted that if no statural growth has occurred in the last 6 months, then growth and development of the jaws is near completion. This criterion is easier to observe than cephalograms or hand-wrist films with a 2-year evaluation period. Some authors have suggested lateral cephalograms of 2 consecutive years of no changes. Although it is difficult to superimpose radiographs taken over several years, this criteria is the best indication that the pubertal growth spurt is finished and the majority of facial growth is finished.

The two criteria that make the implant site most at risk are (1) a male patient and (2) a central incisor. When a male patient has a delayed growth spurt, a 4-inch change in height may be observed, whereas a female patient may grow 1 to 2 inches. The central incisor position will be most noticeable, even if slight changes occur, whereas it is common for one lateral incisor to be shorter than the contralateral side.

If all four criteria are fulfilled (i.e., minimum age, endocrine changes, recent stature growth, 2-year lateral cephalometric radiographs with no changes) it is very likely the patient has completed their maxillary anterior jaw growth, and the implant may be inserted with little risk or compromise.

Challenging Esthetics

The esthetics of a maxillary anterior single crown on a natural tooth is often one of the most difficult challenges in restorative dentistry. The challenge to fabricate a natural-looking crown on an implant abutment is even greater. The implant is often 5 mm or less in diameter and round in cross section. A natural maxillary anterior crown cervix region is 4.5 to 7.0 mm in mesiodistal cross section and is never completely round.[99] In fact, the natural central incisor and canine teeth are often larger in their faciopalatal dimension at the CEJ than the mesiodistal dimension[100] (Table 16-4; Figure 16-27). Implants replicating the exact radicular cross section of a tooth would not be practical because it would be much more difficult to surgically prepare the bone and insert the implants. In addition, because the bone is lost first in faciopalatal width, the greater width of implants in this dimension would require even greater augmentation than presently advocated. As a result, the cervical esthetics of a single-implant crown must accommodate a round-diameter implant and balance

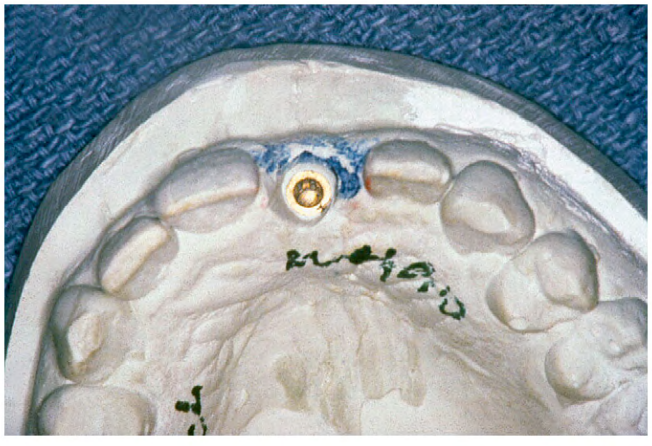


Figure 16-27 Cross sections of teeth are not round and are often larger in the faciopalatal dimension. The cervical emergence profile of a crown on a round implant platform needs to be created prosthetically.

WWW.HIGHDENT.IR

Table 16-4 Maxillary Anterior Teeth Dimension

TYPE AND NUMBER OF TEETH	MESIODISTAL CROWN (mm)	MESIODISTAL CERVIX (mm)	MESIODISTAL CEMENT-ENAMEL JUNCTION (–2 mm)	FACIOLINGUAL CROWN (mm)	FACIOLINGUAL CERVIX (mm)
Central incisor	8.6	6.4	5.5	7.1	6.4
Lateral incisor	6.6	4.7	4.3	6.2	5.8
Cuspid	7.6	5.6	4.6	8.1	7.6

hygiene and esthetic parameters. Additional prosthetic steps and components with varied emergence profiles or customized tooth-colored abutments are often required to render the illusion of a crown on a natural abutment.

The laboratory technician for a maxillary anterior implant crown should have specific training. Often a soft tissue model is required to transfer the soft tissue clinical condition to the laboratory. The soft tissue emergence from the bone and through the soft tissue drape is customized for each case. Rarely are these unique techniques required for implant restorations needed for a crown on a natural tooth. As a consequence, the learning curve for the restoring dentist and laboratory technician is more extended on implant crowns in the esthetic zone, and an increased risk of esthetic failures and redoing the restoration exists.

Crown Height Space

The interocclusal space should be assessed carefully. Patients with Angle's Class II Division II skeletal patterns, an inadequate maxillomandibular relationship, or a severe deficiency in the vertical dimension are poor candidates for many treatment options; therefore they are contraindicated for dental implants without prior corrections. In fact, limited vertical space complicates all options to replace a maxillary anterior tooth. Removable partial dentures often fracture because the occlusal aspects in a deep overbite do not permit proper thickness of the acrylic base. The FPD abutment tooth preparation is often inadequate to allow clearance for proper casting thickness. An implant abutment is usually too short for proper retention of the crown. This may mean orthognathic surgery, orthodontic therapy, or both is required before any tooth replacement.

Mesiodistal Space

An adequate mesiodistal space is necessary for an esthetic outcome of an implant restoration and the interproximal soft tissue of the adjacent teeth (Figure 16-28). A traditional two-piece implant should be at least 1.5 mm from an adjacent tooth. When the implant is closer than this to an adjacent tooth, any bone loss related to the microgap, the biological width, or stress will cause the implant and adjacent tooth to lose bone. This may compromise interproximal esthetics and sulcular health of the implant and natural tooth.[101,102] The smallest-diameter implant body offered by most commercial companies is 3.2 mm. However, the crest module of these two-piece implants is usually 3.5 mm or more. Therefore the mesiodistal edentulous space for a two-piece implant should be 6.5 mm or greater. The average maxillary lateral incisor is 6.6 mm in width, but patients with congenitally missing teeth often have contralateral anterior teeth narrower than typical. Therefore under these conditions, even orthodontic therapy to increase the intratooth space is inadequate for a single-tooth replacement. In addition, when the lateral incisor is missing, the root of the adjacent teeth may be angled toward the edentulous site, further decreasing the intratooth bone dimension for implant placement. Orthodontic treatment to reposition the roots out of the edentulous root space may not be accepted by the patient and therefore contraindicate the implant surgery. As a consequence, under these conditions a cantilevered FPD may be the treatment of choice.

One-piece dental implants may be fabricated in 2.5-mm to 3.0-mm diameters to accommodate a reduced mesiodistal dimension criterion.[103-105] These implant

Figure 16-28 Adequate mesiodistal space is required for an acceptable esthetic restoration. This implant is unable to be restored with an esthetic result.

WWW.HIGHDENT.IR

Figure 16-29 The bone in the implant site should be ideal, because it influences the soft tissue drape, implant size, implant position, and esthetic results. This central incisor site required significant augmentation of hard and soft tissue before implant placement.

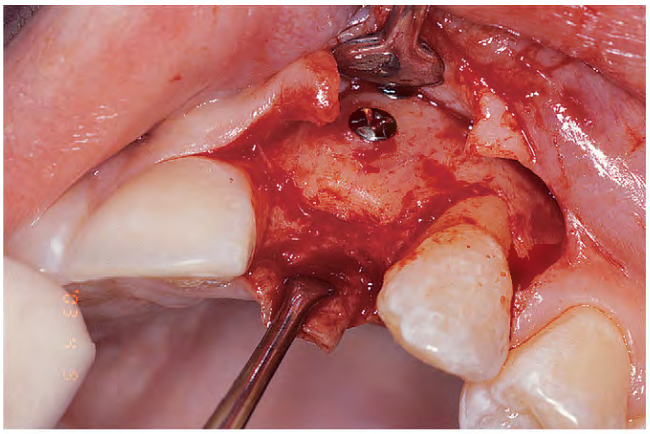


Figure 16-30 After bone augmentation the midosseous crest is too high, and osteoplasty before implant insertion is indicated.

designs do not have a microgap, and the vertical defect is narrower than most two-piece implant systems. As such, they may be placed as close as 1 mm from an adjacent tooth and therefore can accommodate a 5-mm mesiodistal missing tooth space (see Chapter 35).

Bone Height

The available bone for implant insertion in esthetic regions should be closely evaluated because it will greatly influence the soft tissue drape, implant size, implant position (angulation and depth), and ultimately the final esthetic outcome. An ideal hard tissue topography is a prerequisite to an optimal, esthetic implant restoration (Figure 16-29). Not only is available bone volume necessary but also the position of the osseous crest is specific. The ideal midcrestal position of the edentulous site should be 2 mm below the facial CEJ of the adjacent teeth. On occasion, the bone crest may be above this position when a bone graft was performed (Figure 16-30). Ideally, the interproximal bone should be scalloped 3 mm more incisal than the midcrestal position.

The position of the interproximal crest of bone is an important anatomical consideration, especially for the development of the interproximal soft tissue height. Becker et al. found the range of interproximal bone height above the midfacial scallop was from less than 2.1 mm to more than 4.1 mm and classified the interproximal bone height as flat for 2.1 mm, scalloped for 2.8 mm, and pronounced scalloped for 4.1 mm above the midcrestal bone level.[105a] The flat anatomy ideally corresponds to a square tooth shape, the scalloped to a ovoid tooth shape, and the pronounced scalloped to a triangular-shaped tooth. However, these relationships do not always exist. When a flat interdental-to-crest dimension is found on triangular teeth, the interproximal space will usually not be filled with soft tissue, because the dimension of the interproximal contact to the bone will be greater than 5 mm.

Often the osseous crest may be more apical than ideal, in both the implant site and the adjacent tooth roots. Under these conditions, ideal crown contour, soft tissue emergence, and interproximal tissue conditions are less likely. Bone and soft tissue changes after maxillary anterior tooth loss are rather rapid and of considerable consequence. As a result, many maxillary anterior edentulous sites require at least some bone and/or soft tissue modification before, in conjunction with, or at implant uncovery. Under ideal conditions, the implant body should not be inserted until the bone and soft tissue are within normal limits.

A careful evaluation of the three-dimensional soft and hard tissue complex in the edentulous space and the adjacent teeth is necessary before committing to a specific treatment modality. The crestal height of bone is one of the more important considerations for an implant in the esthetic zone. Overall bone height in an apical direction is not a problem in the anterior regions, it is often deficient at the crest of the ridge and greatly affects the position of the implant in relation to the adjacent teeth and the related soft tissue configuration. Bone grafting for height adjacent to a tooth is more difficult and less predictable than grafting for width or in extraction sockets. It is a clinical challenge when a single-tooth site has inadequate bone height at the crest and the adjacent roots also have lost bone, because it is not predictable to grow bone on a natural tooth root. Therefore growing bone height both in the edentulous site and growing bone on the roots of the adjacent teeth is not predictable.

To grow crestal bone height on the adjacent roots, in relation to the ideal crest of the ridge, orthodontic extrusion of the teeth may be considered. However, in cases of significant crestal bone height loss, endodontic therapy and a crown on the affected tooth is often indicated after treatment. After the bone on the adjacent teeth is at an ideal height, the edentulous site may be augmented with increased success.

WWW.HIGHDENT.IR

Figure 16-31 The cause of maxillary anterior tooth loss most often is associated with facial bone loss.

Box 16-15 Local Contraindications for the Anterior Single-Tooth Implant

1. Inadequate bone volume
   a. Faciopalatal (<5.5 mm)
   b. Mesiodistal (<6 mm for a 3.2-mm implant to allow 1.5 to 2 mm from adjacent roots; varies in function of tooth replaced)
   c. Height (<9 mm)
2. Inadequate crown height
3. Mobility of two to four adjacent teeth >+1

Faciopalatal Width

Most of the conditions that lead to single-tooth loss result in the loss of some or all of the facial bone in the region of the missing tooth (Figure 16-31). In addition, a 25% decrease in faciopalatal width occurs within the first year of tooth loss and rapidly evolves into a 30% to 40% decrease within 3 years. As a result, even an intact alveolus 6 to 8 mm wide is often adequate in width after 1 year for a root form implant in a central incisor position. After 3 years it almost never presents adequate available bone for the properly sized implant. The bone width loss is primarily from the facial region, because the labial plate is very thin compared with the palatal plate, and facial undercuts are often found over the roots of the teeth (Figure 16-32). A bone graft is necessary to restore the proper anatomy of the ridge and avoid a compromised implant position more palatal and apical. The final crown under these compromised conditions appears longer than the adjacent teeth, and the prosthetic "solution" often compromises long-term daily maintenance of the facial sulcular region. Therefore the majority of edentulous maxillary central incisor single-tooth sites require bone grafting for ideal cervical esthetics and hygiene.[106-107]

The amount of available bone width (faciopalatal) should be at least 2.0 mm greater than the implant diameter at implant insertion and ideally more than 3 mm greater in width; a 3.5-mm implant requires at least 5.5 mm of bone width (Box 16-15). Bone augmentation in width is very predictable. In many instances it is performed before implant placement but may be performed at the time of implant insertion, especially when no dehiscence of implant is visible. The implant diameter measurement is made at the crest module of the implant; 3.75-mm diameter implant bodies are 4.1 mm at the crest module. Therefore in these situations, the mesiodistal limitation is 7.1 mm, and the faciolingual width limitation is 6.1 mm.

Soft Tissue Drape

The position and architecture of the interdental papillae are noted before developing the implant treatment plan. The soft tissue in the region of the edentulous site should ideally have the same color and form as the adjacent teeth. When a tooth is lost, the thin interseptal bone disappears and the bone remodels in a sloping fashion from the palatal to the more apical facial bony plate. As a result, the interdental papillae are often depressed compared with their level between healthy adjacent teeth (Figure 16-33). The papilla height is also affected as a result of the lack of interproximal contact with the missing tooth. The use of a soft tissue removable prosthesis often accelerates the collapse of the soft tissue and its apical migration. Therefore once the tooth is extracted, interdental papillae are rarely at the desired height and are most often apically depressed. Soft tissue manipulation to restore their proper contour

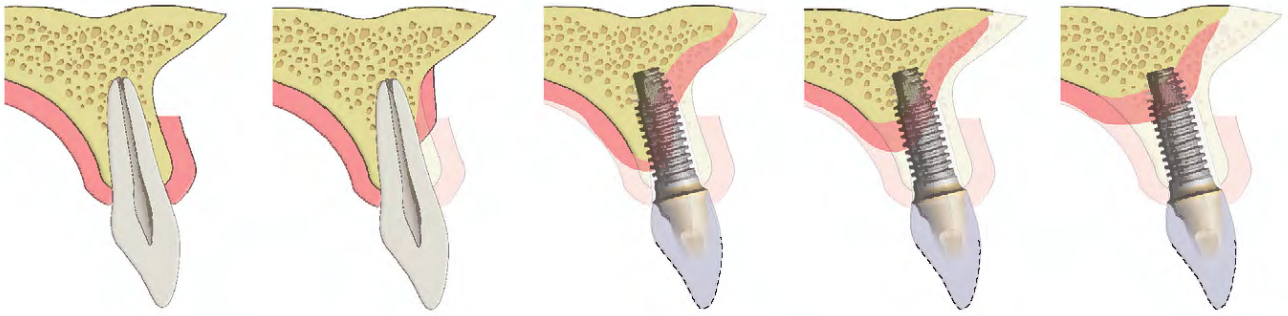


Figure 16-32 The initial bone loss on an anterior tooth before failure frequently occurs on the facial bone. Once the tooth is extracted, the bone volume is decreased in width and then height (division A, B, C–w, C–h).

WWW.HIGHDENT.IR

Figure 16-33 The interdental papillae are often depressed after initial socket healing.

Figure 16-34 A soft tissue–borne transitional device often moves (hence the name *flipper*). The movement against the tissue may compromise the developing bone and the soft tissue.

is often required in conjunction with implant therapy. Several authors have proposed additional parameters for predictable papillae that may guide the practitioner in the choice of the most appropriate modality.[108-111]

Transitional Prosthesis

The transitional restoration for a three-unit bridge is an acrylic fixed prosthesis that is fabricated the day the procedure is started. The transitional restoration for a single-tooth implant is often a removable prosthesis, which lacks stability and retention (hence the name *flipper*). Instead, it is strongly suggested that a resin-bonded fixed restoration be fabricated to provide improved function, especially when crestal bone regeneration is performed. The soft tissue–borne transitional restoration enhances crestal bone loss during the graft healing, may cause bone loss around the implant during Stage I healing (or even implant failure from the early loading), and may depress the interdental papillae of the adjacent teeth (Figure 16-34). As a result, a resin-bonded fixed prosthesis is fabricated for the extended healing, and a removable devise may be used short term for cosmetic emergencies should the device become debonded. When a resin-bonded restoration is used, the adjacent teeth are not prepared and the device is bonded to the tooth regions below the centric occlusal contacts of the teeth.

The transitional restoration for the single-tooth implant has the benefits of being off the soft tissue drape, the developing bone augmented site, and the healing implant-bone interface. Several options to the resin-bonded device permit these goals. An Essix appliance is an acrylic shell, similar to a bleaching tray, that has a denture tooth attached to replace the missing tooth.[112] This device is the easiest for tooth replacement postsurgical procedures. When an adjacent tooth requires a crown in the overall treatment plan, the adjacent tooth may be prepared and a cantilevered transitional FPD with a pontic over the surgical site may be used. When the patient requires orthodontics, a denture tooth and an attached bracket may be added to the orthodontic wire. A cast-clasp RPD with indirect rest seats to prevent rotation movements on the surgical site is also an excellent option.

SPECIFIC SINGLE-TOOTH IMPLANT INDICATIONS

Anodontia

The most common maxillary anterior tooth replaced by an implant is a central incisor lost from trauma (e.g., endodontic failure, fracture, root resorption) and/or a lateral incisor lost as a result of agenesis.[113-116] The absence of one or more teeth is known as *anodontia* and may be complete (very rare) or partial (also called *hypodontia*). It is many times more common than supernumerary teeth.[117] The primary cause of partial anodontia is familial heredity, and incidence ranges from 1.5% to as high as 10% in the U.S. population.[113] In addition, a number of syndromes exist in the literature that include multiple missing teeth, of which ectodermal dysplasia is the most common.

A high correlation is found between primary tooth absence and a permanent missing tooth; however, a missing tooth occurs more frequently in the permanent dentition. Caprioglio et al.[116] evaluated the records of almost 10,000 patients between the ages of 5 to 15 years of age. Of all the missing single teeth, the mandibular second premolar was most often missing (38.6%), followed by the maxillary lateral incisor (29.3%), the maxillary second premolar (16.5%), and the mandibular central incisor (4.0%). The remaining teeth were absent at a rate of only 0.5% to 1.8%, with the maxillary first molar being the least affected. The missing mandibular second premolar primarily occurred in male patients, whereas the maxillary lateral incisor primarily occurred in female patients (Figure 16-35). The most common multiple teeth lost (other than third molars) are the

WWW.HIGHDENT.IR

Figure 16-35 **A,** A periapical radiograph of a deciduous second molar and no permanent second premolar in a male patient. **B,** A periapical radiograph of a missing lateral incisor in a female patient.

maxillary lateral incisors, followed by the mandibular second premolars and maxillary second premolars. Congenitally missing teeth are therefore a common scenario in a general practice. Fortunately less than1% of those missing teeth are missing more than two teeth, and less than 0.5% of this group are missing more than five permanent teeth.

An emotional aspect exists to the replacement of a congenitally missing tooth. Because the cause is genetic, the parent with the genetic defect often feels a psychological healing when the implant returns their son or daughter to normal. An implant appears to be less traumatic, because the adjacent healthy teeth do not require preparation. These conditions make the parent eager for an implant, regardless of the time or cost of the procedure. However, if the bone graft, implant, or both should fail, then emotional consequences result. It is especially dangerous to put an adjacent tooth at risk under these conditions. If the young patient loses an adjacent tooth because of improper implant insertion or a consequence of a bone graft, then the patient-doctor relationship is stretched to the limit. As such, the clinician should use highly predictable augmentation procedures with care, ensuring that adequate space and bone are present before implant placement.

When acceptable conditions can be created, an anterior single-tooth implant is the treatment of choice for a congenitally missing anterior tooth (Figure 16-36). This condition is especially of benefit for a lateral incisor, because the ideal cervical region of the tooth is similar to the implant diameter. However, the roots of the adjacent natural teeth often impinge on the edentulous bone, or the mesiodistal length is insufficient. As a consequence, orthodontic therapy before implant placement should often be considered.[80,118] An additional advantage of orthodontics before or in conjunction with implant treatment for the congenital missing tooth is that the missing lateral incisor may be restored provisionally by a denture tooth attached to the orthodontic wire, to provide an esthetic replacement without trauma to the augmented ridge or implant during healing.

The missing maxillary lateral incisor is the tooth most often replaced with a dental implant, because the other orthodontic or prosthetic options are usually poor alternatives. The dentist should first determine whether space-opening (maintenance) procedures or space closure (orthodontics) is the treatment of choice for the missing tooth. The treatment options are usually different for a mandibular second premolar compared with a maxillary lateral incisor.

A congenital missing mandibular second premolar most often has a deciduous second molar. When the patient is 5 to 6 years old, the deciduous molar may be extracted. The permanent first molar may then erupt in a more mesial position. When the first deciduous molar is lost naturally (around the age of 9 to 11 years) the first permanent premolar and first molar may be orthodontically positioned adjacent to each other. This approach eliminates the need for a second premolar replacement. Because the second premolar space is eliminated with orthodontics, no bone graft, implant surgery, or crown (or combination of these treatments) is required to replace the tooth. Very few disadvantages exist to the use of orthodontics to eliminate this posterior missing tooth space.

When the deciduous second molar is maintained, it often becomes ankylotic. As a result, the opposing maxillary second premolar extrudes. Because the deciduous molar is 1.9 mm larger than a premolar, the mesiodistal space is larger than the usual premolar space after the deciduous molar is lost at a later date in the adult patient's life. The deciduous tooth does not

WWW.HIGHDENT.IR

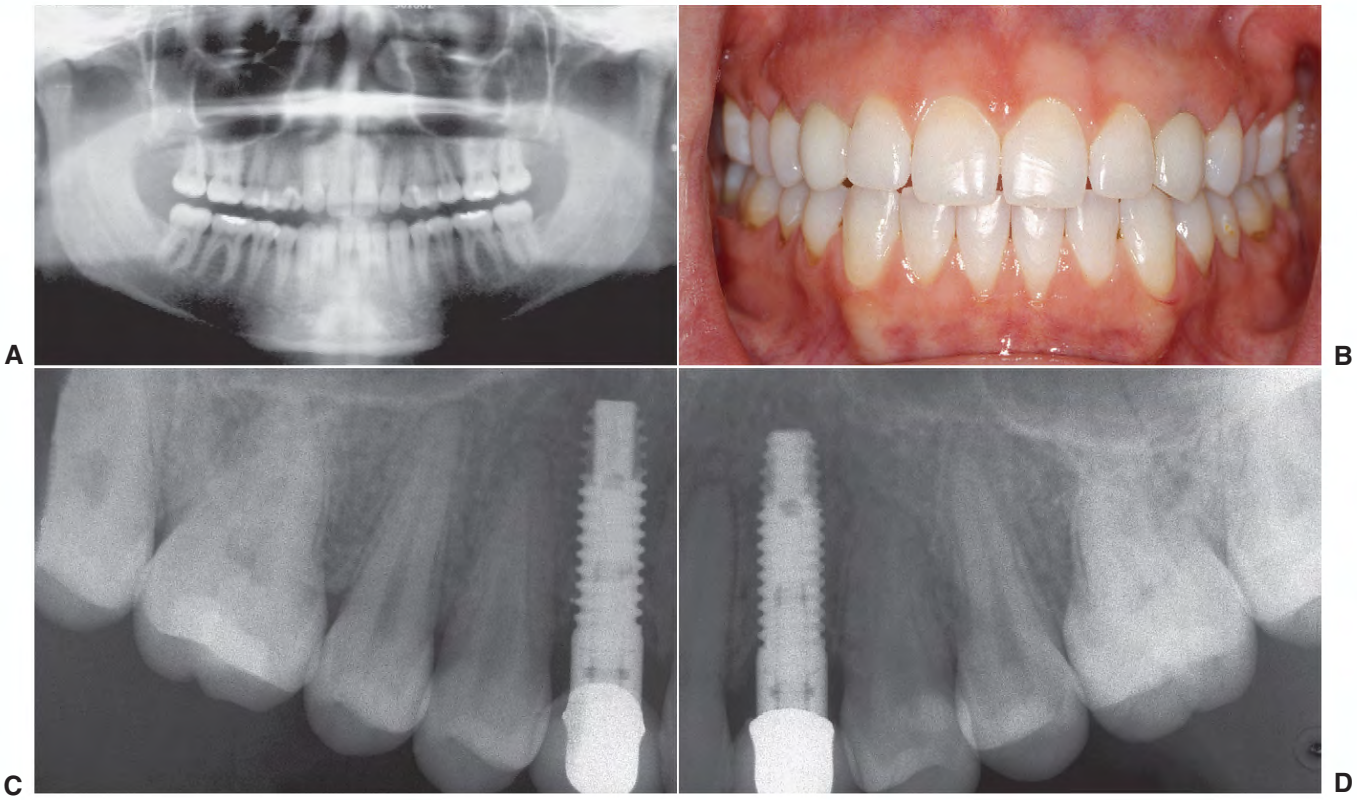


Figure 16-36 **A,** A panoramic radiograph of a young woman missing permanent canines bilaterally. The patient is older than 15 years, is able to menstruate, is taller than her mother, and had no statual height changes during the last 6 months. **B,** The missing canines are restored with single-tooth implant crowns. **C,** A periapical radiograph of the right canine implant and crown. **D,** A periapical radiograph of the left canine implant and crown. These radiographs demonstrate no crestal bone loss on the BioHorizons implants after 1 year.

have a buccolingual width of bone that is adequate for a larger-diameter implant. The crown for this larger tooth dimension is supported by a regular-size implant, which increases forces on the abutment screw and increases the risk of screw-loosening complications.

When the patient is missing a maxillary lateral incisor, space closure is less often indicated. When a maxillary canine is orthodontically moved to a lateral position, several compromises occur. The midline between the central incisors is often shifted to the missing tooth side. The canine eminence over the canine root is positioned under the nose, rather than in the usual position. This results in a depression, lateral to the naris, and a less full maxillary lip on one side of the midline. These differences are more evident as the patient ages. The maxillary canine is larger faciopalatally than mesiodistally. As a result, the cervical emergence is different from the contralateral incisor, even when restored with a laminate facing. The height of gingival contour is also higher than the lateral incisor on the other side of the arch. As a consequence of these limitations, a space-opening and maintenance procedure followed by an implant and single-crown restoration is usually the treatment of choice for a missing maxillary lateral incisor.

Graber[117] noted a strong correlation between a missing single tooth and altered tooth size, shape, or both. A common condition is a missing lateral incisor, where the contralateral lateral incisor is smaller than usual or a peg lateral. As such, the mesiodistal space is often limited to less than 6.5 mm. In these instances a nonfunctional, small-diameter implant of 2.5 to 3.0 mm may be considered. When the intratooth space is smaller than 5 mm, a cantilevered FPD from the canine abutment is considered.

Orthodontic Implant Site Development

In specific situations, the management of the patient in the early treatment phase may require orthodontics before the implant insertion to replace the missing tooth. Situations exist in which orthodontic site development is advantageous. If the patient has a missing tooth because of the loss of the permanent teeth, then it is likely that some soft and hard tissue reconstruction will be necessary to restore the proper context for the missing tooth. If bone height is insufficient and bone loss is also present on the adjacent teeth, then orthodontic extrusion of the adjacent teeth, followed by individual crowns, may be considered to improve the overall situation before bone grafting the edentulous site.

WWW.HIGHDENT.IR

Figure 16-37 Internal resorption may be stopped using endodontic therapy. This periapical radiograph indicates that structural failure of the tooth is eminent. Extraction of the canine is indicated.

In some cases, specifically congenitally missing teeth, the preparation of the implant site with orthodontic treatment can favorably influence the treatment outcome (presented in the next section). In other instances, when the patient has a failing tooth, a strategy may be developed to preserve as many features of the hard and soft tissue architecture with orthodontic extraction of the tooth instead of trying to recreate the tissues after tooth loss. The following are specific situations that illustrate the importance of early planning, not only for replacement of a tooth but also for the integration of hard and soft tissue management to restore the area.

For situations in which the doctor detects a missing lateral incisor in a child before the eruption of the permanent canine, Kokich[119] and Kokich et al.[120] proposed the following treatment modality:

1. The maxillary deciduous lateral incisor is prematurely extracted.
2. The permanent canine is encouraged to erupt in the missing lateral incisor position. In this way the bone around the canine (which is larger than usual in the site and may be greater than 7 mm in diameter) forms in the lateral incisor position.
3. The deciduous canine is extracted after the eruption of the permanent canine in the lateral position.
4. The canine is orthodontically retracted into the ideal canine position.
5. The remaining lateral incisor bone volume is abundant and ideal for an endosteal single-tooth implant.
6. After growth and development of the child has occurred, an implant may be inserted. In this manner, a bone graft will not be required before implant insertion, even when the implant procedure is delayed several years.

Box 16-16 Orthodontic Extraction

Advantages

- Improved bone volume
- Lower cost
- Less time
- Improved tissue drape
- Allows immediate extraction implant

Disadvantages

- Poor esthetics during treatment
- Higher patient cooperation
- Ankylosed teeth
- More difficult if porcelain-fused-to-metal crown on tooth

Root Resorption

Root resorption may cause the loss of a single anterior tooth. Two major categories of root resorption exist: (1) external and (2) internal. Each entity is related to trauma and consists of several different categories beyond the scope of this chapter. However, when structural failure is evident and the extraction of the tooth is eminent, two different treatment options related to the type of resorption exist.

Endodontic therapy is often warranted for internal root resorption and may stop the process. However, when structural failure has occurred or is likely, the treatment of choice is often orthodontic extraction[121] (Figure 16-37). The periodontal ligament space is still evident with internal root resorption. Orthodontic extrusion may be used to slowly extract the tooth with minimum risk of root fracture, which might result in labial plate bone loss and the need for more extensive augmentation. As the tooth migrates coronally during orthodontic extrusion, bone fills in the previous apical region. Because the tooth root is tapered, a 3-month extraction process produces sufficient movement so that the remaining root diameter in the bone is smaller than the implant diameter. As a consequence, after 3 months of orthodontic extrusion, no void exists around the implant at the time of extraction and implant insertion. An immediate implant insertion after extraction may be selected that fills the extraction site, yet is 1.5 to 2 mm away from each of the adjacent teeth.[121,122] In addition, the soft tissue drape often follows the coronal migration of the tooth and provides an improved condition for interdental papilla and soft tissue cervical profile for the implant reconstruction (Figure 16-38).

The time required for orthodontic extraction is often less than that required for bone regeneration after extraction and socket grafting (Box 16-16). The cost of treatment is usually less with orthodontic extraction

WWW.HIGHDENT.IR

than with hard and soft tissue grafting. The orthodontic extraction method is often the treatment of choice when a tooth requires extraction before implant insertion and when clinical conditions permit.

When external root resorption is the cause of structural failure of the tooth root, resorption may be taking place in conjunction with bone replacing the root defect. No evidence of a periodontal ligament space around the defect is seen, and orthodontic extrusion is not possible under these conditions. Instead the root is usually ankylosed, at least in the region of the lesion. Although some atraumatic techniques of extraction have been proposed for ankylosed teeth that core out the tooth from incorporated bone without encroaching on the lateral bone surfaces, they are not always applicable. The extraction of an ankylosed tooth often is accomplished at the cost of extensive labial bone loss and resultant bony wall defects. The bone graft needed to restore the defect is more of a clinical challenge than grafting a socket with intact bony walls. The soft tissue management of

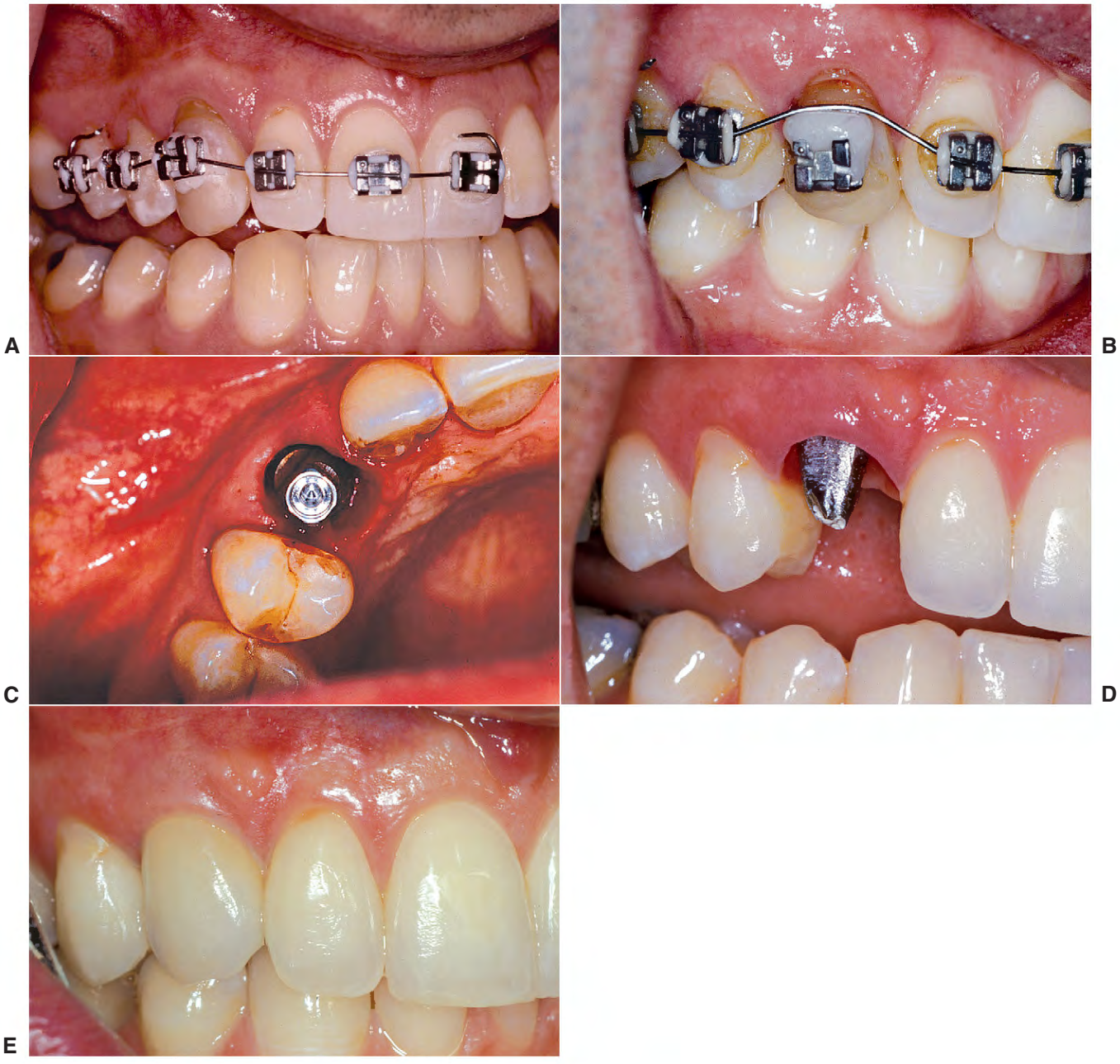


Figure 16-38 **A,** Orthodontic extrusion before extraction allows growth of the surrounding bone, a decrease in size of the root defect, and positioning of the soft tissue drape more incisal to compensate for shrinkage after extraction. **B,** The tooth is usually ready for extraction after 3 months. The canine orthodontic bracket is now near the incisal position of the tooth. **C,** The canine is extracted. If the bony walls are intact, then the implant may be inserted. Because the tooth root is tapered and has been extruded, the surrounding walls of bone are in direct contact with the implant. **D,** The implant and soft tissue drape after initial healing of 5 months. **E,** The implant crown in position replacing the maxillary right canine after orthodontic extraction.

WWW.HIGHDENT.IR

the area is an even greater task, because this situation often leads to the loss of interseptal bone height and loss of papilla height.

Because the replacement resorption is replacing the tooth with bone, consideration is given to delaying the extraction as long as possible. Once the structural failure is eminent , the remaining root segments may be cored out during the implant osteotomy procedure. As a result, the remaining tooth structures may be removed at the same time as the implant insertion. If the surgical defect is too large for immediate implant insertion, then the osteotomy is grafted and the implant procedure is delayed.

Remaining Maxillary Anterior Teeth

The anterior implant is successful only if the final restoration it supports is fully integrated within the adjacent dentition. The exponential growth of the field of implant dentistry has been paralleled by an exciting growth of esthetic dentistry and plastic regenerative surgery. This has brought the profession to realize that the restoration of the peri-implant soft and hard tissue to an optimal architecture is the key to a successful implant restoration. It is no longer sufficient to only achieve osteointegration with an implant. The implant restoration complex in the esthetic zone should be achieved in a context that respects all biological tissues.

When the goal of the restoring dentist for maxillary incisor single-tooth replacement is to obtain an ideal result, the practitioner should first evaluate not only the edentulous site but also the remaining anterior teeth. Because only one tooth is missing, the adjacent teeth most often dictate its length, contour, shape, and position. If not satisfactory, then a potential modification may need to be integrated into the overall treatment plan.

Figure 16-39 The patient's left maxillary central incisor was replaced with an implant and crown. The tooth is wider than the right central incisor. Orthodontics could have reduced the horizontal overjet of the lateral and central incisors and resulted in more symmetrical teeth. A second option is a veneer on the right central incisor to correct the rotation and make the natural tooth more symmetrical to the implant crown.

Parameters for a healthy esthetic anterior restoration have been established. The following guidelines have been proposed by esthetic and cosmetic dentistry colleagues. All these parameters play a determinant role in the final result and should not be overlooked. The patient must be educated about their present status before the onset of treatment, and the starting point should be documented. The patient, once fully informed of the existing discrepancies and their potential negative effect on the envisioned result, may decide to address and correct the existing problems of the adjacent teeth or simply elect to accept the compromise. Correction may be as simple as bleaching the remaining teeth or as complex as full esthetic rehabilitation with crown lengthening, soft tissue plastic surgery, veneers or crowns, and orthodontic therapy (or a combination of these procedures).

Tooth Size

The two maxillary central incisors should appear symmetrical and of similar size. This is most important to evaluate when the missing tooth is one central incisor (Figure 16-39). Outline asymmetry is visually acceptable the more distal from the midline that the eye travels. When one maxillary tooth is missing, the remaining space may be compromised from drifting of the adjacent teeth. Orthodontic correction is strongly encouraged when the missing tooth is a central incisor with a mesiodistal space less or more than the size of the corresponding central incisor.[123,124] The other option is to modify the existing central incisor with a veneer to make it similar in size and shape to the missing tooth restoration. This has the advantage of lowering the mesial interproximal contact and making the two centrals more square shaped, which decreases the height requirement of the papilla. The shades of the two centrals are also easier to match when made at the same time in the laboratory.

The average clinical crown length of the maxillary central incisor is 10.2 mm for a male patient and 9.4 mm for a female patient.[125] Surgical crown lengthening and longer anterior teeth may be indicated to reduce gingival exposure during a high-smile lip position. Because the clinical crown height of an implant-supported central incisor is often longer than the adjacent tooth, an esthetic crown lengthening on the natural tooth may be used to align the gingival margins. When an implant crown is longer than the corresponding natural tooth, a crown-lengthening procedure may be more predictable on the natural tooth than attempting to cover the implant crown with soft tissue. However, the clinical crowns of natural teeth are rarely more than 12 mm high.

The width of the average maxillary central is 8.6 mm for a male patient and 8.1 mm for a female patient.

WWW.HIGHDENT.IR

Figure 16-40 A triangular tooth shape has the steepest gingival scallop, and the interproximal bone is farthest from the tip of the papilla. After an extraction, shrinkage of the tissue makes the soft tissue drape in this tooth shape the most difficult to restore.

Although the male teeth are slightly longer and wider, the length-to-width ratio is similar to female teeth, (0.85 for male patients and 0.86 for female patients). A ratio range of 0.70 to 0.86 has been reported to be acceptable for the central incisors when they are similar. When the anterior teeth are made longer and both centrals have the same width, an acceptable result may be obtained.

The average clinical crown length of the maxillary lateral incisor is 8.7 mm for a male patient and 7.8 mm for a female patient. Therefore the average lateral incisor is almost 1.5 mm shorter than the central (at both the gingival region and the incisal edge). Gingival margins of the maxillary lateral incisors may be similar to centrals and canines, but should not be higher than the neighboring teeth. Therefore an implant crown on the lateral incisor should not be longer than the central or canine.

The average width of a lateral incisor is 6.6 mm for a male patient and 6.1 mm for a female patient, but this is more variable than for any other anterior tooth. The length-to-width ratio is slightly greater for a female patient (0.79, compared with the male patient ratio of 0.76). A lateral incisor space may be slightly narrower than the other natural tooth; however, when replacing the lateral incisor, it may be preferable to perform a slight mesial stripping of the adjacent canine to even out the size of the lateral incisors.

The average male canine clinical crown length is 10.1 mm and width is 7.6 mm, with a ratio of 0.77. The canine is essentially the same height as the central but 1 mm narrower—yet 1 mm wider than the lateral incisor. The female canine height averages 8.9 mm (0.5 mm shorter than the central) and 7.2 mm in width (1 mm narrower), with a ratio of 0.81. As a general rule, regardless of sex, the central incisor is 2 mm wider than the lateral incisor and 1 mm wider than the canine. However, on the horizontal plane, the canine is 1 to 2 mm shorter than the central incisor and corresponds to the curvature of the lower lip during smiling.

Tooth Shape

Three basic shapes of maxillary anterior teeth exist: (1) square, (2) ovoid, and (3) triangular. The tooth shape will influence the interproximal contact and the gingival embrasure. The square tooth shape is the most favorable to obtain an ideal soft tissue drape and papillae around the crown, because the interproximal contact is more apical and more tooth structure fills the interproximal region. In contrast, a triangular tooth shape has a more incisal interproximal contact, a steeper gingival scallop, and is farther from the interproximal bone (Figure 16-40). As a result, a space often exists between the interproximal contact and the interdental papilla of the remaining teeth. This is especially noteworthy to observe at the initial examination. When the soft tissue fills the interproximal space of the remaining anterior teeth that have a triangular shape, the tissues may be very liable and easily vanish during the healing phases after implant surgery. Care should be taken if the adjacent soft tissue requires reflection for a bone graft before the implant insertion. The ideal restoration of the soft tissue with a triangular-shaped tooth is less predictable.

The cervical embrasure of the adjacent teeth to the edentulous site should be particularly evaluated. A triangular tooth is narrow at the cervical, and the base of the interproximal tissue is wide. In addition, the adjacent tooth contact is often higher off the tissue, with an increased risk of a black triangular space. When such a condition is present on the adjacent teeth of the missing tooth, it is likely that the interdental papillae region will also be compromised on the implant crown.

The tooth shape also affects the topography of the underlying hard tissues. The roots of triangular tooth shapes are positioned farther apart and therefore have thicker facial and interproximal bone. This may decrease the amount of crestal bone loss after an extraction. In addition, the prognosis for an immediate implant insertion is more favorable, because the bone defect is smaller in diameter and the interproximal bone more likely to provide the recommended 1.5 mm or more of interproximal bone from the adjacent tooth. The square-shaped tooth is more likely to have less interproximal bone between the roots. Therefore it presents a greater risk of crestal or interproximal bone loss with an immediate implant insertion, making it less favorable for immediate implant insertion after extraction.

Soft Tissue Drape

The height of the maxillary lip when smiling (high lip line) is one of the most important criterion to evaluate when observing the cervical region of the maxillary anterior teeth. Its position is usually related to age, with older men showing the least amount of teeth and soft

WWW.HIGHDENT.IR

tissue and younger female patients displaying the most. Some patients (15% of male patients and 6% of female patients) show only the incisal half of the anterior teeth when they smile.[126] Those patients should be identified and told an ideal soft tissue result in the gingival region is not mandatory. Clinical results in emergence contours, interdental papilla presence, and even shade and contour of the crown are much less demanding. Therefore the extra surgeries and cost may not be necessary when these patients are willing to accept a slight compromise in ideal esthetics (Figure 16-41).

Ideally the height of the maxillary lip should rest at the junction of the free gingival margin on the facial aspect of the maxillary centrals and canine teeth.[126-128] Thus the interdental papillae are visible, but little gingival display is seen over the clinical crowns. Almost 70% of patients have this ideal smile position. A "gummy" smile displays more than 2 mm of soft tissue above the clinical maxillary crowns and is more acceptable in the female patient. It may occur in more than 14% of the female population and 7% of the male population. The higher the high lip line is, the more ideal the esthetic requirements are for the remaining teeth and for the single-tooth replacement. Therefore the existing maxillary anterior teeth condition is closely scrutinized when a high lip line exists and ideal results are desired.

The soft tissue drape of the remaining teeth should also be evaluated, especially if exposed during the high lip position of smiling. Under ideal conditions, soft tissue completely fills the interproximal space, with no dark triangles from the absence of light within the oral cavity. The interproximal contact between the maxillary central incisors should begin in the incisal third of the teeth and continue to the height of the central interdental papilla. In a healthy patient, very little to no space is seen between the papillae and interproximal contact. According to Kois,[129] the distance from the facial free gingival margin to the height of the central midinterproximal papilla is usually 4 to 5 mm; therefore the interdental papilla height is approximately 40% to 50% of the exposed tooth length.[129] Interproximal contacts at the incisal position start progressively more gingivally from central to canine. The greatest papilla height is often between the centrals, slightly lower between the centrals and laterals, and even lower between laterals and canines (Figure 16-42). However, the papilla height is often similar between the centrals and from the centrals to the laterals.

Under ideal conditions the osseous scallop of bone in the maxillary anterior region begins 2 mm below the CEJ midfacial to a point 3 mm more incisal in the interproximal region. The soft tissue follows this osseous scallop. A soft tissue biological dimension of approximately 3 mm in height above the bone is present at the midfacial position (1 mm above the CEJ) and 3 to 5 mm above the interproximal bone. Therefore if the interproximal contact is within 3 to 5 mm of the interproximal bone, then the interdental papilla will most often completely fill the space.

Figure 16-41 **A,** The maxillary left central incisor was restored with an implant restoration. The patient desired a soft tissue graft to cover the implant crest module. **B,** The high lip position during smiling did not display the cervical region of the patient's central incisors. Although this is not an ideal result, additional surgeries would not improve the crown's appearance within the esthetic zone, and the soft tissue pocket created may increase the risk of peri-implantitis.

Tarnow et al.[130] and Norland and Tarnow[131] measured the distance from the bottom of the interproximal contact to the vertical height of interproximal bone on natural teeth and observed how frequently the interproximal space would be completely filled by soft tissue. The distances ranged from 3 to 10 mm, with 88% of the contacts to the bone at 5, 6, or 7 mm; the most common measurement was 6 mm (40%), followed by 5 mm (25%) and 7 mm (22%) (Figure 16-43).

When the contact point to bone was 3 to 5 mm, the papilla almost always filled the space. When the contact was 6 mm, an absence of papilla was noted almost 45% of the time; at a 7-mm distance the papilla did not fill the space 75% of the time (Figure 16-44). In other words, a difference of 1 to 2 mm from the interproximal contact to the interseptal bone is very significant in relation to the interproximal soft tissue. Therefore it is critical to evaluate this dimension before implant surgery. If the height of the interproximal bone is lost or the interproximal contact is more incisal, then the soft tissue will less likely fill the interproximal space. In addition, contact distances to bone of 7 mm sometimes present a papilla initially, but after surgical reflection the

WWW.HIGHDENT.IR

Figure 16-42 The interdental papillae are often highest between the central incisor, with progressively less height as they proceed distal. The high lip line during smiling shows the interdistal papillae in more than 85% of the patients. (NOTE: The maxillary right lateral incisor is an implant restoration.)

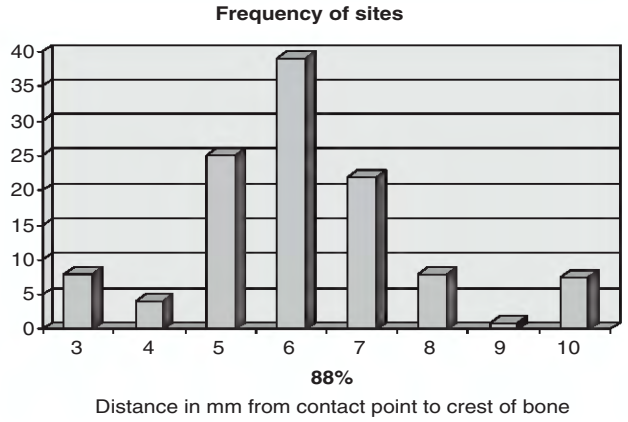


Figure 16-43 The distance from the interproximal contact to the crest of bone with natural teeth most often measures 5, 6, or 7 mm.[58] *(Data from Tarnow DP, Magner AW, Fletcher P: The effect of the distance from the contact point to crest of bone on the presence or absence of the interproximal papilla,* J Periodontol *63:995-996, 1992.)*

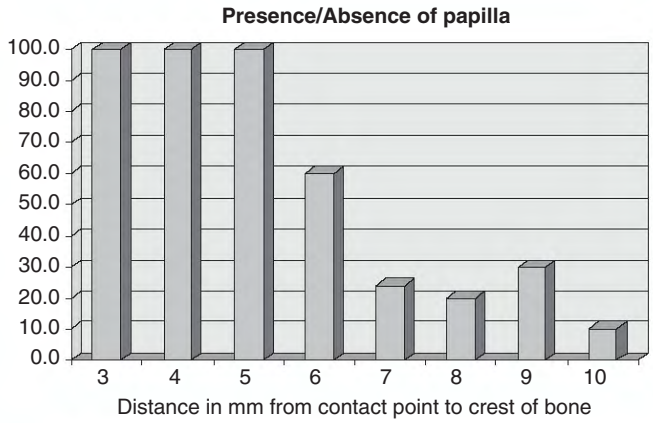


Figure 16-44 When the interproximal contact-to-bone distance is 5 mm or less, the interdental papilla completely fills the space between the teeth. When the contact-bone distance is 6 mm (the most common measurement), almost 40% of the time a black triangular space occurs between the teeth from the absence of papilla filling the space. A 7-mm or greater contact-bone distance nearly always has an incomplete fill of the interproximal space with the soft tissue.

chance this papilla will return to the original position may be less than 25%.

The higher the gingival scallop, or difference between the height of the papilla and the free gingival margin, the higher the risk for gingival loss after extraction. Likewise, once the tooth is extracted and an edentulous site is healed, the less likely the surgical and restorative procedures will be able to restore an ideal soft tissue contour. In contrast, a flatter gingival scallop and an interproximal tissue close to the osseous crest are conducive to minimal tissue shrinkage and a more ideal outcome.

The height of the facial gingival contour is in the middle of the tooth for the maxillary lateral incisors and the four mandibular anterior teeth. However, it is slightly to the distal on the central incisors and canines. The height of the free gingival margins of the two centrals are similar to both canines. The cervical height of the lateral incisors may be level or below the centrals and canines but symmetrical to each other. It may be easier to lengthen the cervical contour of the contralateral incisor when replacing a missing lateral incisor with an implant, rather than attempting to lower the gingival contour on the implant crown when gingiva and bone shrinkage has occurred. The least desirable gingival contour is seen when one anterior tooth is higher than the rest. Unfortunately this is a common occurrence with an implant crown when the bone and/or soft tissue is not augmented in conjunction with implant insertion or uncovery.

The color and texture of the tissue is also evaluated in the edentulous tooth site. The attached keratinized gingival tone and coral-pink color should be similar around the implant abutment compared with the healthy adjacent teeth. The biotype of the gingiva is usually called *thick* or *thin*. Thicker tissue is more resistant to the shrinkage or recession and more often leads to the formation of a periodontal pocket after bone loss. Thin gingival tissues around the teeth are more prone to shrinkage after tooth extraction and are more difficult to elevate or augment after tooth loss. Gingival recession is the most common esthetic complication of thin biotypes after anterior single-tooth extraction and is also a concern after implant surgery, uncovery, or both.

According to Kois,[108] predictability of the maxillary anterior single-tooth implant is ultimately determined by the patient's own presenting anatomy.[108] Favorable conditions include the following: (1) when the tooth position is more coronal relative to the full gingival margin, (2) square tooth shapes, (3) flat scallop periodontium forms, (4) thick periodontium biotypes, and (5) high (<3 mm) facial osseous crest positions of the teeth and midcrestal. Unfavorable patient anatomy

WWW.HIGHDENT.IR

includes the following: (1) aligned or apical preexisting tooth (relative to the free gingival margin), (2) triangular tooth shapes, (3) high scallop periodontium form, (4) thin periodontium types, and (5) low (>4 mm) facial osseous crest positions in relation to adjacent teeth and midcrestal.

IMPLANT CREST MODULE DESIGN

The two most common complications of anterior single-tooth implant replacement are abutment screw loosening and crestal bone loss. The crestal bone loss causes an increased risk of peri-implantitis or shrinkage of the tissue and poor cosmetic results. Both of these conditions are in part related to the implant crest module design. An implant body with an antirotational feature should be used for the single-tooth implant. The greater the dimension of the external or internal hex (or antirotational feature), the greater the resistance to shear forces once the abutment is inserted, which corresponds to a decrease in abutment screw loosening.

The crest module of an implant body should also be designed to transmit some compression and tensile forces to the crestal bone. Smooth metal on the crest module increases the crestal bone loss of the biological width (once the implant is uncovered) and transmits shear forces to the bone. Both these conditions increase the risk of crestal bone loss around the implant. The crestal bone loss may cause a shrinkage of the soft tissue drape and compromise esthetics (see Figure 16-41, *A*). Therefore smooth metal collars on the implant crest module should be limited to approximately 0.5 mm.

Implant Size

The first factor that influences the size of an implant is the mesiodistal dimension of the missing tooth. The average mesiodistal dimension of a central incisor is 8.6 mm for a male patient and 8.1 mm for a female patient. For a lateral incisor it is 6.6 mm for a male patient and 6.1 mm for a female patient, and for a canine it is 7.6 mm for a male patient and 7.2 mm for a female patient. However, the implant body should obviously not be as wide as the natural tooth or clinical crown. Otherwise, the emergence contour and interdental papillae region cannot be properly established.

The teeth become narrower as they proceed toward the bone. The mesiodistal dimensions of the maxillary central incisor at the cervix (ideally 1 mm below the free gingival margin) averages 6.4 mm; the lateral incisor dimension is 4.7 mm, and canine natural teeth at the cervix are 5.6 mm[99,100] (see Table 16-4). However, these dimensions are also too large for an implant.

The bone level on natural teeth is 2 mm below the CEJ, and the natural tooth dimensions at this bone level are reduced to 5.5 mm for central incisors, 4.3 mm for lateral incisors, and 4.6 mm for canines[132] (Figure 16-45). Therefore the latter dimensions most closely resemble an ideal implant diameter to mimic the emergence profile of a natural tooth. However, this ideal dimension is usually too large to adequately restore the soft tissue drape of the missing anterior tooth.

The second factor that determines the mesiodistal ideal implant diameter is the necessary distance from an adjacent tooth root.[102] Initial vertical bone loss around an implant during the first year of loading is variable and ranges from 0.5 to more than 3 mm. The horizontal dimension of a wedge-shaped bone defect around an implant from the biological width, implant design, or occlusal overload at the crest of the ridge is approximately 1.4 mm. When the implant is closer than 1.5 mm to an adjacent root, the wedge-shaped vertical defect may become a horizontal defect, causing bone loss on the adjacent tooth root (Figures 16-46 and 16-47).

The height of the interseptal (interimplant) bone in part determines the incidence of presence or absence of the interdental papillae between the teeth.[130] When the distance from the interseptal bone to interproximal contact is 5 mm or less, the papilla fills the space. When the distance is 6 mm, a partial absence of papilla exists 45% of the time; at 7 mm the risk of a compromise in the interproximal space is 75%. Therefore the intraseptal bone height is relative to the maintenance of the interdental papilla and should be preserved. As a consequence, the implant should be at least 1.5 mm from the adjacent teeth whenever possible, and the interseptal bone on the adjacent teeth should be within 5 mm of the eventual interproximal crown contact position.

In summary, two mesiodistal parameters determine the ideal implant size. The ideal width of the single-tooth implant should ideally correspond to the width of the

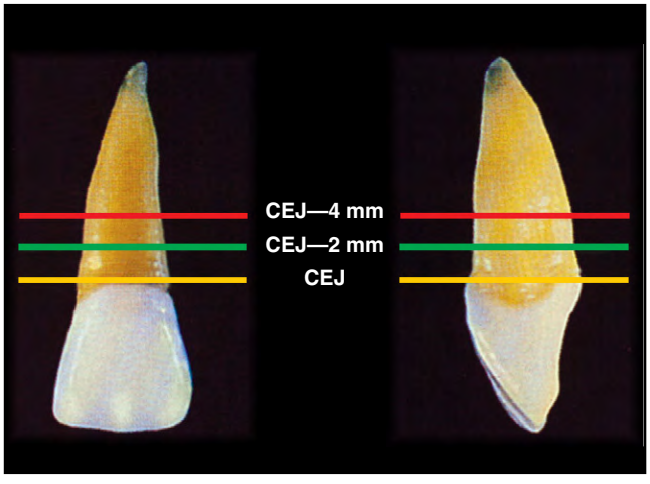


Figure 16-45 The ideal facial bone level on a natural tooth is 1 to 2 mm below the cement-enamel junction (CEJ) to accommodate for the connective tissue attachment zone The ideal implant size should relate to the root dimension at this level, with at least 1.5 mm of distance from the natural tooth on each side of the implant.

WWW.HIGHDENT.IR

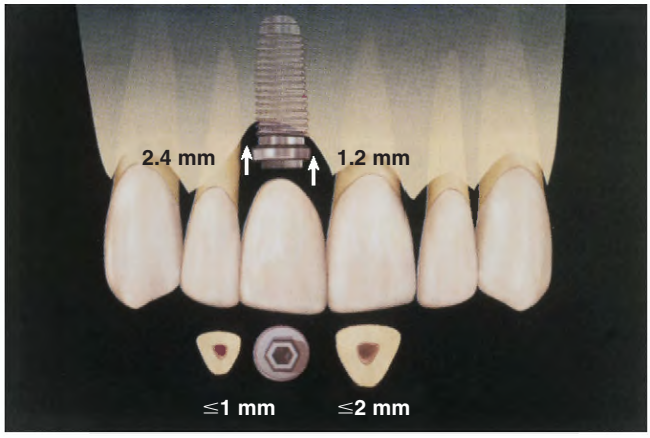


Figure 16-46 When the implant is placed closer than 1.5 mm next to the adjacent tooth, crestal bone loss on the implant (or tooth) results in a horizontal defect between both entities. Therefore 2-mm vertical bone loss on the implant can result in 2-mm bone loss on the tooth and an increase in the interproximal contact-bone distance. When the implant is more than 1.5 mm from the adjacent tooth, bone loss on the implant (or tooth) results in a vertical defect, and the interproximal contact-bone dimension remains the same.

missing natural tooth, 2 mm below the CEJ. However, the distance between the roots of the adjacent teeth should also be measured. The implant diameter plus 3 mm (1.5 mm on each side) should be equal to or less than the distance between the adjacent roots at the crest of the ridge (which is 2 mm below the interproximal CEJ).

The next dimension that determines the width of an anterior implant is the faciopalatal dimension of bone. The ideal width of bone would allow at least 1.5 mm on the facial aspect of the implant so that if a vertical defect forms around the crest module, that defect would not become horizontal and change the cervical contour of the facial gingiva (Figure 16-48). In a report by Spray et al.,[59] facial crestal bone loss incidence was greatest when the facial bone was less than 1.4 mm thick on the facial implant. However, the faciopalatal width dimension is not as critical on the palatal aspect of the implant. The reasons for this include the following: (1) the palatal bone is dense cortical bone and more resistant to bone loss, and (2) the palatal area is not within the esthetic zone, and a change in bone and soft tissue dimension is not an esthetic consequence. Therefore the faciopalatal width of bone at the crest for an anterior implant should have at least 1.5 mm on the facial, plus the dimension of the implant at the crest module, plus 1 mm on the palatal. Facial bone grafting at the time of implant insertion is frequently needed because the bone volume in width is often compromised.

The dimensions of the implant reflect the size of the crest module, not the implant body dimension. For example, a 4.1-mm crest module (on a 3.75-mm implant body) needs 7.1 mm of mesiodistal bone, a 3.5-mm crest module (on a 3.25-mm implant body) should have 6.5 mm of bone, and a 5.2-mm crest module requires 8.2 mm of bone.

A

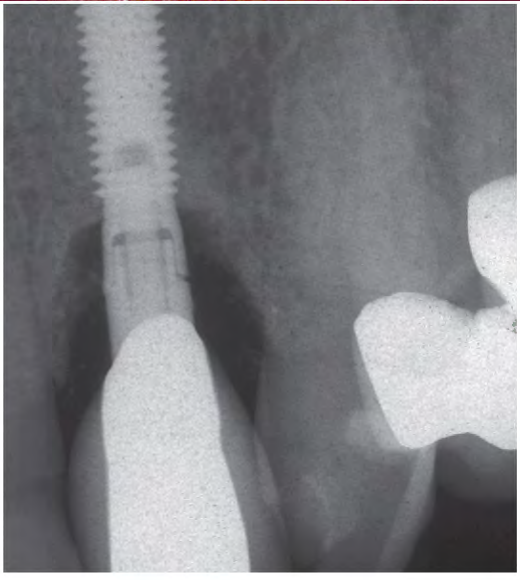

B

Figure 16-47 A clinical view **(A)** and radiographic image **(B)** of the consequence of an implant placed too close to the lateral incisor and crestal bone loss. The horizontal defect on the implant crown causes a black triangular space and bone loss on the implant and natural tooth. The gingival shrinkage resulting from the bone loss also increases the clinical crown dimension of the natural incisor and implant crown. The contact-bone distance has been maintained on one side, because the natural tooth interproximal bone is unaffected by the implant bone loss when the implant is greater than 1.5 mm from the tooth.

The ideal width of the implant should not only mimic the emergence of a natural tooth but also help preserve the bone and health of the adjacent teeth. The natural intraroot distance of the two central incisors distance is approximately 2 mm. However, the natural roots of the central to lateral and lateral to canine are often less than 1.5 mm apart, and often only 0.5 mm separates them. As a consequence, the ideal size of the single-tooth implant is usually *smaller* in diameter than the natural tooth root. Often the implant ideal diameters used to replace the average-size tooth result in a 4.2-mm to 5.2-mm implant for a central incisor, a 3.0- to 3.5-mm implant for a lateral incisor, and a 3.7- to 4.2-mm implant for a canine.

The difference in the emergence profile of a 4-mm-diameter implant and a 5-mm-diameter implant is

WWW.HIGHDENT.IR

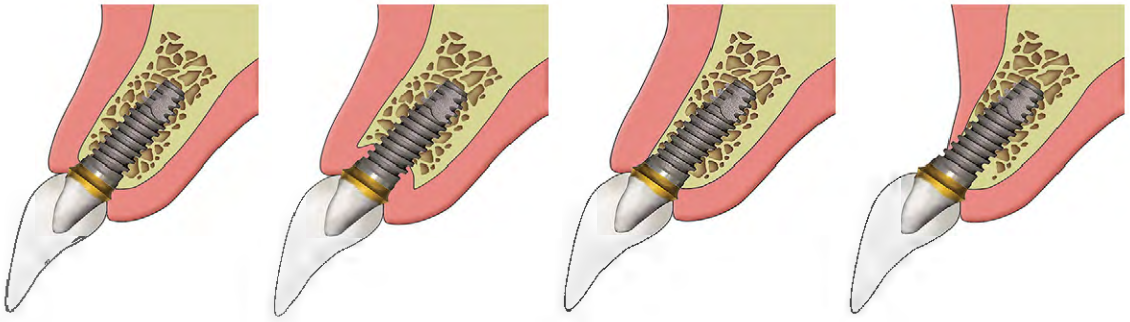


Figure 16-48 **A,** When the faciopalatal width of bone allows 1.5 mm or more of bone on the facial of the implant, crestal bone loss will not cause gingival shrinkage and maintain proper crown contour emergence **(B). C,** When the facial palatal bone is less than 1.5 mm thick, bone loss will be accompanied by soft tissue shrinkage and poor esthetics **(D).**

negligible and often not clinically relevant for an anterior tooth, because a 0.5-mm difference occurs on each side of the implant (Figure 16-49). Therefore when in doubt, the clinician should use an implant with a smaller diameter. As such, a 4-mm-diameter implant may often be used in the central implant position for a single-tooth replacement. Likewise, a 3.0- to 3.5-mm implant is often used for a lateral incisor single-tooth restoration. The exceptions to this rule may be their use in patients with moderate to severe bruxing or when placing an implant in the posterior regions of the mouth (where bite forces are higher). The larger-diameter implant will decrease abutment screw loosening, crestal bone loss, and risk of long-term implant body failure.

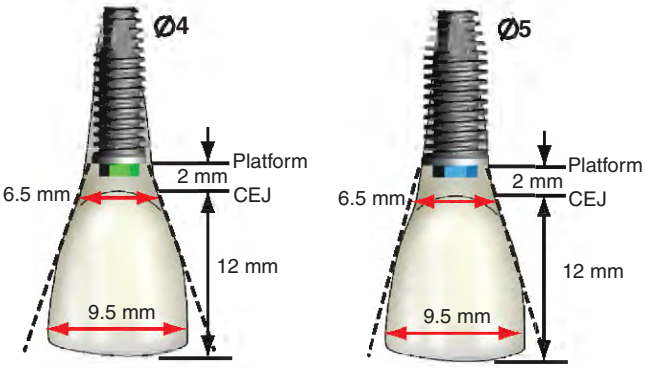


Figure 16-49 The emergence profiles of a 4-mm **(A)** and 5-mm **(B)** diameter implant are similar as the tooth emerges from the soft tissue drape. Placement of an implant slightly narrower than the natural tooth root often is indicated to decrease the risk of lateral perforation of the bone, intrusion on the incisive canal (for central incisors), or position next to the adjacent tooth root.

SUMMARY

A missing single tooth is a common scenario in restorative dentistry. The options for single-tooth replacement usually are an FPD or a single-tooth implant. Rarely are FPDs the primary treatment option in the posterior regions of the mouth. Abutment teeth caries and endodontic procedures place these teeth at increased risk of loss. On occasion, the posterior tooth may not be replaced (e.g., a mandibular second molar or a small space in which the surrounding teeth are interdigitated to prevent extrusion or tipping). The primary method of placement should be a single-tooth implant of adequate size, design, and material.

An anterior missing tooth more often may require the use of a traditional three-unit FPD or cantilever (to restore a lateral incisor). However, these indications are unusual and include adjacent tooth mobility and inadequate mesiodistal dimensions. When the intratooth is adequate and bone is present or can be created, the implant restoration is the treatment of choice.

It should be noted that use of an anterior single-implant crown to replace a central incisor is one of the more difficult procedures in dentistry. Several soft tissue, hard tissue, and implant surgical procedures may be necessary for an ideal result. Despite this, the single-tooth implant in the posterior and anterior regions of the mouth is the treatment of choice in the majority of patients.

References

1. Meskin LH, Brown LS: Prevalence and patterns of tooth loss in U.S. employed adult senior populations, *J Dent Educ* 52:686-691, 1988.
2. Hayden JW: Dental health services research utilizing comprehensive clinical data bases and information technology, *J Dent Educ* 61:47-55,1997.
3. Shugars DA, Bader JD, White BA et al: Survival rates of teeth adjacent to treated and untreated posterior bounded edentulous spaces, *J Am Dent Assoc* 129: 1085-1095, 1998.
4. Palmquist S, Swartz B: Artificial crowns and fixed partial dentures 18 to 23 years after placement, *Int J Prosthodont* 6:279-205, 1993.
5. Holm C, Tidehag P, Tillberg A et al: Longevity and quality of FPDs: a retrospective study of restorations

WWW.HIGHDENT.IR

30, 20 and 10 years after insertion, *Int J Prosthodont* 6: 283-289, 2003.
6. Priest GF: Failure rates of restorations for single tooth replacements, *Int J Prosthodont* 9:38-45, 1996.
7. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
8. Shillingburg HT, Hobo S, Whitsett LD et al: *Fundamentals of fixed prosthodontics*, ed 3, Chicago, 1997, Quintessence.
9. Cheung GSP, Dimmer A, Mellor R et al: A clinical evaluation of conventional bridgework, *J Oral Rehabil* 17:131-136, 1990.
10. Chalmers I: The Cochrane collaboration: preparing, maintaining, and disseminating systematic reviews of the effects of health care, *Ann N Y Acad Sci* 703:156-165, 1993.
11. Aquilino SA, Shugars DA, Bader JD et al: Ten year survival rates of teeth adjacent to treated and untreated posterior bounded edentulous spaces, *J Prosthet Dent* 85:455-460, 2001.
12. Carlsson GE, Hedegard B, Kiovumina KK: Studies in partial denture prosthesis IV: a 4 year longitudinal investigation of dentogingivally supported partial dentures, *Acta Odontol Scand* 23:443-472, 1965.
13. Bell B, Rose CL, Damon A: The Normative Aging Study: an interdisciplinary and longitudinal study of health and aging, *Int J Aging Hum Dev* 3:5-17, 1972.
14. Kapur KK, Glass RI, Leftus ER et al: The Veterans Affairs longitudinal study of oral health and disease, *Int J Aging Hum Dev* 3:125-137, 1972.
15. Hansson O: Clinical results with resin bonded prostheses and an adhesive cement, *Quintessence Int* 25:125-132, 1994.
16. Barrack G, Bretz WA: A long term prospective study of the etched-cast restoration, *Int J Prosthodont* 6:428-434, 1993.
17. Thompson VP, deRij KW: Clinical evaluation and lifetime predictions for resin-bonded prostheses. In Anusavice K, editor: *Quality evaluation of dental restoration: criteria for placement and replacement*, Chicago, 1989, Quintessence.
18. Wood M, Kern M, Thomson VP et al: Ten year clinical and microscopic evaluation of resin bonded restorations, *Quintessence Int* 27:803-807, 1996.
19. Misch CE: Single tooth implants—difficult, yet overused, *Dent Today* 11:46, 1992.
20. Fugazzotto PA: A comparison of the success of root resected molars and molar position implants in function in a private practice: results of up to 15-plus years, *J Periodontol* 72:113-123, 2001.
21. American Dental Association Survey Center: Changes in dental services rendered 1959-1990. In *ADA 1990 survey of dental services rendered*, Chicago, 1994, American Dental Association.
22. Johnston JE, Phillips RN, Dykema RW, editors: *Modern practice in crown and bridge prosthodontics*, Philadelphia, 1971, WB Saunders.
23. Creugers NH, Kayser HF, Van't Hof MA: A meta-analysis of durability data on conventional fixed bridges, *Community Dent Oral Epidemiol* 22:448-452, 1994.
24. Walton JN, Gardner FM, Agar JR: A survey of crown and fixed partial denture failures, length of service and reasons for replacement, *J Prosthet Dent* 56:416-421, 1986.
25. Schwartz NL, Whitsett LD, Berry TG: Unserviceable crowns and fixed partial dentures, life span and causes for loss of serviceability, *J Am Dent Assoc* 81:1395-1401, 1970.
26. Scurria MS, Bader JD, Shugars DA: Meta-analysis of fixed partial denture survival: prostheses and abutments, *J Prosthet Dent* 79:459-464, 1998.
27. Jackson CR, Skidmore AE, Rice RT: Pulpal evaluation of teeth restored with fixed prostheses, *J Prosthet Dent* 67:323-325, 1992.
28. Reuter JE, Brose MO: Failures in full crown retained dental bridges, *Br Dent J* 157:61-63, 1984.
29. Randow K, Glantz PO, Zoger B: Technical failures and some related clinical complications in extensive fixed prosthodontics: an epidemiological study of long-term clinical quality, *Acta Odontol Scand* 44:241-255, 1986.
30. Payne BJ, Locker D: Oral self-care behaviours in older dentate adults, *Community Dent Oral Epidemiol* 20: 376-380, 1992.
31. Bergenholtg G, Nyman S: Endodontic complications following periodontal and prosthetic treatment of patients with advanced periodontal disease, *J Peridontol* 55:63-68, 1984.
32. Jemt T, Lekholm U, Grondahl K: Three year follow up study of early single implant restoration ad modum Brånemark, *Int J Periodontics Restorative Dent* 10:340-349, 1990.
33. Andersson B, Odman P, Lidvall AM et al: Single tooth restoration supported by osseointegrated implants results and experience from a prospective study after 2 to 3 yrs, *Int J Oral Maxillofac Implants* 10:702-711, 1995.
34. Schmitt A, Zarb GA: The longitudinal clinical effectiveness of osseointegrated dental implants for single tooth replacement, *Int J Prosthodont* 6:187-202, 1993.
35. Ekfeldt A, Carlsson GE, Borjesson G: Clinical evaluation of single tooth restorations supported by osseointegrated implants: a restrospective study, *Int J Oral Maxillofac Implants* 9:179-183, 1994.
36. Cordioli G, Castagna S, Consolati E: Single tooth implants rehabilitation: a retrospective study of 67 implants, *Int J Prosthodont* 7:525-531,1994.
37. Haas R, Mensdorff Pouilly N, Mailath G et al: Brånemark single tooth implants: a preliminary report of 76 implants, *J Prosthet Dent* 73:274-279, 1995.
38. Jemt T, Laney WR, Harris D et al: Osseointegrated implants for single tooth replacement, a 1-year report from a multicenter prospective study, *Int J Oral Maxillofac Implants* 6:29-36, 1991.
39. Laney WR, Jemt T, Harris D et al: Osseointegrated implants for single tooth replacement: progress report from a multicenter prospective study after 3 years, *Int J Oral Maxillofac Implant* 9:49-54, 1994.
40. Henry PJ, Laney WR, Jemt T et al: Osseointegrated implants for single tooth replacement: a prospective 5-year multicenter study, *Int J Oral Maxillofac Implants* 11:450-455, 1996.
41. Becker W, Becker BE: Replacement of maxillary and mandibular molars with single endosseous implant restorations, a retrospective study, *J Prosthet Dent* 74:51-55, 1995.
42. Becker W, Becker BE, Alsuwyed A et al: Long-term evaluation of 282 implants in maxillary and mandibular molar positions: a prospective study, *J Periodontol* 70: 896-901, 1999.
43. Simon RL: Single implant-supported molar and premolar crowns: a ten-year retrospective clinical report, *J Prosthet Dent* 90:517-521, 2003.

WWW.HIGHDENT.IR

44. Levin L, Laviv A, Schwartz-Arad D: Long-term success of implants replacing a single molar, *J Periodontol* 77: 1528-1532, 2006.
45. Kline R, Hoar JE, Beck JH et al: A prospective multicenter clinical investigation of a bone quality based dental system, *Implant Dent* 11:224-234, 2002.
46. Misch CE, Poitras Y, Dietsh-Misch F: Endosteal implants in the edentulous posterior maxilla: rationale and clinical report *Oral Health*, 90:7-16, 2000.
47. Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study, *J Periodontol* 77: 1340-1347, 2006.
48. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
49. Bragger U, Krenander P, Lang NP: Economic aspects of single tooth replacement, *Clin Oral Implants Res* 16: 335-341, 2005.
50. Avivi-Arber L, Zarb GA: Clinical effectiveness of implant supported single tooth replacement: the Toronto study, *Int J Oral Maxillofac Implants* 11:311-321, 1996.
51. Bahat O, Handelsman M: Use of wide implants and double implants in the posterior jaw, a clinical report, *Int J Oral Maxillofac Implants* 11:379-386, 1996.
52. Balshi TJ, Hernandez RE, Pryzlak MC et al: A comparative study of one implant versus two replacing a single molar, *Int J Oral Maxillofac Implants* 11:372-378, 1996.
53. Balshi TJ, Wolfinger GJ: Two-implant-supported single molar replacement: interdental space requirements and comparison to alternative options, *Int J Periodontics Restorative Dent* 17:426-435, 1997.
54. Gomez-Roman G, Schulte W, d'Hoedt B et al: The Frialit-2 Implant System: 5-year clinical experience in single tooth and immediately post extraction applications, *Int J Oral Maxillofac Implants*, 12:209-309, 1997.
55. Muftu A, Chapman RS: Replacing posterior teeth with freestanding implants: four-year prosthodontic results of a prospective study, *J Am Dent Assoc* 8:1097-1998.
56. Boggan RS, Strong TT, Misch CE et al: Influence of hex geometry and prosthetic tooth width on static and fatigue strength of dental implants, *J Prosthet Dent* 82:436-440, 1999.
57. Binon PP: Evaluation of three slip fit hexagonal implants, *Implant Dent* 5:235-548, 1996.
58. Binon PP: The effect of implant/abutment hexagonal misfit on screw joint stability, *Int J Prosthodont* 9:149-160, 1996.
59. Spray JR, Black CG, Morris HF et al: The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering, *Ann Periodontol* 5:119-128, 2000.
60. Small PN, Tarnow DP, Cho SC: Gingival recession around wide-diameter versus standard-diameter implants: a 3- to 5- year longitudinal prospective study, *Pract Proced Aesthet Dent* 13:143-146, 2001.
61. Malevez C, Hermans M, Daelemans P: Marginal bone levels at Brånemark system implants used for single tooth restoration. The influence of implant design and anatomical region, *Clin Oral Implants Res* 7:162-169, 1996.
62. Sullivan DY: Wide implants for wide teeth, *Dent Econ* 84:82-83, 1994.
63. Rangert B, Krogh PH, Langer B et al: Bending overload and fixture fracture: a retrospective clinical analysis, *Int J Oral Maxillofac Implants* 10:326-334, 1995.
64. Langer B, Langer L, Herrman I et al: The wide fixture: a solution of special bone situations and a rescue for the compromised implant, *Int J Oral Maxillofac Implants* 8:400-408, 1993.
65. Davarpanah M, Martinez H, Kibir M et al: Wide-diameter implants: new concepts, *Int J Periodontics Restorative Dent* 21:149-159, 2001.
66. Sato Y, Shindoi N, Hosokawa R: A biomechanical effect of wide implant placement and offset placement of three implants in the posterior partially edentulous region, *J Oral Rehabil* 27:15-21, 2000.
67. Ivanoff C-J, Grondahl K, Sennerby L et al: Influence of cariations in implant diameters: a 3- to 5-year retrospective clinical report, *Int J Oral Maxillofac Implants* 14:173-180, 1999.
68. Renouard F, Arnoux JP, Sarment D: Five mm diameter implants without a smooth surface collar. Report on 98 consecutive placements, *Int J Oral Maxillofac Implants* 14:101-107, 1999.
69. Bakaeen LG, Winkler S, Neff PA: The effect of implant diameter, restoration design, and occlusal table variations on screw loosening of posterior single-tooth implant restorations, *J Oral Implantol* 27:63-72, 2001.
70. Geramy A, Morgano SM: Finite element analysis of three designs of an implant-supported molar crown, *J Prosthet Dent* 92:434-440, 2004.
71. Tarnow DR, Cho SC, Wallace SS: The effect of inter-implant distance on the height of inter-implant bone crest, *J Periodontol* 71:546-549, 2000.
72. Watson MT: Implant dentistry, a 10 year retrospective report, *Dent Prod Rep* 25-32, December 1996.
73. American Dental Association Survey Center: *ADA 2000 Survey of current issues in dentistry: surgical dental implants*, Chicago, 2002, American Dental Association.
74. Zuolo ML, Ferreira MO, Gutmann JL: Prognosis in periradicular surgery: a clinical prospective study, *Int Endod J* 33:91-98, 2000.
75. Sjogren U, Hagglund B, Sundqvist G et al: Factors affecting long-term results of endodontic treatment, *J Endod* 16:498-504, 1990.
76. Weiger R, Axman-Kremar D, Lost C et al: Prognosis of conventional root canal treatment revisited, *Endod Dent Traumatol*, 14:1-9, 1998.
77. Priest G: Single-tooth implants and their role in preserving remaining teeth: a 10 year survival study, *Int J Oral Maxillofac Implants* 14:181-188, 1999.
78. Tan K, Pjetursson BE, Lang NP et al: A systematic review of the survival and complication rates of fixed partial dentures (FPDs) after an observation period of at least 5 years, *Clin Oral Implants Res* 15:654-666, 2004.
79. Priest G, Priest J: How to profit from implants. The economics of implants for single missing teeth, *Dent Econ* 130-138, 2004.
80. Misch CE, D'Alessio R, Misch-Dietsh F: Maxillary partial anodontia and implant dentistry-maxillary anterior partial anodontia in 255 adolescent patients: a 15-year retrospective study of 276 implant site replacement, *Oral Health* 95:45-57, 2005.
81. Wennstrom JL, Ekestubbe A, Grondahl K et al: Implant-supported single-tooth restorations: a 5-year prospective study, *J Clin Periodontol* 32:567-574, 2005.
82. Zarone F, Sorrentino R, Vaccaro F: Prosthetic treatment of maxillary lateral incisor agenesis with osseointegrated implants: a 24-39 month prospective clinical study, *Clin Oral Implants Res* 17:94-101, 2006.

WWW.HIGHDENT.IR

83. Creugers NH, Kreuler PA, Snoek RJ et al: A systematic review of single tooth restorations supported by implants, *J Dent* 28:209-217, 2000.
84. Lindhe T, Gunne J, Tillberg A: A meta-analysis of implants in partial edentulism, *Clin Oral Implants Res* 9:80-90, 1998.
85. Kemppainen P, Eskola S, Ylipaavalniemi A: Comparative prospective clinical study of two single tooth implants: a preliminary report of 102 implants, *J Prosthet Dent* 77:382-387, 1997.
86. Kan JY, Rungcharassaeng K: Immediate implant placement and provisionalization of maxillary anterior single implants: a surgical and prosthodontic rationale, *Pract Periodontics Aesthet Dent* 12:817-824, 2000.
87. Choquet V, Hermans M, Adriaenssens P et al: Clinical and radiographic evaluation of the papilla level adjacent to single tooth dental implants: a retrospective study in the maxillary anterior region, *J Periodontol* 72:1364-1371, 2001.
88. Groisman M, Frossard WM, Ferreira H et al: Single tooth implants in the maxillary incisor region with immediate provisionalization: 2-year prospective study, *Pract Proced Aesth Dent* 15:115-122, 2003.
89. Krenmair G, Pichslinger, E, Wagner H: Status of teeth adjacent to single-tooth implants, *Int J Prosthodont* 16:524-528, 2003.
90. Misch CE: The heath-esthetic compromise in implant dentistry, *Compend Contin Dent Ed* 18:11-19, 1997.
91. Misch CE: The maxillary anterior single tooth implant aesthetic-health compromise, *Int J Dental Symp* 3:4-9, 1995.
92. Odman, J, Grondahl K, Lekholm U et al: The effect of osseointegrated implants on the dento-alveolar development: a clinical and radiographic study in growing pigs, *Eur J Orthod* 3:279-286, 1991.
93. Thilander B, Odman J, Grondahl K et al: Osseointegrated implants in adolescents: an alternative in replacing missing teeth? *Eur J Orthod* 16:84-95, 1994.
94. Jemt T: Measurements of tooth movements in relation to single-implant restorations during 16 years: a case report, *Clin Implant Dent Relat Res* 7:200-208, 2005.
95. Bernard JP, Schatz JP, Christou P et al: Long-term vertical changes of the anterior maxillary teeth adjacent to single implants in young and mature adults: a retrospective study, *J Clin Periodontol* 31:1024-1028, 2004.
96. Thilander B, Odman J, Jemt T: Single implants in the upper incisor region and their relationship to the adjacent teeth: an 8-year follow-up study, *Clin Oral Implants Res* 10:346-355, 1999.
97. Oosterle LJ: Implant considerations in the growing child. In Higuchi KW, editor: *Orthodontic applications of osseointegrated implants*, Chicago, 2000, Quintessence.
98. Op Heij DG, Opdebeeck H, Steenberghe DV et al: Age as compromising factor for implant insertion, *Periodontol 2000* 33:172-184, 2003.
99. Westwood RM, Duncan JM: Implants in adolescents: a literature review and case reports, *Int J Oral Maxillofac Implants* 11:750-755, 1996.
100. Wheeler RC: *A textbook of dental anatomy and physiology,* ed 4, Philadelphia, 1965, Lea & Febiger, pp 185-283.
101. Tarnow DP, Eskow RM: Preservation of implant esthetics, soft tissue and restorative considerations, *J Esthet Dent* 8:12-19, 1996.
102. Tarnow DP, Cho SC, Wallace SS: The effect of interimplant distance on the height of interimplant bone crest, *J Periodontol* 71:546-549, 2000.
103. Misch CE, Wang HL: The procedures, limitations and indications for small diameter implants and a case report, *Oral Health* 94:16-26, 2004.
104. Misch CE: Treatment options for congenitally missing lateral incisor—a case report, *Dent Today* 23:92-95, 2004.
105. Vigolo P, Givani A, Majzoub Z et al: Clinical evaluation of small-diameter implants in single-tooth and multiple-implant restorations: a 7-year retrospective study, *Int J Oral Maxillofac Implants* 19:703-709, 2004.
105a. Becker W, Ochsenbein C, Tibbetts L et al: Alveolar bone anatomic profiles as measured from dry skulls: clinical manifestations, *J Clin Periodontol* 24:727-731, 1997.
106. Misch CM, Misch CE: The repair of localized severe ridge defects for implant placement using mandibular bone grafts, *Implant Dent* 4:261-267, 1995.
107. Misch CM, Misch CE, Resnik RR et al: Reconstruction of maxillary alveolar defects with mandibular symphysis grafts for dental implants, a preliminary procedural report, *Int J Oral Maxillofac Implants* 7:360-366, 1992.
108. Kois JC: Predictable single tooth peri-implant esthetics—five diagnostic keys, *Compend Contin Educ Dent* 22:199-218, 2001.
109. Salama H, Salama M, Garber DA et al: Techniques for developing optimal peri-implant papillae within the esthetic zone: guided soft tissue augmentation, *J Esthet Dent* 7:125-129, 1995.
110. Andersson B, Odman P, Lidvall AM et al: Single tooth restoration supported by osseointegrated implants: results and experience from a prospective study after 2 to 3 years, *Int J Oral Maxillofac Implants* 10:702-711, 1995.
111. Furhauser R, Florescu D, Benesch T et al: Evaluation of soft tissue around single tooth implant crowns: the pink esthetic score, *Clin Oral Implants Res* 16:639-644, 2005.
112. Sheridan J, Ledoux W, McMinn R: Essix technology for the fabrication of temporary anterior bridges, *J Clin Orthod* 18:482-486, 1994.
113. Maklin M, Dummett CO Jr, Weinberg R: A study of oligodontia in a sample of New Orleans children, *J Dent Child* 46:478-482, 1979.
114. Holland T, Mullane DO, Clarksson J et al: Trauma to permanent teeth of children, aged 8, 12 and 15 years, in Ireland, *J Paediatr Dent* 4:13-16, 1988.
115. Silverman NE, Ackerman JL: Oligodontia: a study of its prevalence and variation in 4,032 children, *J Dent Child* 46:470-477, 1979.
116. Caprioglio D, Vernole B, Aru G et al: *Le agenesie dentali*, Milan, Italy, 1988, Masson, pp 1-14.
117. Graber TM: Anomalies in number of teeth. In Graber TM, editor: *Orthodontics principles and practice,* ed 2, Philadelphia, 1966, WB Saunders.
118. Kinzer GA, Kokich VO Jr: Managing congenitally missing lateral incisors. III. Single-tooth implants, *J Esthet Restor Dent* 17:202-210, 2005.
119. Kokich V: Esthetics and anterior tooth position: an orthodontic perspective. III. Mediolateral relationships, *J Esthet Dent* 5:200-207, 1993.
120. Kokich VG, Crabill KE: Managing the patient with missing or malformed maxillary central incisors, *Am J Orthod Dentofacial Orthop* 129(suppl 4):S55-S63, 2006.
121. Salama H, Salama M, Kelly J: The orthodontic periodontal connection in implant site development, *Pract Periodont Aesthet Dent* 8:923-932, 1996.
122. Salama H, Salama M, Garber D et al: The interproximal height of bone: a guidepost to predictable aesthetic

WWW.HIGHDENT.IR

strategies and soft tissue contours in anterior tooth replacement, *Pract Periodont Aesthet Dent* 10:1131-1141, 1998.
123. Kokich VG, Spear FM, Kokich VO: Maximizing anterior esthetics: an interdisciplinary approach: esthetics and orthodontics. In McNamara JA, editor: *Craniofacial growth series*, Ann Arbor, Mich, 2001, Center for Human Growth and Development, University of Michigan.
124. Lombardi RE: The principles of visual perception and their clinical application to denture esthetics, *J Prosthet Dent* 29:358, 1973.
125. Sterett JD, Olivier T, Robindon F et al: Width and length ratios of normal clinical crowns of maxillary anterior dentition in man, *J Clin Periodontol* 26:153-157, 1999.
126. Tjan AH, Miller GD, The JG: Some esthetic factors in a smile, *J Prosthet Dent* 51:24-28, 1984.
127. Kokich V: Esthetics and anterior tooth position: an orthodontic perspective. Part I. Crown length, *J Esthet Dent* 5:19-23, 1993.
128. Kokich VO Jr, Kiyak AH, Shapiro PA: Comparing the perception of dentists and lay people to altered dental esthetics, *J Esthet Dent* 11:311-324, 1999.
129. Kois MCJ: Altering gingival levels: the restorative connector. I. Biologic variables, *J Esthet Dent* 6:3-9, 1994.
130. Tarnow DP, Magner AW, Fletcher P: The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal papilla, *J Periodontol* 63:995-996, 1992.
131. Nordland WP, Tarnow DP: A classification system for loss of papillary height, *J Periodontol* 69:1124-1126, 1998.
132. Hebel KS, Gajjar K: Achieving superior esthetic results: parameters for implant and abutment selection, *Int J Dent Symp* 4:42-47, 1997.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 17

Maxillary Arch Implant Considerations: Fixed and Overdenture Prostheses

Carl E. Misch

More than 18 million people, or 10.5% of the adult population of the United States, are completely edentulous. Maxillary dentures usually are tolerated better by patients than their mandibular counterparts. As such, many treatment plans initially concentrate on the problems associated with the mandibular denture (Figure 17-1). However, once the patient enjoys a stable, retentive, and perhaps fixed mandibular prosthesis, often the patient's attention is brought to the inadequacies of the maxillary prosthesis. In addition to this segment of the population without any teeth, 7% of the adult population wears a maxillary denture opposing some remaining mandibular teeth. This means that 17% of the U.S. population (30 million adults) have no natural maxillary teeth.

The first chapter of this book addressed the esthetic and psychological consequences of the loss of maxillary teeth. Once patients become aware of the anatomical and esthetic consequences of missing teeth, the desire for implant restorations increases. As a result of patient and doctor education related to the loss of teeth, implant restoration of the edentulous maxilla will become more prevalent.

EDENTULOUS ANTERIOR MAXILLA

In a 20-year review of the literature compiled by Goodacre et al., restorations associated with the edentulous maxilla have the highest early implant failure rate compared with any other situation. In this review, overdentures in the maxillary arch averaged 19% implant failure. Fixed prostheses in the edentulous maxilla had an early implant failure of 10%. In comparison, mandibular overdentures or fixed restorations demonstrated a 3% implant failure rate.[1]

Several factors affect the condition of the edentulous maxilla and may result in a decrease in implant survival or an increase in prosthetic complications. The facial cortical plate of the premaxilla is thin over the roots of the teeth and may be resorbed from periodontal disease or is often fractured during the extraction of these teeth. In addition, the facial cortical plate rapidly resorbs during initial bone remodeling, and the anterior ridge loses 25% of its width within the first year after tooth loss and 40% to 50% over 1 year, mostly at the expense of the labial plate. As a result, the residual available bone migrates to a more palatal position.[2-7]

The patient is more likely to wear and functionally accommodate a maxillary complete denture compared with its mandibular counterpart. The greater retention, support, and stability compared with the lower restoration are well documented. As such, the patient often is able to wear the maxillary removable prosthesis for longer periods before complications arise. From a patient's perspective, the need to replace the denture is more related to a desire for a fixed restoration as the motivating factor. By the time the patient notices problems of stability and retention caused by resorption of the premaxilla, the maxillary bone often has advanced atrophy and may be Division C–h or D in volume. Therefore the completely edentulous, anterior bony

Figure 17-1 Implant treatment plans for the completely edentulous patient often address the mandible, as it is the least stable unit. The maxilla is often restored with a traditional denture.

WWW.HIGHDENT.IR

ridge is often inadequate for ideal endosteal implant insertion.

As the bone resorbs from Division B to C–w in the anterior edentulous mandible, the cross section of the residual ridge is triangular (with a wide base). As a consequence, an osteoplasty removes the narrower crestal bone and the residual ridge becomes wider, often converted to a Division A bone volume. In the maxilla, however, the Division B to C–w crest often remains narrow almost to the floor of the nose. An osteoplasty to gain bone width results in a Division C–h to D ridge. Therefore bone augmentation is more often required in the anterior maxilla compared with the anterior mandible.

It is the doctor's responsibility to inform the patient about the continued bone loss in the maxilla before complications arise. Bone grafting is much more predictable for width gains rather than increases in height. Division B bone grafting may use a synthetic bone component for the graft; Division C–w often requires autologous bone from the mandible as a donor. However, when an edentulous maxilla requires height augmentation (C–h or D), the dentist often must resort to the iliac crest or other extraoral donor sites for large volumes of bone. As such, the maxillary completely edentulous patient should understand that the surgical rehabilitation is much more complex and extensive as the volume of bone needed to reconstruct the atrophic maxilla increases. Therefore notifying patients of their continued maxillary bone loss is even more important than in the anterior mandible, rather than waiting until problems with their removable restoration develop.

In most patients with available bone, the bone is less dense in the anterior maxilla than in the anterior mandible, where a dense cortical layer surrounds coarse trabeculae of adequate bone strength to provide implant support. In contrast, the maxilla presents thin porous bone on the labial aspect, very thin porous cortical bone on the floor of the nasal and sinus region, and a more dense cortical bone on the palatal aspect.[8,9] The trabecular bone is usually fine and is less dense than the anterior region of the mandible. The trabecular bone of D3, often found in the maxilla, is 45% to 65% weaker than the trabecular bone of D2, usually found in the anterior mandible.

To achieve predictable esthetics for a maxillary full-arch fixed prosthesis, the hard and soft tissue, volume, and character should be adequate in most aspects. Available bone should be evaluated closely for implant insertion in esthetic regions because of its influence on the soft tissue drape, implant size, implant insertion (angulation and depth), and the final prosthetic result. Bone loss after maxillary anterior tooth loss is rapid and has considerable consequences. Therefore most maxillary anterior edentulous sites require at least some bone and soft tissue augmentation before or during implant insertion and at implant uncovery.

Box 17-1 Premaxilla Compromised Anatomical Conditions

- Narrow ridge
- Esthetics required—facial cantilevers
- Oblique centric contacts
- Increased moment loads with lateral forces during mandibular excursions
- Reduced trabecular bone density
- Absence of thick cortical plate
- Accelerated bone loss in incisor region
- Crown height space often greater than ideal

The farther forward the maxillary anterior teeth are positioned from the implants, the greater the moment force leverage on the bone-implant interface, abutment screws, and implants. Yet many dentists attempt to do plastic surgery with plastic, hoping to eliminate vertical lines in the lip by bulking out the labial flange and teeth of an overdenture and positioning the teeth farther forward than the natural tooth position. Patients desiring to eliminate wrinkles in the lips from bone loss should have plastic surgery and bone augmentation, not plastic added to a maxillary prosthesis. This is even more important when the patient desires a fixed prosthesis. Bone and soft tissue augmentation is usually required to restore the natural appearance of the face without the help of a labial denture flange when a fixed restoration is planned. The facial position of the lip relative to esthetics is an important criterion to evaluate at the onset of treatment, before the placement of the implants. This criterion alone may indicate an overdenture rather than a fixed prosthesis.

From a biomechanical perspective, the implant-restored anterior maxilla is often the weakest section compared with other regions of the mouth. Compromised anatomical conditions and their consequences include the following (Box 17-1):

1. Narrow ridges form soon after tooth extractions. Bone augmentation is often necessary and may mandate the need for smaller-diameter implants. Their use results in increased stress concentrations in the implant and contiguous interfacial tissues, particularly at the crestal region.
2. In the premaxilla, esthetics and phonetics dictate that the replacement teeth be placed at or near their original position, often cantilevered from the implants and the residual ridge, which usually is resorbed palatally and superiorly. The use of facial cantilevers results in increased moment loads at the implant crest and often leads to localized crestal remodeling bone loss and soft tissue recession.
3. The arc of closure of the mandible is anterior to the maxillary residual ridge; as a consequence, the moment force is great against the maxillary anterior

WWW.HIGHDENT.IR

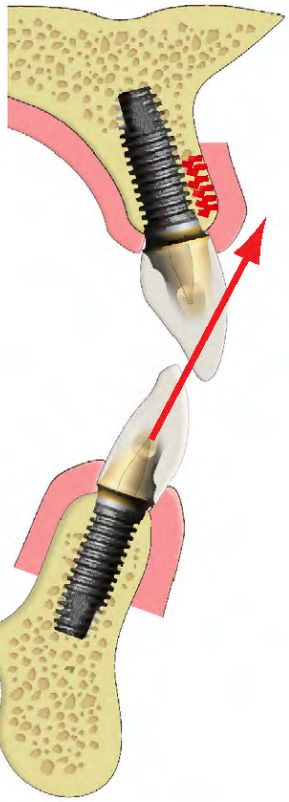


Figure 17-2 The arc of closure is anterior to the maxillary ridge and, as a result, increased forces are applied to the thinner facial cortical plate.

Figure 17-3 Mandibular excursions place lateral forces on maxillary anterior implants, which increase the resultant stresses.

crowns supported by implants. The force is also directed against the thinner facial bone (Figure 17-2). Oblique centric contacts result in potentially harmful, off-axis load components.

4. All mandibular excursions place lateral forces on the maxillary anterior teeth, with resulting increased stress on the crestal bone of the supporting implants, especially on the labial aspect. These lateral loads in excursion further increases the moment loads applied to the implant (Figure 17-3).
5. A mandibular arch receives a load from the outside of the arch toward the center (on the top left). An arch is constructed for this force direction. A maxillary arch receives a force from within the arch to the outside of the structure (on the right). An arch is not as effective to resist this type of force (Figure 17-4).

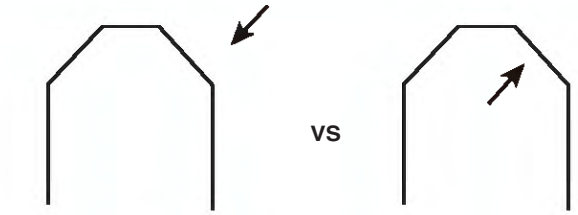


Figure 17-4 During function, the forces are directed against the mandibular arch form, which is designed to reduce stresses *(left)*. In the maxilla, forces are directed outside the arch form *(right)*. An arch is much less effective against outward forces than inward forces.

6. Reduced trabecular bone density of the maxilla results in compromised bone strength and a weaker implant-bone interface.
7. Absence of thick cortical plate at the crest of the premaxilla results in loss of high-strength implant support and less resistance to angled loads.
8. The accelerated bone volume loss in the incisor region often results in the inability to place central and lateral incisor implants without substantial augmentation procedures.
9. Crown height space (CHS) is often greater than ideal and is a force magnifier to any angled or cantilevered force.

TREATMENT OPTIONS

The treatment options for the restoration of a complete or partially edentulous patient with all six maxillary anterior teeth missing include a removable partial or complete denture, an implant-supported overdenture, or an implant-supported fixed prosthesis.

An independent, fixed implant-supported restoration has become the treatment of choice for most patients with complete or partial edentulism. A fixed prosthesis presents several advantages over an overdenture for a maxillary edentulous patient. Because many traditional maxillary dentures have adequate retention and stability, function and sore spots are rarely a problem. Therefore little benefit is perceived with an implant overdenture (IOD). The major disadvantage of the maxillary denture is most often the psychological aspect of removable teeth. In contrast, a fixed prosthesis presents significant benefits for the maxillary denture patient. In fact, after 3 years of function, most patients feel the maxillary fixed prosthesis is as good as or better than their natural teeth. On the other hand, an IOD is always considered by the patient as a removable prosthesis. Contraindications to a fixed partial denture include long edentulous spans, poor abutment support, and inadequate edentulous bone for proper prosthetic contour.

WWW.HIGHDENT.IR

The primary reason for a conventional maxillary removable denture is economic reasons or because a patient is unwilling to undergo implant surgery. However, the easiest interim treatment prosthesis for the replacement of several anterior teeth during implant submerged healing is a removable restoration. If bone augmentation is necessary, this prosthesis may need to be used for longer than 1 year before delivery of the final implant restoration.

Sequence of Treatment

Maxillary Labial Lip Position

Whether a denture, an overdenture, or a fixed prosthesis is being fabricated, a full-arch maxillary reconstruction begins with the determination of the facial position of the maxillary incisal edge. Its modification at a later step may alter all other measurements. A baseplate and wax rim may determine the labial contour of the maxillary lip. Most often the facial surfaces of the central incisors are 12.5 mm from the most posterior aspect of the incisive papilla.[10,11] The wax rim is initially positioned with this in mind. The farther forward the labial flange and teeth position, the higher the resting position of the lip and the greater the incisal edge exposure. The philtrum of the lip should have a visible depression in the midline under the nose. If the philtrum is too flat, the lip is extended too far, and wax should be removed from the labial aspect of the wax rim.

The position of the maxillary lip also may be determined by the position of the lower lip and chin. A horizontal line, represented by the Frankfort plane, may be drawn from the highest point of the auditory meatus (top of the tragus) to the lowest point on the margin of the orbit, with the patient's head in a vertical position. Ideally, a vertical line drawn from the Frankfort plane to the lower lip should have the maxillary lip anterior to this landmark 1 to 2 mm and the chin 2 mm posterior to this line.

The labial position of the lip in relationship to the premaxilla is the primary criterion to determine whether a fixed restoration, a bone graft and fixed restoration, or a maxillary overdenture is indicated. When the labial position of the wax rim is forward of the residual ridge more than 5 mm, a bone graft prior to implants is required for a fixed restoration, or a maxillary overdenture is considered (Figure 17-5). The maxillary anterior region with multiple teeth missing often is restored with an overdenture or FP-3 prosthesis.

Key Implant Positions

Once the prosthesis type and tooth position are determined, the patient force factors and bone density in the implant sites are evaluated. The key implant positions are then determined for the maxillary restoration.[12-14] An important parameter in treatment planning is to provide adequate biomechanical position and surface area of support for the load transmitted to the prosthesis. Four guidelines were presented in Chapter 8 for key implant positions in an implant prosthesis include:

1. No cantilever.
2. No three adjacent pontics.
3. The canines and first molar implant sites.
4. An arch is a five-sided open pentagon and should have at least one implant in each section of missing teeth.

Of these guidelines, number 3 and number 4 are often violated in the treatment plan of an edentulous maxilla, and therefore deserve further discussion.

Guideline 3: The Canine and First Molar Site

A fixed prosthesis replacing a canine tooth is at greater risk than most any other tooth in the mouth. The maxillary lateral incisor is the weakest anterior tooth, and the first premolar is often the weakest posterior tooth. A traditional prosthodontic axiom indicates a fixed prosthesis is contraindicated when a canine and two or more adjacent teeth are missing.[15] Therefore if a patient desires a fixed restoration, implants are required whenever the following adjacent teeth are missing: (1) the first premolar, canine, and lateral incisor (Figure 17-6); (2) the canine, lateral incisor, and central incisor (Figure 17-7, *A*); and (3) the canine, first premolar, and second premolar (Figure 17-7, *B*).

When any of these three missing teeth combinations are present, a fixed restoration is contraindicated because of the length of the span (three pontics), the amount of force (forces greater in the canine region compared with the anterior), and the direction of the force (angled forces to the canine region).

A tooth-supported prosthesis is less at risk than an implant-supported restoration when the canine and

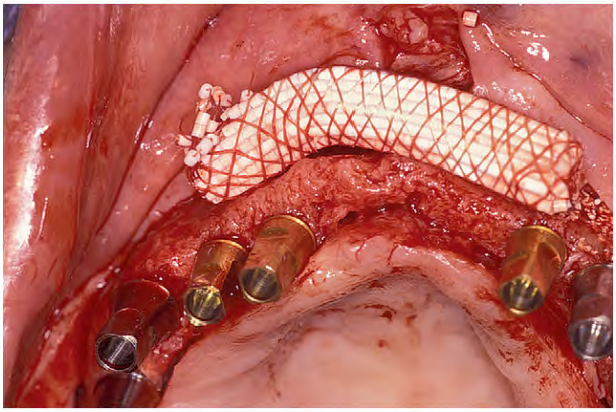
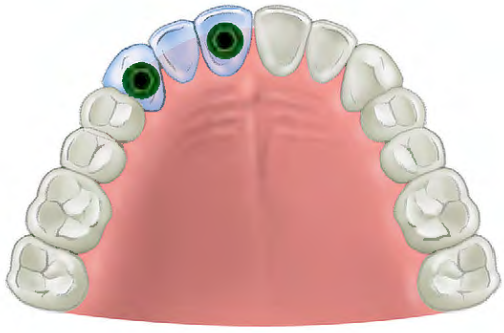


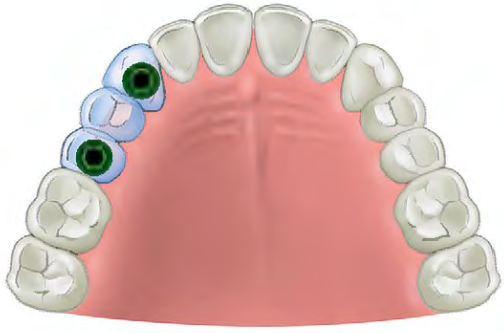
Figure 17-5 When the labial position of the teeth needed to support the upper lip is more than 5 mm anterior to the residual ridge, a bone graft or hydroxyapatite graft is required for a fixed prosthesis. Another method to support the maxillary lip is an implant overdenture with a labial flange.

WWW.HIGHDENT.IR

Figure 17-6 When the patient is missing a first premolar, canine, and lateral incisor, two to three implants are indicated.



A

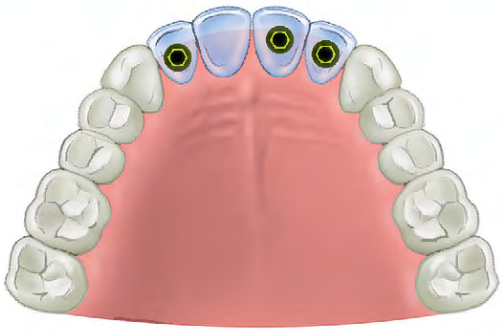


B

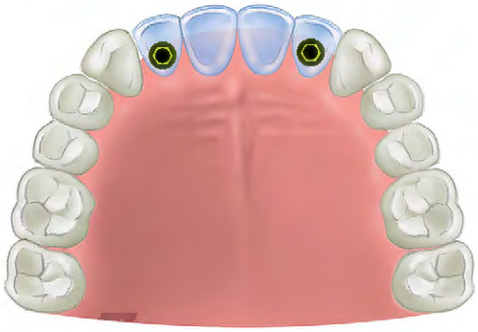
Figure 17-7 **A,** A patient missing a canine, lateral, and central incisor requires at least two implants to restore these teeth. **B,** A patient missing a second premolar, first premolar, and canine requires at least two implants to restore these three missing teeth.

two adjacent teeth are missing. Because teeth are more mobile than implants, a stress relief mechanism reduces the flexure, force, and effect of an angled force. Despite this, it is contraindicated to design three pontics in a fixed prosthesis whenever the natural canine and two adjacent teeth are missing. Therefore, under these conditions with implant treatment plans, at least two implants are indicated to support an independent fixed restoration (usually in the terminal positions of the span to eliminate cantilever forces) (Figure 17-8).

Using the missing canine and two adjacent natural teeth guideline, a fixed prosthesis is also contraindicated when missing a right canine, right lateral incisor, right central incisor, left central incisor, left lateral incisor,



A



B

Figure 17-8 **A,** When all four incisors are missing and the dental arch form is ovoid, three implants are indicated to support the prosthesis. To ensure adequate implant spacing, narrow-diameter implants may be placed in the central and adjacent lateral position. **B,** When all four incisors are missing and the dental arch is square, two implants in the lateral position may be sufficient to support a final prosthesis. In such cases, the implant diameter should be a standard dimension.

Figure 17-9 It is contraindicated to replace three adjacent teeth, which include a canine. This implant prosthesis has at least five pontics that include a canine.

and left canine without considerable anterior implant support (Figure 17-9). Yet in some improper treatment plans, implants are placed in each posterior maxillary quadrant and a fixed restoration with six pontics is fabricated to replace the anterior teeth (Figure 17-10).

WWW.HIGHDENT.IR

Figure 17-10 A panoramic radiograph of a fixed prosthesis with five adjacent pontics, including a canine. This prosthesis is undersupported, especially as it opposes natural dentition in a younger patient.

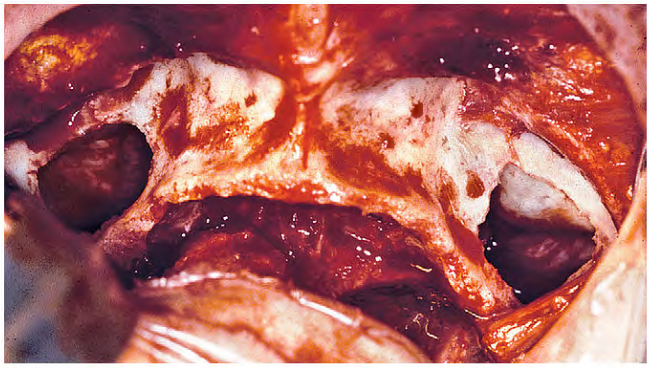


Figure 17-11 In the posterior maxillary, a Tatum lateral wall approach is used to graft the floor of the maxillary sinus with synthetic bone materials. For predictable results, the premaxilla and premolar regions most often require autologous bone. When six to eight sites need augmentation, an extraoral bone graft may be indicated.

Apparently the rationales for violating the prosthetic guidelines established in the literature for teeth are the following:

1. To augment a premaxilla, autologous bone grafts are usually necessary, whereas synthetic materials can be used to predictably graft in the posterior maxilla. The large volumes of bone required for the premaxilla often requires an iliac crest graft (which patients do not want, and for which few doctors are properly trained) (Figure 17-11).
2. The impression is that implants are more rigid and therefore stronger than natural roots. However, this is false security. The fact that implants are more rigid than teeth makes the three adjacent pontics and canine position guidelines more important to follow when the adjacent abutments are implants. The rigid abutments magnify the problem of flexibility of the metal and the direction of force applied to the prosthesis. Therefore, the canine is an especially important implant site when the anterior six teeth are missing. When available bone is not present, an autologous graft in the canine position is indicated before implant insertion, or an implant in both the lateral and first premolar site to compensate for the missing canine may be considered.

The first molar is an important abutment position in an edentulous maxilla. The first molar natural tooth surface area is two times greater than the premolars. The bite force in this region increases to 200 lb, compared with half this amount in the premolar sites. In addition, the bone density in the molar region is often poorer than the premolar regions of the jaws. As a result, larger-diameter implants are also suggested.

The anatomical problem for implant treatment in the posterior maxilla is the rapid expansion of the maxillary sinus after tooth loss. As a result, the edentulous posterior maxilla rarely has enough bone height without sinus grafting. A trend to cantilever the posterior missing teeth from anterior implants has developed. Posterior cantilevers from anterior maxillary implants are less predictable than cantilevers from anterior mandibular implants for all the reasons addressed in the beginning of this chapter. Instead, sinus grafting and larger-diameter implants (or two implants instead of one) are indicated in the first molar region.

Posterior implants (premolar and molar) without implant support in the premaxilla are sometimes connected with a full-arch bar for a maxillary overdenture. When a bar extends from molar to molar around an arch, the overdenture prosthesis is completely implant supported (RP-4), because it does not move during function or parafunction. As such, the overdenture acts as a fixed restoration (Figure 17-12). The removable implant prosthesis under these conditions should have the same implant support as a complete arch fixed restoration (not less).

Guideline 4: Five-Sided Arch

The maxillary arch may be divided into five segments, similar to an open pentagon (Figure 17-13). The central

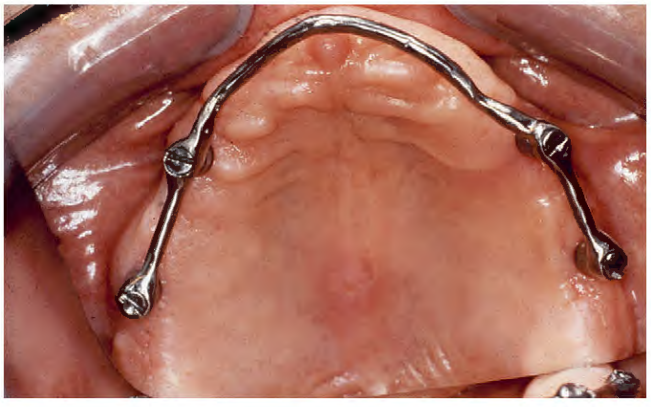


Figure 17-12 This overdenture bar extends from second premolar to second premolar. The anterior bar represents a span of eight pontics and is too long for proper prosthetic support. The overdenture, when in place, is rigid over this bar design, and as such, requires as much support as a fixed prosthesis.

WWW.HIGHDENT.IR

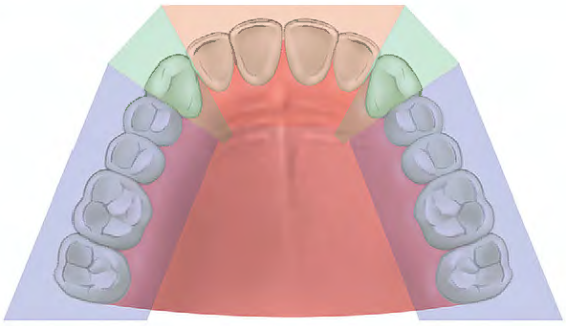


Figure 17-13 The maxillary arch may be treated as an open pentagon, with five straight-line segments. When teeth are missing in multiple segments, at least one implant is required in each section.

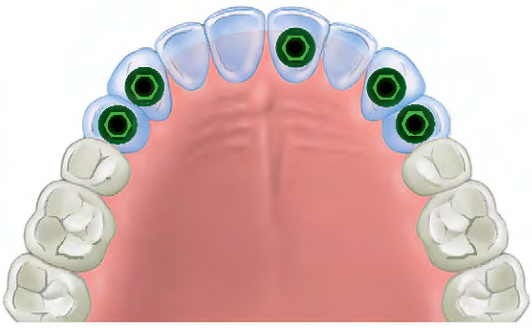


Figure 17-14 When eight anterior teeth (first premolar to first premolar) are missing, implants should be placed in each segment to provide adequate support.

and lateral incisors represent one segment, each canine a separate segment, and the posterior premolars and molars individual segments. In other words, each segment is essentially a straight line, with little resistance to lateral forces. However, when splinted together, an arch form dynamic becomes evident.

At least one implant should be placed in each of the five sections missing teeth and then splinted together when replacing multiple adjacent teeth missing in the maxilla. At least three implants usually are required to replace the anterior six teeth in the premaxilla: one in each canine position and one in any of the four incisor positions.[12-16] When posterior teeth are also missing, additional posterior implants are required (Figures 17-14 and 17-15).

Previous studies by Bidez and Misch have shown that the force distributed over three abutments results in less localized stress to the crestal bone than two abutments.[17] Because these three elements are aligned along an arch, connecting at least three segments creates a tripod effect and provides an anteroposterior distance (A-P spread) with mechanical properties superior to a straight line and with greater resistance to lateral forces. The A-P distance for the anterior cantilevers in the premaxilla restoration corresponds to the distance between the center of the most distal implants on each side (in the splint) and the anterior aspect of the most anterior implant.

A poor treatment option for fully edentulous maxillae is the placement of implants in each posterior quadrant

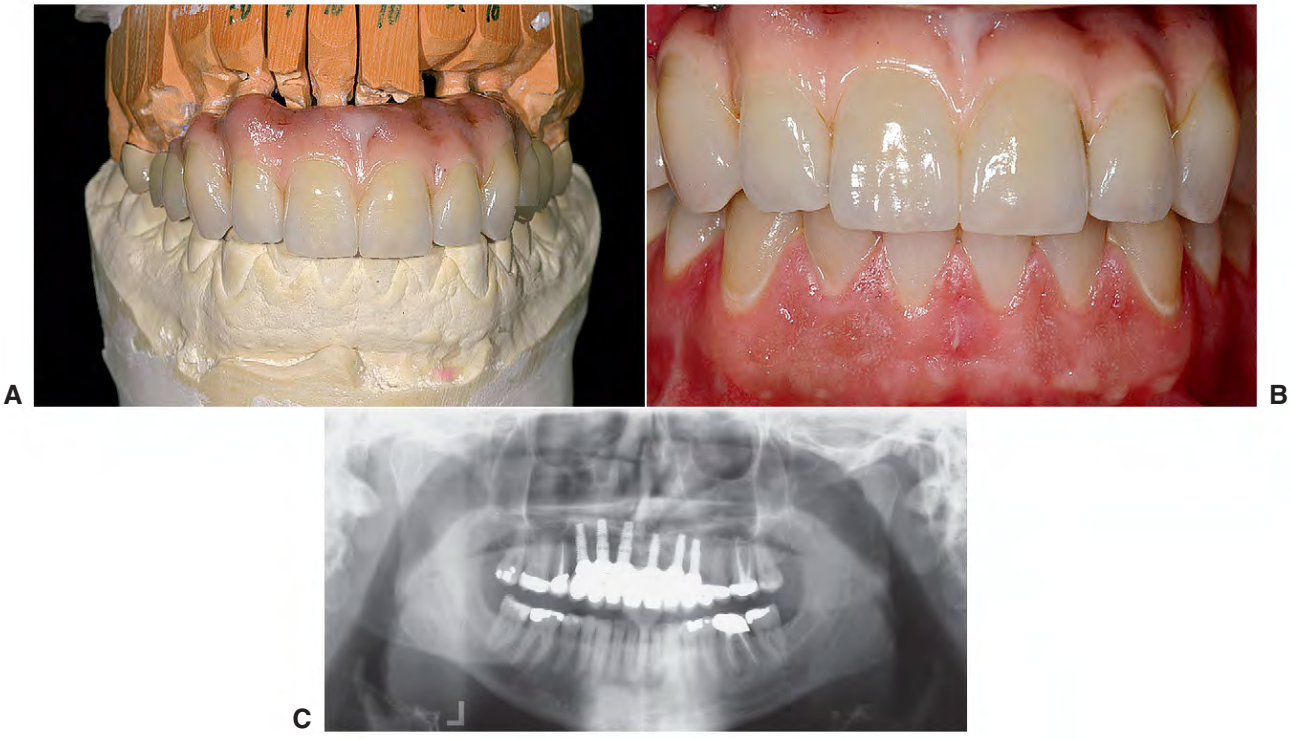


Figure 17-15 **A,** The dentate arch form may be different than the residual bone form, as the ridge resorbs apically and away from the original tooth position. In such cases, the prosthesis is designed to restore the proper tooth contour and lip support. **B,** The fixed FP-3 prosthesis replaces eight adjacent anterior teeth and is supported by six implants. **C,** Panoramic radiograph of the same patient.

WWW.HIGHDENT.IR

Table 17-1 Treatment Plan for Edentulous Premaxilla

ARCH FORM	ANTERIOR CANTILEVER (MM)	NUMBER OF IMPLANTS	IMPLANT POSITION
Square	< 8	2	Canines
Ovoid	8-12	3	Two canines and one incisor
Tapering	> 12	4	Two canines and two incisors

with independent bar segments and an overdenture. This treatment option is prone to failure. The maxillary overdenture rocks back and forth during excursions (if not, the overdenture is really a fixed restoration). The posterior implants are in a straight line and cannot resist the lateral forces. Eventually, almost all the implants on one side are lost. Maxillary complete prostheses and overdentures have a greater incidence of implant failure and complications than mandibular counterparts.[1,18,19] These observations further emphasize the need for more implants and fewer pontics in the restoration of a maxilla compared with the mandible.

Premaxilla Arch Form

The arch form of the maxilla influences the fixed prosthesis treatment plan of the edentulous premaxilla. Three typical dental arch forms for the maxilla are square, ovoid, and tapering. As a consequence of bone resorption, the edentulous ridge arch form may be different from the dentate arch form. The dental arch form of the patient is determined by the final teeth position in the premaxilla and not the arch shape of the residual ridge. A residual ridge may appear square because of resorption or trauma. However, the final teeth position may need to be cantilevered facially with the final prosthesis. In other words, a dental ovoid arch form may be needed to restore a residual edentulous square arch form. The number and position of implants are related to the arch form of the final dentition (restoration), not the existing edentulous arch form (Table 17-1).

The dental arch form in the anterior maxilla is determined by the distance from two horizontal lines. The first line is drawn from one canine incisal edge tip to the other. This line most often bisects the incisive papilla. The second line is drawn parallel to the first line, along the facial position of the anterior teeth (Figure 17-16). When the distance between these two lines is less than 8 mm, a square dental arch form is present. When the distance between these two lines is 8 to 12 mm, an ovoid dentate arch form is present—the most commonly observed. When the distance between the two lines is greater than 12 mm, the dentate arch form is tapering.

In a dental square arch form, lateral and central incisors are not cantilevered very much facially from the canine position. Therefore mandibular excursions and occlusal forces exert less stress on the canine

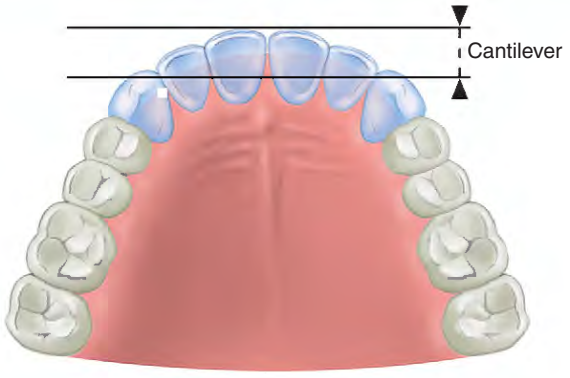


Figure 17-16 Two horizontal lines are drawn. The first line bisects the incisal papilla and connects the tips of the canines. The second line is parallel and along the facial position of the central incisor. The distance between these lines determines whether the dentate arch form is square, ovoid, or tapering.

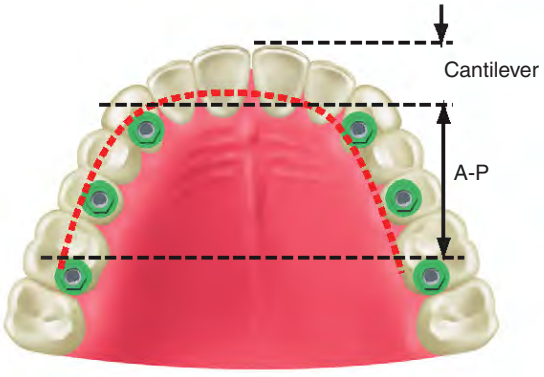


Figure 17-17 When force factors are low, a square dentate arch form may use six implants for a fixed or RP-4 prosthesis. *A-P,* Anteroposterior distance.

implants. As a result, implants in the canine position to replace the six anterior teeth may suffice when the force factors are low and if they are splinted to additional posterior implants (Figure 17-17). The four pontics between the canines counters Rule 2 of key implant positions (no three adjacent pontics) because (1) the forces are lowest in the incisor region and (2) in a square arch form in the maxilla, minimal cantilevers are placed on the canines (Figures 17-18 to 17-20).

If the final teeth position is an ovoid arch form, at least three implants should be inserted into the premaxilla: one in each canine and preferably one in a central incisor position (Figure 17-21). The central

WWW.HIGHDENT.IR

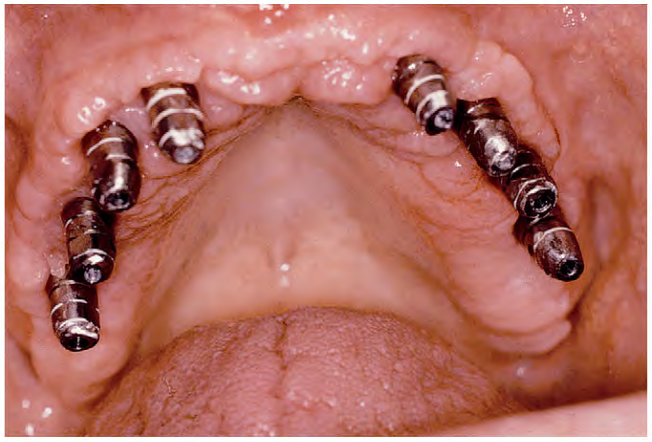


Figure 17-18 Eight implants were used in this square arch form to fabricate a fixed prosthesis.

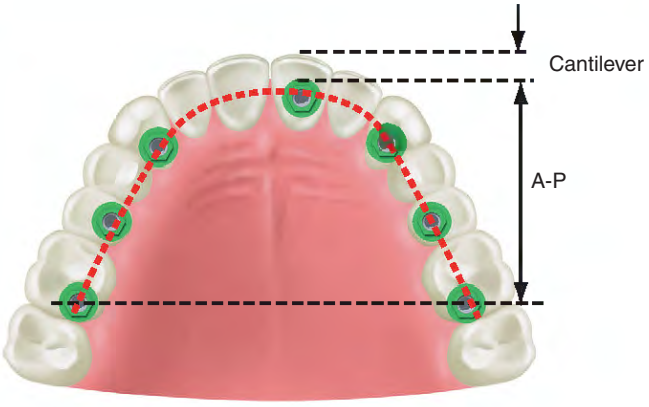


Figure 17-21 In an ovoid dentate arch form, three implants should be planned in the premaxilla: one in each canine position and one additional implant. In addition, at least four posterior implants should be splinted to form an arch. *A-P,* Anteroposterior distance.

Figure 17-19 The fixed prosthesis of the patient in Figure 17-18 follows a square dentate arch form. As a result, the anterior teeth are not cantilevered from the canine implants.

Figure 17-22 An ovoid arch form in a patient with three implants in the premaxilla and a total of nine implants for an FP-3 prosthesis.

Figure 17-20 A panoramic radiograph of the patient from Figures 17-18 and 17-19 with a square arch form. Additional implants were indicated, as the patient is a male and the opposing fixed prosthesis is implant supported.

incisor position increases the anterior posterior distance from the canine to central and provides improved biomechanical support to the prosthesis. In long-term edentulous maxillae, this most likely will require bone augmentation before implant insertion. When patient force factors are low to moderate, the anterior implant may be positioned in a lateral incisor site. These three implant positions resist the additional forces created in this arch form, enhance prosthesis retention, and reduce the risk of abutment screw loosening (Figures 17-22 to 17-25).

The restoration of a tapered dental arch form places the greatest forces on anterior implants, especially during mandibular excursions when the residual bone is an ovoid or square ridge form. The anterior teeth create a significant facial cantilever from the canine position. As such, four implants should be considered to replace the six anterior teeth (Figure 17-26).

The bilateral canine and central incisor positions represent the best option. These positions are preferred when other force factors are greater, such as crown height, parafunction, and masticatory muscular dynamics. When more than six anterior teeth are missing, additional posterior implants also should be splinted to the anterior segment (Rule 4). The worst-case scenario is a patient requiring restoration of a dental tapered arch form with a square residual ridge form. Not only are four implants then ideally required to compensate for the cantilevered tooth position, but these implants should be connected to additional posterior implants,

WWW.HIGHDENT.IR

Figure 17-23 A fixed FP-3 prosthesis follows an ovoid anterior arch form supported by implants in the premaxilla region.

Figure 17-24 The patient in Figures 17-22 and 17-23 was restored with maxillary and mandibular full arch implant-supported prostheses.

Figure 17-25 The panoramic radiograph of the patient in Figure 17-24 with an ovoid dentate arch form. An iliac crest bone graft to both the maxilla and mandible was restored with nine maxillary and seven mandibular implants to support the final FP-3 prostheses.

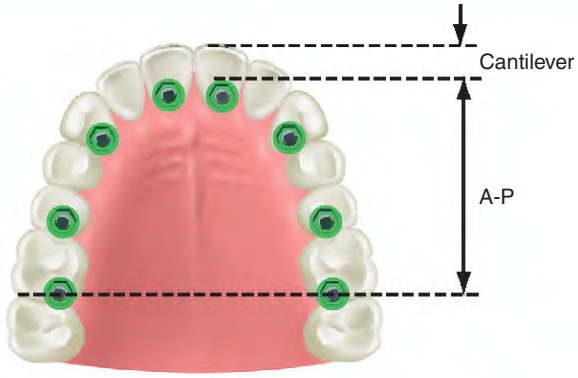


Figure 17-26 In a tapered arch form, the anterior cantilever is greater and should be supported by implants in the premaxilla. At least four posterior implants should be added to restore the completely edentulous arch. *A-P,* Anteroposterior distance.

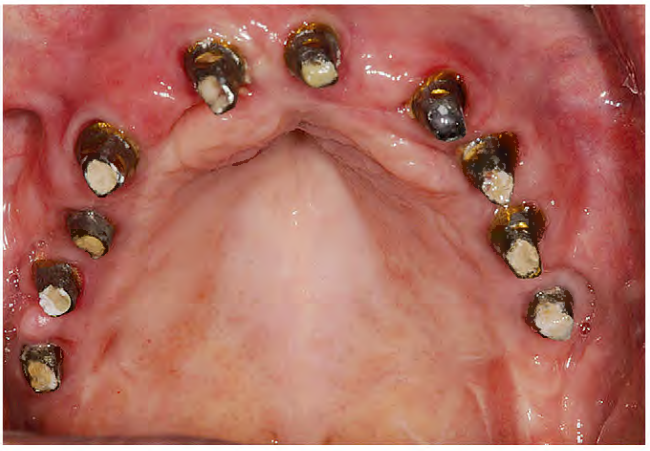


Figure 17-27 A patient with a tapered arch form (after an iliac crest graft) was restored with four implants in the premaxilla (10 implants total).

Figure 17-28 The fixed FP-3 prosthesis of the patient in Figure 17-27 is supported by implants in the canine and central incisors position. Additional posterior implants are used and splinted together.

preferably to include implants as far distal as the second molar sites (Figure 17-27 to 17-30).

When one canine region cannot be used to place an implant in the edentulous premaxilla, at least one implant on each side of the missing canine is required to compensate for this vital position (a first premolar and lateral incisor implant). A central incisor implant and canine position in the contralateral section can be splinted to these other two implants to act as abutments for the fixed or overdenture restoration.

When force factors are greater than usual, four implants in the premaxilla are suggested. The four implants in

WWW.HIGHDENT.IR

Figure 17-29 The maxillary FP-3 fixed prosthesis in place.

Figure 17-30 A panoramic radiograph of the patient with a tapered arch form in Figures 17-27 to 17-29. Iliac crest grafts to the maxilla 10 implants (four in the premaxilla) and a fixed prosthesis is opposing six implants in the mandible. The patient is a male of moderate age and force factors.

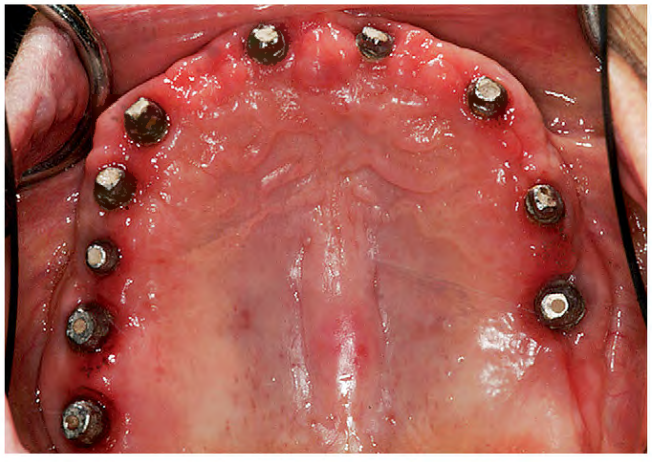


Figure 17-31 Moderate force factors usually require four implants in the premaxilla, splinted to additional posterior implants.

the premaxilla should be splinted together and share any lateral forces during excursions. This means a minimum of four implants are needed to replace the anterior six teeth with high force factors. In the presence of these forces (e.g., moderate to severe bruxism), larger-diameter implants also should be used, especially in the canine positions (which have increased load angulation

Figure 17-32 A fixed FP-3 porcelain-fused-to-metal restoration supported by 10 implants on a patient with moderate force factors and softer bone type.

Figure 17-33 Panoramic radiograph of the patient in Figures 17-31 and 17-32, illustrating the 10 maxillary implants opposing an implant prosthesis in the mandible.

in excursions and higher bite forces). In other words, in most instances the completely edentulous anterior maxilla is restored with three or four implants splinted together to replace the anterior six teeth.

Posterior cantilevers should not be placed on maxillary anterior implants. (Rule 2 in key implant positions). If posterior teeth also are being replaced in the prosthesis, additional implants are required. Seven to 10 implants often are inserted to restore a completely edentulous maxilla with a fixed prosthesis, especially when opposing natural dentition or a fixed restoration (Figures 17-31 to 17-34).

It should be noted that most full-arch maxillary prostheses are FP-3 fixed restorations or RP-4 overdentures. In either scenario a mesiodistal implant position does not have to strictly correlate with tooth position. In other words, the faciopalatal position is often more important than the mesiodistal tooth site, as the gingival aspect of the restoration separates the clinical crown from the implant site. As such, the implant site is determined more for biomechanics, interimplant spacing, or available bone, rather than a strict tooth site position (as in an FP-1 prosthesis).

WWW.HIGHDENT.IR

Figure 17-34 The high smile lip dynamics of the patient in Figures 17-31 to 17-33 with moderate force factors.

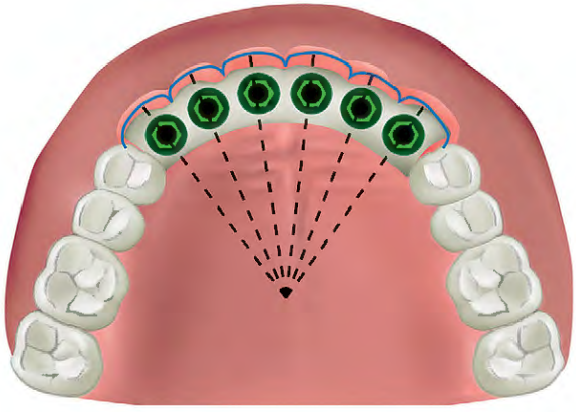


Figure 17-35 When implants are placed in a premaxilla with abundant bone, the arch dimension allows implant placement in the original teeth location.

PREMAXILLA PARTIALLY EDENTULOUS TREATMENT PLANS

Several factors affect the strategic selection of implant size and position to restore a completely edentulous maxillary arch. In general, two implant bodies should be more than 3 mm apart. Tarnow et al.[20] have observed that the horizontal dimension of a crestal defect next to an implant measures almost 1.5 mm. As such, if two implants are closer than 3.0 mm, a vertical angular defect on each implant may result in horizontal bone loss between the two implants. This bone loss in turn may favor the proliferation of anaerobic bacteria in the sulcular environment, or the tissues may shrink and compromise the interdental/implant soft tissue contours in a highly esthetic area. Degidi and Misch have found the vertical defect width may be 0.5 mm rather than 1.3 mm, dependent upon implant design.[21] However, the 3-mm guideline is an ideal safety factor for an implant distance.

The edentulous square-to-ovoid arch form dimension often does not accommodate interimplant spacing for more than four anterior implants. The largest interimplant distance is typically found in a tapered arch form. As the radius of the circle becomes smaller (from labial resorption or patient size), the premaxilla interimplant distance is reduced (Figures 17-35 and 17-36). As a result, usually no more than four implants are used to replace the anterior six teeth, even when bone grafting restores a more compatible residual ridge form.

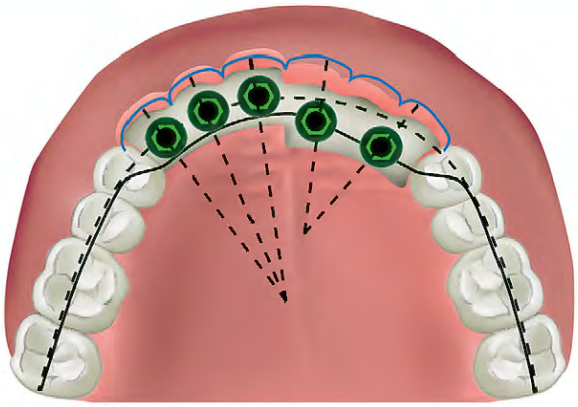


Figure 17-36 When the arch form is reduced, most often only two implants can be positioned to replace the three teeth *(on the right)*.

When the natural canines are present and the missing teeth are the four maxillary incisors, the implant number is not as dependent upon the dental arch form. As a general rule, the remaining arc of bone is smaller than the dentate arch. As a result, one implant per tooth results in the implants being too close to each other (less than 3 mm). Therefore the most common scenario is to place three Division B root forms (3.5 mm wide) in the lateral incisors and one central incisor region.

When four anterior teeth are missing in an FP-1 prosthesis with a high smile lip position, the interdental papilla and cervical emergence of a pontic may be more esthetic than the soft tissue drape between two implants in the central incisor regions. Therefore the treatment plan may include only two implants in the lateral incisor sites to replace the four anterior teeth. However, if the patient has a tapered arch form or parafunction, this treatment plan is insufficient. The risk of screw loosening, crestal bone loss and resultant soft tissue changes is greater and rarely warrants the risk of reducing the implant number. The two-implant treatment option is often designed for a square dental arch form, in an older female, with little to no parafunction to fabricate an FP-1 prosthesis and a high lip line when the natural canines are present.

When the patient is missing a lateral and both central incisors, the three missing teeth may be safely restored with two implants. One implant (Division B) is positioned in the lateral incisor region, and the other implant (Division A) is placed in the opposite central incisor area (Figure 17-37, *A*). This eliminates a cantilever and decreases the risk of screw loosening. The

WWW.HIGHDENT.IR

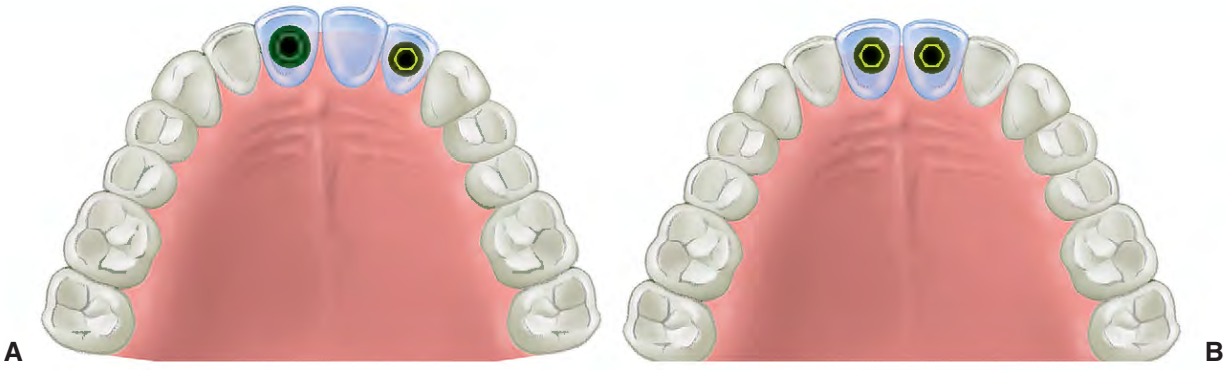


Figure 17-37 **A,** When two central and a lateral incisors are missing, two implants are indicated to restore the missing span. A smaller-diameter implant may be used in the lateral incisor position. **B,** Missing central incisors should be restored with two implants. Most often, narrow-diameter implants are indicated.

interdental papilla of the adjacent natural canine and lateral incisor determine the papilla height next to the adjacent implant crowns.

In the presence of horizontal marginal bone loss on the anterior teeth adjacent to an implant site, orthodontic extrusion may be indicated to correct the interproximal bone level. When this option is used, a veneer is usually required to restore the adjacent tooth after orthodontic extrusion. The restoring dentist may place a veneer and lower the adjacent contact and modify the crown shapes on the implant and natural teeth to a more square form, which decreases the height requirements of the papilla and eliminates the absence of tissue in the interproximal area found in the original condition. These treatment plan decisions should be made before implant insertion.

When the patient is missing the two central incisors, there should be two implants to restore the site. One implant with a cantilevered pontic increases the risk of screw loosening, crestal bone loss, and component fracture. Whenever two implants are used to replace two adjacent central incisors, the implant diameter should most often be Division B (3.5 mm) (Figure 17-37, *B*). This provides greater soft tissue volume between the implants, allows the surgeon to place the implant more distal to avoid the incisive foramen, and leaves a thicker facial cortical plate.

When the patient is missing a lateral and central incisor, two Division B implants may also be used to restore this condition, rather than using a Division A implant in the central incisor position. The additional interproximal space permits an improved soft tissue drape condition.

In general, the premaxilla requires the most varied surgical approaches to improve success and is the most critical region for esthetics and phonetics. Options for Division B and C–w bone more often require augmentation rather than osteoplasty, as advocated in other intraoral regions. The opposing landmark is the floor of the nose, and this structure may be modified slightly by nasal elevation of 1 to 2 mm to improve implant support in C–h ridges for FP-3 or RP-4 prostheses.

COMPLETELY EDENTULOUS MAXILLAE TREATMENT PLANS

A review of the literature indicates that full maxillary fixed implant–supported prostheses are fabricated on an average of six standard-diameter implants with posterior molar cantilevers. An average of four to six implants also are used to support bar overdentures. Yet the edentulous maxilla has the lowest implant survival for either fixed or removable implant restorations, compared with mandibular prostheses.[22-27] All reports concur with the finding that maxillary bone tends to be of poorer quality and volume and presents several biomechanical disadvantages. The author suggests that to compensate for the poor local conditions, a greater number of implants should be planned, along with a greater A-P distance. Therefore there is usually a need for sinus grafts and premaxilla reconstruction to restore the edentulous maxillary arch.

With these concerns in mind, the minimum implant number for a completely edentulous maxillary fixed or RP-4 prosthesis is usually seven in the ovoid arch form. The suggested locations for this arch are: at least one central (or lateral) incisor position, bilateral canine positions, bilateral second premolar sites, and bilateral distal half of the maxillary first molar sites. These are the same positions as for a fixed restoration (see Figure 17-21). These seven implants should be splinted together to function as an arch. These implant positions create sufficient space between each implant to allow for greater implant diameters (when required for force or bone density factors), without concern for the adjacent site. A square dental arch form may use a minimum of six implants: bilateral canines, bilateral second premolars and bilateral first molar sites. The first molar implant sites in a completely edentulous maxilla

WWW.HIGHDENT.IR

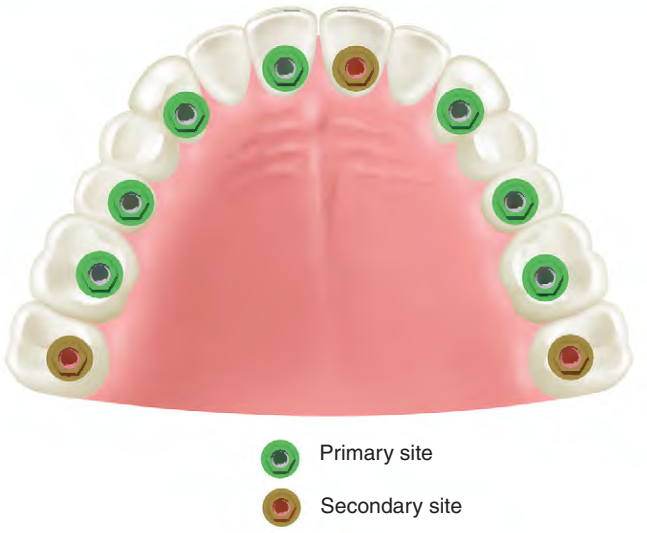


Figure 17-38 The ideal seven-implant positioning for a maxillary edentulous arch includes at least one central incisor position, bilateral canine positions, bilateral second premolar sites, and bilateral sites in the distal half of the first molars. In case of heavy stress factors, an additional anterior implant and bilateral second molar positions (to increase the anteroposterior distance) may be of benefit.

almost always require sinus grafting, because most edentulous maxillary posterior regions are inadequate in height.

When force factors are moderate or the dental arch form is tapered, the minimum implant number should increase to eight implants. When eight implants are selected, the additional implant is usually placed in the premaxilla, in the contralateral central (or lateral) incisor position. When force factors are greater than usual or bone density is poorer, additional implants should be used in any of the arch forms. In the square and ovoid arch form, at least one additional implant is positioned in the premaxilla. In addition, for patients with higher force factors or poor bone density, two additional implants are planned in the distal half of the second molar position to improve the arch form, increase the A-P distance compared with the first molar site, and add an additional implant where the bone density is poor and force factors are increased (Figure 17-38). This also is an excellent biomechanical design to minimize stress when a dentate tapered arch form is restored in an ovoid or square residual ridge form. These implant number and position guidelines also may counter the effect of an incisal cantilever off the residual anterior bone for an esthetic tooth position and is indicated for patients with chronic parafunction (such as bruxism).

The disadvantage of second molar implants for an ideal treatment plan is the additional cost of the second molar implant and restorations. Many patients do not display the second molar and do not require this tooth for function. As such, to decrease the cost, the restoring dentist may use a coping splinted to the arch, rather than a porcelain-fused-to-metal crown. The reason for this implant position is for force transfer, not necessarily esthetics or function.

Box 17-2 Multiple Implant Size Selection Criteria for FP-1 Prostheses in the Maxilla

- Natural tooth 2 mm below cement-enamel junction
- 1.5 mm from adjacent tooth
- 1.5-mm facial bone
- 3 mm from adjacent implant
- 3- to 4-mm-diameter for anterior maxilla
- 4-mm-diameter minimum for posterior maxilla

In conclusion, the number of implants used in an edentulous maxilla may range from 6 to 10. The number of implants needed in an edentulous premaxilla is related to arch shape. When force factors are moderate to severe or bone density is poor, more implants should be inserted and in greater diameter to enhance the surface area. In addition, the A-P distance should be increased.

Multiple Anterior Implant Sizes: Anterior Tooth Replacement

Several conditions should be considered for the proper implant diameter, including tooth size, distance from an adjacent tooth, interimplant distance, facial bone dimension, and loading forces.

A primary factor for implant size is the necessary distance from an adjacent tooth root or implant. The horizontal dimension of a wedge-shaped bone defect around an implant at the crest of the ridge from the biological width, implant design, or occlusal overload ranges from 0.5 to 1.4 mm.[20,21] Initial vertical bone loss around an implant during the first year of loading varies and ranges from 0.5 to more than 3 mm. When the implant is closer than 1.5 mm to an adjacent natural root, the wedge-shaped vertical defect may evolve into a horizontal defect, creating bone loss on the adjacent tooth root. This is of utmost importance because the interseptal bone height in part determines the incidence of presence or absence of the interdental papillae between the teeth or implants, as well as the incidence of probing depth greater than 5 mm.[28] As a consequence, whenever possible, an implant should be at least 1.5 mm from the adjacent teeth (Box 17-2).

When implants are placed adjacent to each other, a minimum distance of 3 mm is suggested to accommodate eventual crestal bone loss and maintain interseptal bone levels.[20] When placing two adjacent implants, their diameter should often be reduced, compared with the ideal dimensions of a single-tooth implant. The ideal diameter of an implant for an FP-1 prosthesis also should consider the faciopalatal dimension of bone.

WWW.HIGHDENT.IR

Table 17-2 Maxillary Teeth Dimensions (Average)

TYPE OF TEETH	CERVICO-INCISAL HEIGHT (mm)	MESIODISTAL CROWN (mm)	MESIODISTAL CERVIX (mm)	MESIODISTAL CEJ (–2 mm)	FACIOLINGUAL CROWN (mm)	FACIOLINGUAL CERVIX (mm)
Central incisor	10	8.6	6.4	5.5	7.1	6.4
Lateral incisor	9	6.6	4.7	4.3	6.2	5.8
Canine	10	7.6	5.6	4.6	8.1	7.6
First premolar	8	7.1	4.8	4.2	9.2	8.2
Second premolar	8	6.6	4.7	4.1	9.0	8.1
First molar	8	10.4	7.9	7.0	11.5	10.7
Second molar	7	9.8	7.6	7.0	11.4	10.7

CEJ, Cement-enamel junction.

The 1.4-mm-wide defect, which may form next to the implant after loading, forms 360 degrees around the implant crest module. As such, if less than 1.5 mm of bone is present on the facial aspect of an implant, the vertical defect becomes a horizontal defect and the tissue recedes when it is thin or forms a soft tissue pocket when it is thick. The first condition decreases esthetics, as the implant crest module may even become visible, whereas the second condition increases the risk of peri-implantitis and further bone loss. Therefore the ideal implant position and implant diameter should have 1.5 mm or more of bone on the facial of the implant.

The mean papilla height between two adjacent teeth is 3.4 mm, ranging from 1 to 7 mm, and the usual range of interimplant papillary height is 2 to 4 mm. Therefore, when an FP-1 restoration is desired, the prosthesis design (square tooth forms) and implant positions may need to be altered accordingly to optimize an esthetic result.[28]

The amount of force transmitted to the implant body and abutment screw is also a consideration for implant diameter. The larger the diameter, the less stress to the crestal bone and components. Therefore, in a patient with moderate to severe bruxism, a larger-diameter implant should be used, especially in the canine position, to assist in posterior disclusion of the teeth and canine guidance.

The implant dimension in question is the size of the crest module, not the implant body dimension. For example, a 4.1-mm crest module (on a 3.75-mm implant body) needs 7.1 mm of mesiodistal crestal bone, a 3.5-mm crest module (on a 3.25-mm implant body) is indicated for 6.5 mm of bone, and a 5.2-mm crest module requires 8.2 mm of bone.

The difference in the emergence profile between a 4-mm-diameter implant and a 5-mm-diameter implant is negligible and often not clinically relevant. However, the larger implant diameter has less surrounding soft tissue and is more difficult to control the creation of a papilla. Therefore, when in doubt, a smaller-diameter implant should be selected in the esthetic zone. Thus a 3.5- to 4-mm-diameter implant often is used in the central implant position for an FP-1 prosthesis. Likewise, a 3-mm-diameter implant often is used for a lateral incisor FP-1 restoration. The exceptions to this rule may be in a bruxing patient when the benefits of a larger-diameter implant with decreased occurrence of abutment screw loosening, crestal bone loss, and long-term body failure are more desirable. However, splinting multiple implants is more effective than implant diameter to decrease stress complications.

When the implants are out of the esthetic zone (FP-2, FP-3, RP-4, RP-5 prostheses), the diameter of the implant is more related to the amount of force applied to the implant-bone-prosthetic system. Facial bone dimensions and interimplant spacing may be less important. The posterior regions should most often use 3.7- to 4.2-mm-diameter implants in premolars and 5-mm diameter implants in molars, because the force factors are greater and the bone density is poorer. The natural maxillary molars have the greatest diameter and largest surface area of any teeth. The maxillary molars have a 200% increase in surface area compared with the premolar teeth. The first molar is 10.4 mm in mesiodistal dimension, and the second molar is 9.8 mm. The cement-enamel junction (CEJ) dimensions of these teeth are 7.9 mm and 7.6 mm, respectively, and 2 mm below the CEJ, these teeth are both 7 mm in size (Table 17-2). However, the ideal implant diameter is 5 mm to 6 mm for the maxillary molars. Because titanium is 5 to 10 times more rigid than natural teeth, the modulus of elasticity for an implant of sizes greater than 6 mm

WWW.HIGHDENT.IR

Figure 17-39 A large crown height space may be restored with a hybrid prosthesis of a metal framework, porcelain denture teeth, and acrylic.

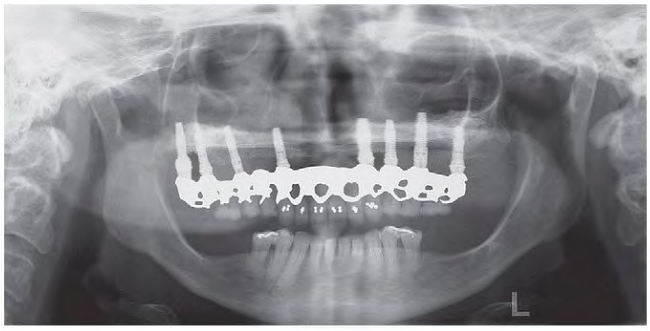


Figure 17-40 Panoramic radiograph of an FP-3 hybrid maxillary prosthesis, with a metal framework, porcelain denture teeth, and acrylic.

may be too great and may cause stress shielding and bone loss. The 6-mm diameter should not be used in the anterior regions, because the magnitude of the force is not large enough to strain the bone within the ideal physiologic window next to such a large implant. As a consequence, more crestal bone loss is often observed.

When the diameter or designs of molar implants do not provide sufficient surface area, the number of implants should be increased. Rather than one implant replacing a molar, two implants of 4 mm in diameter should be considered to compensate for very soft bone types or unfavorable force factors (e.g., parafunction).

The number of implants in the patient with multiple missing posterior teeth is often increased, especially when force factors are high, bone density is poor, and larger implant diameters are not used. When multiple adjacent posterior teeth are missing, the number of implants is more important than the implant size. When adjacent molars are missing, three regular-size implants are often considered when implant diameter cannot be increased to 5 or 6 mm.

The dentist may use the following guidelines for implant locations in a completely edentulous maxilla:

1. The bilateral midcanine position is a key implant position and is planned for 4-mm-diameter implants (Guideline 3).
2. The center of the first premolar is planned 7 mm distal from the center of the canine implant (for a 4.1-mm-diameter implant). This is an optional implant site when parafunction is moderate to severe.
3. The center of the second premolar is 7 mm distal from the first premolar site (14 mm from the midcanine position) for a 4.1-mm-diameter implant on each side. This is a key implant position (Guideline 2).
4. The center of the first molar is 8 to 10 mm distal from the mid second premolar implant (this places the implant in the distal of the first molar and increases the A-P distance. Ideally, the implant should be 5 to 6 mm in diameter. This is a key implant position (Guideline 3).
5. The center of the second molar is 8 to 10 mm distal from the center of the first molar. This position is most important for the edentulous arch with a tapered dentate arch form, D4 bone types, or severe force factors.

Figure 17-41 The hybrid fixed maxillary prosthesis in Figure 17-40 may be made esthetic, has reduced laboratory costs, is lightweight, and may be repaired more easily.

Fixed Prosthesis Design

The amount of CHS in an edentulous region greatly varies. The larger crown height space may facilitate the fabrication process of removable prostheses through easier denture tooth setup and greater bulk of acrylic to strengthen the prosthesis. However, the same space may be of concern for porcelain-metal fixed restorations. The replacement teeth appear elongated and often need the addition of gingival tone materials to replace the soft tissue in the esthetic regions. The greater impact force on implants compared with teeth, coupled with an increased crown height, results in increased moment forces on implants and increased risk of component and material fracture. These problems are noted especially with less favorable biomechanics on cantilevered sections of fixed restorations. An excessive crown height may cause greater prosthesis failure through mechanical increase in force (vertical cantilever) and fabrication complications (metal and porcelain shrinkage after casting or baking). In addition, the increased crown height dramatically increases the weight and the cost of the prosthesis.

WWW.HIGHDENT.IR

Table 17-3 Comparison of Maxillary Restorations

FACTOR	FP-3	RP-4
Psychological	+++	+
Material	Porcelain fused to metal	Bar, metal and overdenture
Lip support	+	+++
Esthetics	Pink porcelain (=)	Resin (=)
Phonetics (airway, leakage)	+	+++
Function (food entrapment)	+	+++
Maintenance	+++	More change of attachments (–)
Repair	–	+
Force factors	–	Remove at night (+)
Number of implants	=	=

+++, Best; +, better; =, similar; –, poor.

An alternative method to fabricate fixed prostheses for a CHS of 15 mm or more is the fixed complete denture hybrid prosthesis, with a smaller metal framework, denture teeth, and acrylic resin (Figures 17-39 to 17-41). This restoration design was made popular by Zarb et al.[29] and has been used on osteointegrated implants for more than two decades. This design is less expensive to fabricate, is highly esthetic (premade denture teeth), easily replaces teeth and soft tissue in appearance (pink acrylic) (FP-3), is lightweight, and is easier to repair. The denture teeth in these prostheses should not be acrylic or composite, owing to a high fracture rate. Instead, porcelain denture teeth are suggested. Because acrylic resin acts as an intermediary between the porcelain denture teeth and metal substructure, the impact force during dynamic occlusal loading may be reduced compared with a porcelain-metal restoration. As a general rule, fixed hybrid prostheses (FP-3) are used for situations with CHSs of 15 mm or greater. However, when the CHS is less than 15 mm, the decrease in bulk of acrylic increases the risk of fracture and complications. Therefore a porcelain-metal restoration is suggested.

Maxillary Overdenture Options

The primary advantage of a maxillary IOD compared with a fixed prosthesis is the ability to provide a flange for maxillary lip support and the reduced fee compared with a fixed restoration. As a consequence, before the selection of a specific prosthesis type and to facilitate the diagnosis, the labial flange above the maxillary teeth of the existing denture (or wax try-in of a new prosthesis) may be removed and the facial appearance of the maxillary lip without labial support assessed. If the maxillary lip requires additional support, two options are available:

1. A bone graft to the premaxilla is performed before or in conjunction with implant insertion or at uncovery for a fixed implant prosthesis.
2. A maxillary IOD is fabricated with a labial flange on the prosthesis.

Maxillary IOD complications, such as attachment wear and prosthesis or component fracture, are more frequent than with a fixed restoration and primarily occur as a result of inadequate bulk of acrylic and minimal strength of the small framework, compared with a fixed restoration (Table 17-3).

Fewer reports have been published for maxillary IOD compared with the mandible. Most of these reports discuss RP-5 restorations with posterior soft tissue support and anterior implant retention. According to Goodacre, the restoration with the highest implant failure rate is a maxillary overdenture (19% failure rate).[1]

In 1994, Palmqvist et al.[30] reported similar poor results in a 5-year prospective, multicenter study on 30 maxillae and 103 mandibles. Jemt and Lekholm[31] reported that the survival rate of mandibular implants was 94.5% versus 100% for mandibular prostheses. In the maxillae the implant survival was 72.4% and the prosthesis survival was 77.9%. The authors suggested that the treatment outcome may be predicted by bone volume and quantity. A prospective study by Johns et al. reported on maxillary IODs at 1 year, 3 years, and 5 years.[32-34] Sixteen patients were followed throughout the whole study with a cumulative success rate of 78% for prostheses and 72% for implants. A pooled implant survival of maxillary removable designs was reported at 76.6% at 5 years.[22-27,29-35]

Alternatively, Misch followed 75 maxillary IODs (RP-4) and 615 implants for 10 years with 97% implant survival and 100% prosthesis survival. The primary differences in these treatment modalities have been a completely implant-supported, retained, and stabilized maxillary IOD (RP-4); a greater implant number; and key implant positions following the guidelines of treatment planning based upon basic biomechanical concepts to reduce failure and decrease risks.

Maxillary Implant Overdenture Treatment Options

Only two treatment options are available for maxillary IODs, whereas five treatment options are available for the mandibular IOD. The difference is due primarily to biomechanical disadvantages of the maxilla compared with the mandible. Independent implants are not an option because bone quality and force direction are severely compromised. Cantilever bars usually are not recommended for the same reasons.[36,37] As such, the two treatment options are limited to an RP-5 restoration

WWW.HIGHDENT.IR

with four to six implants with some posterior soft tissue support, or an RP-4 restoration with 7 to 10 implants (which is completely supported, retained, and stabilized by implants). The CHS is critical for maxillary overdentures, and more often a lack of space may compromise tooth position compared with the mandibular situation (Figure 17-42). The maxillary anterior CHS requirement is greater than the posterior dimension. A minimum of 15 mm of anterior CHS and 12 mm of posterior space is required for IOD because of the greater anterior teeth coronal dimensions and specific locations.

Option 1: Maxillary RP-5 Implant Overdenture

The first treatment option is an RP-5 prosthesis. This option is not as beneficial to the patient, compared with mandibular RP-5 restorations. A maxillary denture often has good retention, support, and stability. An RP-5 maxillary IOD may rock and have more movement than a denture, as the anterior implants act as a fulcrum under the prosthesis. The major advantages of an RP-5 maxillary IOD is the maintenance of the anterior bone and a less-expensive treatment option than an RP-4 or fixed prosthesis. The treatment is less expensive because bilateral sinus grafts are not required and molar implants are not required. Therefore this treatment plan is often used as a transition to an RP-4 prosthesis when financial considerations of the patient require a staged treatment over several years.

The first treatment option for a completely edentulous maxilla uses four to six implants supporting an RP-5 prosthesis, of which at least three are positioned in the premaxilla (Figure 17-43). Based on the poor success rates reported in the literature, specific biomechanical requirements, and poor bone quality, the fewest number of implants for an RP-5 maxillary overdenture should be four, with a wide A-P spread. Implant number and location are more important than implant size, but the implants should be at least 9 mm in length and 3.5 mm in body diameter. The key implants are positioned in the bilateral canine regions (Guideline 3 of treatment planning) and at least one central incisor position (Guideline 2 of treatment planning). Other secondary implants may be placed in the first or second premolar region (Guideline 4 of treatment planning). When an implant cannot be placed in at least one central incisal position, the incisive foramen may be considered for implant insertion. Another alternative is the use of a lateral incisor implant. In such cases, owing to the reduced A-P spread and the lateral incisor in the anterior-most implant site, the second premolar position also should be used on the contralateral side (along with

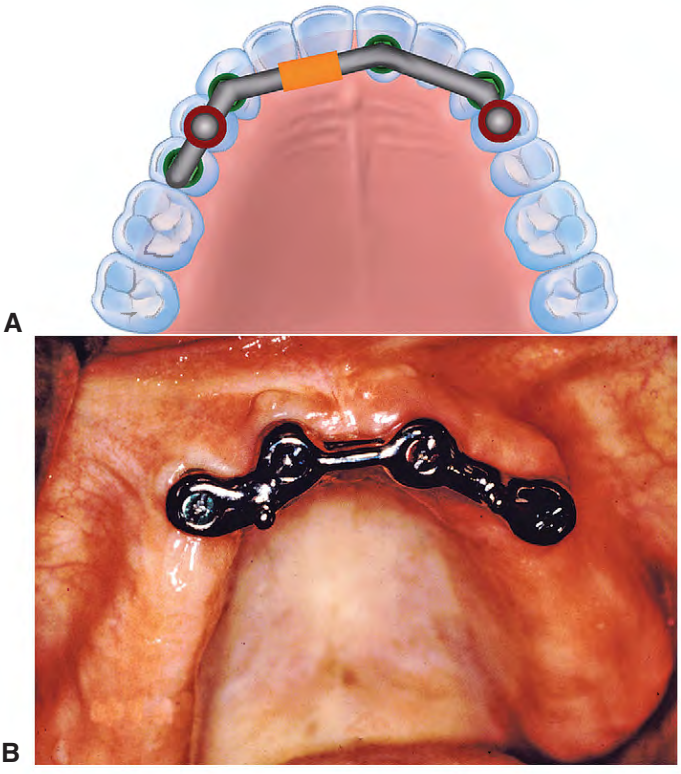


Figure 17-43 **A,** At least four implants, splinted together, are indicated for a maxillary IOD, at least three of which are placed in the premaxilla **(B)**.

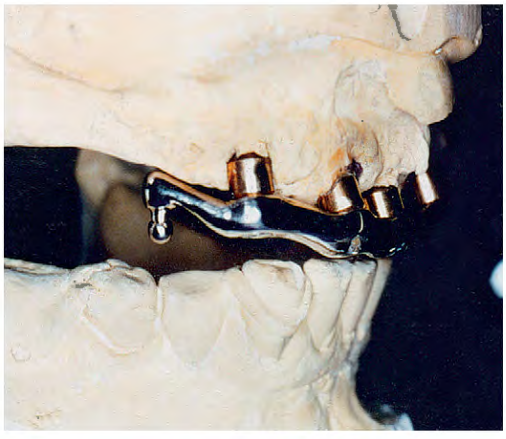


Figure 17-42 An insufficient crown height space is a major problem for the fabrication of a maxillary overdenture.

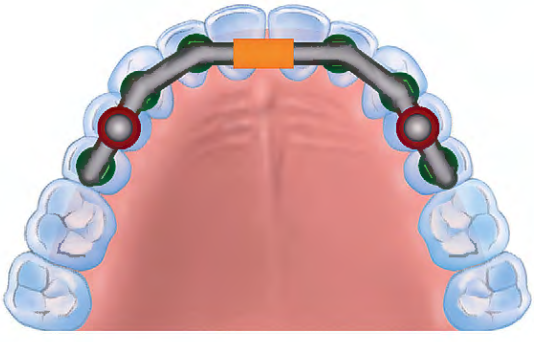


Figure 17-44 An alternate design with six implants includes key implants in the canine regions, implants in the lateral incisor regions, and secondary implants in the second premolar regions to improve the anteroposterior (A-P) spread. Dolder clips or O-rings can be used in such a way as to allow two directions of prosthesis movement.

WWW.HIGHDENT.IR

the canine) to improve the A-P spread. Six implants are often indicated for an RP-5 prosthesis when force factors are greater (Figure 17-44).

The implants are always splinted together with a rigid bar. There is no distal cantilever, and the bar design should follow the dental arch form but slightly lingual to the maxillary anterior teeth. The prosthesis should have at least two directions of movement; however, three or more are preferred. Therefore a Dolder clip or O-ring may be used if it is placed in the center of the arch and perpendicular to the midline. A Dolder clip has a spacer over the clip to allow some vertical movement before rotation. O-rings may be used just distal to the last abutment on each side or between the implants. When intermediate O-rings are used, relief is provided over the top of the implants distal to the bar to allow prosthesis movement toward the tissue under posterior occlusal forces.

The maxillary RP-5 IOD is designed exactly as a complete denture with fully extended palate and flanges. When O-rings are used to retain the restoration, they may be positioned more distal than a Hader clip, often immediately distal to the canine position. The restoration should be allowed to move slightly in the incisal region during function so that the restoration may rotate toward the posterior soft tissue around a fulcrum located in the canine or premolar position. The benefits of an RP-5 maxillary overdenture are retention and stability from the implants. Posterior support is obtained from the soft tissue. Of course, the other primary benefit is the maintenance of the premaxilla bone, because of the implant stimulation. There is also a reduced fee compared with an RP-4 prosthesis, as bilateral sinus grafts are not required for molar implants and the number of implants may be as few as four.

Option 2: Maxillary RP-4 Implant Overdenture

The second option for a maxillary IOD is an RP-4 prosthesis with 7 to 10 implants, which is rigid during function (Figure 17-45). This option is the preferred IOD design because it maintains greater bone volume and provides improved security and confidence to the patient compared with a denture or RP-5 restoration. However, the cost of treatment is similar to a fixed prosthesis. The loss of bone in the premaxilla requires a bone graft or hydroxyapatite graft for lip support for a fixed prosthesis or a labial flange for lip support for a maxillary IOD.

Unfortunately, many practitioners believe that the RP-4 overdenture requires fewer implants and less attention to the biomechanics of occlusal load, just because the restoration is removable. In the author's opinion, this is a primary cause of implant failure in maxillary IODs.

Combined factors such as reduced cost, patient fear of bone grafting, and lack of advanced training of the doctor are often the determining factors motivating the choice for a maxillary IOD. Bone grafts for the entire premaxilla for a fixed prosthesis may require the iliac crest as a donor site because larger volumes of bone are required.

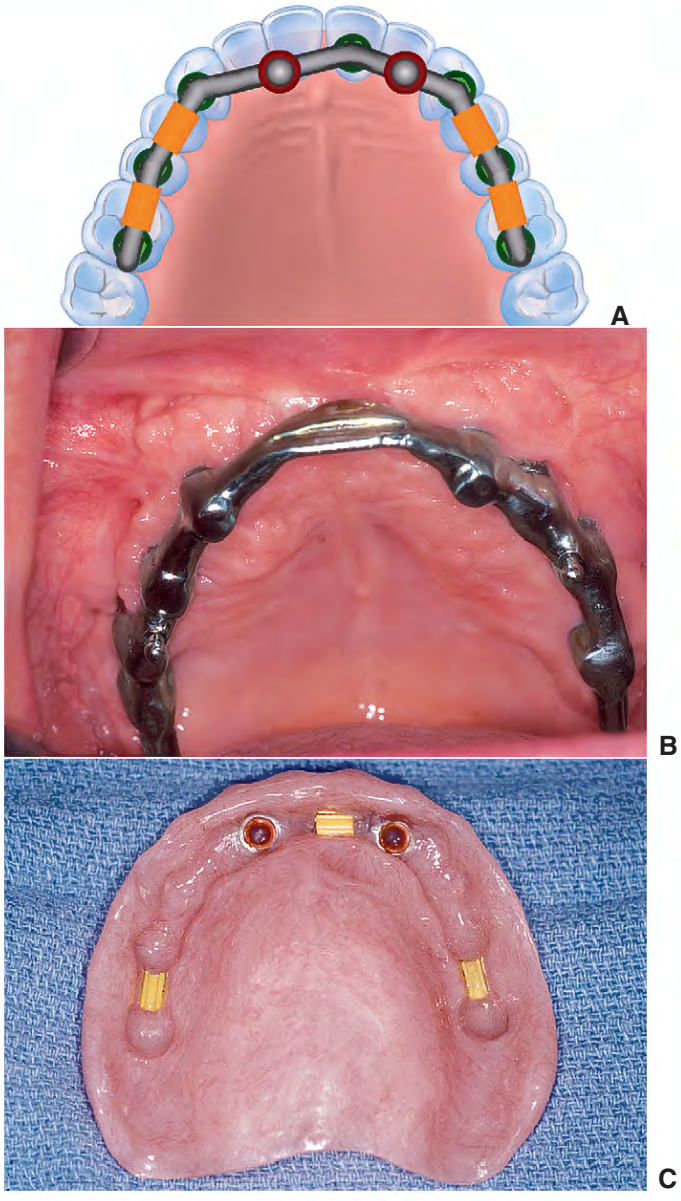


Figure 17-45 **A** and **B,** A maxillary overdenture bar RP-4 prosthesis is supported by 7 to 10 implants and is rigid during function. **C,** A maxillary IOD should have a palatal coverage to decrease fracture, food impaction, and speech problems.

Treatment planning for RP-4 maxillary overdentures is very similar to fixed prosthesis, because the IOD is fixed during function. Two of the key implant positions for the RP-4 maxillary IOD are in the bilateral canines and distal half of the first molar positions (Guideline 3 in treatment planning). These positions usually require sinus grafts in the molar position. Additional posterior implants are located bilaterally in the premolar position (Guideline 2 in treatment planning), preferably the second premolar site. In addition, at least one anterior implant between the canines often is required (Guideline 2 in treatment planning). The anterior implant often

WWW.HIGHDENT.IR

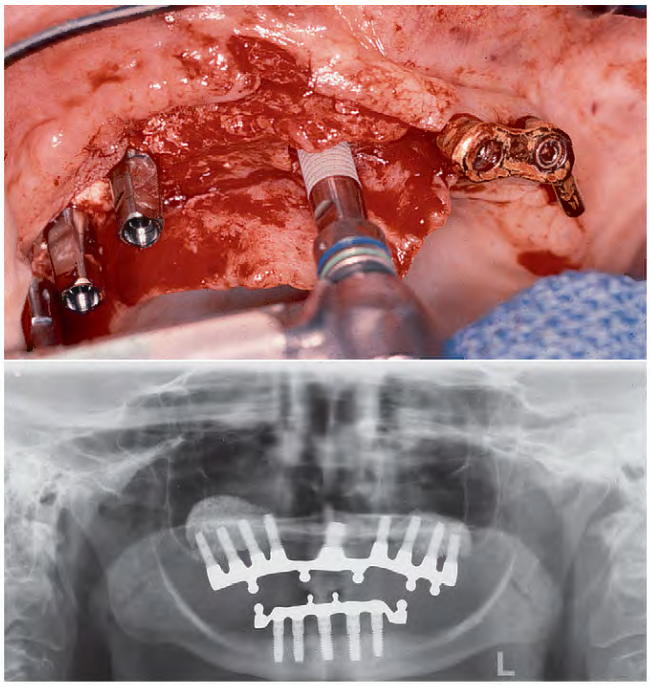


Figure 17-46 **A,** An anterior implant may be positioned in the incisive canal when the residual ridge is insufficient for an implant in the central incisor position. **B,** A panoramic radiograph of maxillary and mandibular implants supporting RP-4 prostheses. The central incisive implant allows the use of three implants in the premaxilla, splinted to posterior implants.

may be placed in the incisive canal, when inadequate bone width is present (Figure 17-46). Therefore, seven implants is the minimum number for an RP-4 treatment option. When force factors are greater, the next most important sites are the second molar positions (bilaterally) to increase the A-P spread and improve the biomechanics of the system. A tenth implant may be placed in the premaxilla for a tapered arch form.

The 7 to 10 implants are splinted together around the arch with a rigid bar (Guideline 5 in treatment planning). Four or more attachments are usually positioned around the arch. This provides a retentive, stable overdenture prosthesis. Usually palatal coverage is maintained. This helps prevent speech problems and food impaction.

The occlusal scheme for this RP-4 restoration is similar to a fixed prosthesis: centric occlusion around the arch and only anterior contact during mandibular excursions (unless opposing a mandibular complete denture). The maxillary overdenture should be removed during sleep to prevent nocturnal parafunction. If the patient wears maxillary and mandibular overdentures, only the mandibular restoration needs to be removed.

Fixed Prosthesis versus Overdenture Complications

The palatal coverage of most maxillary IODs should be similar to a complete denture. The extent of range from full to horseshoe palate designs has been reported in the literature with varied degrees of success.[26,27,36] Many maxillary denture wearers accommodate easily to the acrylic resin palate. Yet many restoring dentists routinely eliminate the palate on maxillary implant, overdentures with consequences such as food entrapment (because the tongue often crushes food against the palate and pushes the food debris under the restoration) and impaired speech (because air is forced under the palatal flange and over the labial flange of the denture). The patient rarely complains of these two problems with a denture and, as a result, is unhappy with the final implant restoration. In addition, the risk of prosthesis fracture is increased when the palate is removed because the bulk of acrylic is reduced. Therefore, the palate of the prosthesis usually should be maintained with the IOD.

Some patients express a primary desire to eliminate the palate of the maxillary complete denture. These patients include gaggers and patients uncomfortable with anything approaching the soft palate, patients with tori or exostoses, singers and actors because of a perceived change of voice caused by the change in volume from the prosthesis, food and wine consumers who use their palate to taste subtle differences in preparations, and a new denture wearer unfamiliar with the palatal aspect of a maxillary denture. As a result, patient needs and desires may require the natural palate of the patient to be left uncovered when wearing a maxillary overdenture.[38,39]

To reduce the complications of speech or food impaction, the following technique has been used with some success. The palate of the preexisting prosthesis is coated with a pressure-indicating paste or spray. The patient is asked to pronounce the linguoalveolar consonants *T* and *D*. In dentate patients, when these sounds are produced, the tip of the tongue contacts the anterior alveolar ridge and the sides of the tongue are in tight contact with the maxillary teeth and palatal gingiva. The maxillary overdenture palate is not eliminated any farther than 5 mm posterior to the tongue contact area. This ensures that the tongue will still contact the acrylic resin on the palate and will prevent food and air from being forced under the denture. The processing model for the prosthesis then is scored 1 mm wide and 1 mm deep with a round bur corresponding to this position. The score line proceeds from the hamular notch in the posterior, along the hard palate 5 mm medial to the alveolar ridge–palatal line angle (position of the greater palatine artery), to the anterior aspect 5 mm distal to the tongue position noted previously. The cast is not scored over the midpalatal suture because this soft tissue is very thin and cannot be depressed readily. When the denture is processed, a small lip of acrylic resin fills this score line, and when the overdenture is inserted, it will depress the tissue along this region gently and ensure intimate tissue contact. This further

WWW.HIGHDENT.IR

prevents food and air from being pushed under the overdenture. Because the "D" and "T" position of the tongue is several millimeters posterior to the position of the maxillary teeth, several millimeters of acrylic resin remain on the premaxilla. This reduces the risk of fracture of the maxillary overdenture.

The anterior implants, connection bar, and attachments should be lingual to the position of the anterior teeth so as not to interfere with proper denture tooth position. However, this position may increase the height of the palatal slope in the region of the premaxilla compared with the original denture. To reduce this occurrence, a low-profile bar and attachment is often the design of choice to minimize the prosthesis bulk. Before designing the connecting bar and attachment design, a vacuum or press form is made of the contour of the preexisting denture and overdenture try-in prosthesis (similar to the method used for the surgical template), and the template helps design the bar-attachment system within the confines of the final prosthesis.

SUMMARY

Maxillary IODs may be as predictable as mandibular overdentures when biomechanical considerations specific to the maxilla are incorporated in the treatment plan. In general, this requires implants in greater numbers and a greater awareness of prosthetic principles.

Only two maxillary IOD treatment options are available. The fewest number of implants for this restoration is four to six implants to support an RP-5 prosthesis. A rigid IOD (RP-4) most often requires the placement of seven or more implants. In other words, maxillary IODs are completely different than their mandibular counterpart. In the completely edentulous maxilla, an IOD is often the treatment of choice. Unlike in the mandible, the maxillary lip often requires additional support as a consequence of bone loss. An ideal high lip line exposes the interdental papillae between the anterior teeth. Using overdentures to replace the hard and soft tissue is easier than attempting to do this with bone and soft tissue or porcelain-to-metal restorations.

A completely implant-supported overdenture requires the same number and position of implants as a fixed restoration. Thus sinus grafts and anterior implants usually are indicated, whether the restoration is fixed or removable.

References

1. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
2. Pietrokowski J: The bony residual ridge in man, *J Prosthet Dent* 34:456-462, 1975.
3. Tatum H: Maxillary and sinus implant reconstruction, *Dent Clin North Am* 30:207-229, 1986.
4. Atwood DA, Coy WA: Clinical cephalometric and densitometric study of reduction of residual ridges, *J Prosthet Dent* 26:200-295, 1971.
5. Gruber H, Solar P, Ulm C: Anatomie und Atrophie bedingte veranderungen der kiefer knochen. In Watzek G, editor: *Enossale Implantate in der Orale Chirurgie,* Berlin, 1993, Quintessence.
6. Botticelli D, Berglundh T, Lindhe J: Hard-tissue alterations following immediate implant placement in extraction sites, *Clin Periodontol* 31:820-828, 2004.
7. Schropp L, Wenzel A, Kostopoulos L et al: Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study, *Int J Periodontics Restorative Dent* 23:313-323, 2003.
8. Misch CE: Bone character, second vital implant criterion, *Dent Today* 7:39-40, 1988.
9. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing and progressive bone loading, *Int J Oral Implantol* 6:23-31, 1991.
10. Lynn BD: The significance of anatomic landmarks in complete denture service, *J Prosthet Dent* 14:456, 1964.
11. Harper RN: The incisive papilla: the basis of a technique to reproduce the positions of key teeth in prosthodontics, *J Dent Res* 27:661, 1948.
12. Misch CE: Treating the edentulous premaxilla. In *Misch Implant Institute manual,* Dearborn, Mich, 1991.
13. Misch CE: Premaxilla implant considerations: surgery and fixed prosthodontics. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.
14. Misch CE: Partial and complete edentulous maxilla implant treatment plans. In Misch CE, editor: *Dental implant prosthetics,* St Louis, 2005, Mosby.
15. Shillinburg HT, Hobo S, Howell D et al: Treatment planning for the replacement of missing teeth. In Shillinburg HI, Hobo S, editors: *Fundamentals of fixed prosthodontics,* ed 3, Chicago, 1997, Quintessence.
16. Misch CE, Bidez MW: Implant protected occlusion: a biomechanical rationale, *Compend Contin Educ Dent* 15: 1330-1343, 1994.
17. Bidez MW, Misch CE: The biomechanics of inter-implant spacing. In *Proceedings of the Fourth International Congress of Implants and Biomaterials in Stomatology,* Charleston, SC, May 24-25, 1990.
18. Widbom C, Soderfeldt B, Kronstrom M: A retrospective evaluation of treatments with implant-supported maxillary overdentures, *Clin Implant Dent Relat Res* 7:166-172, 2005.
19. Kiener P, Oetterli M, Mericske E et al: Effectiveness of maxillary overdentures supported by implants: maintenance and prosthetic complications, *Int J Prosthodont* 4:133-140, 2001.
20. Tarnow DP, Cho SC, Wallace SS: The effect of interimplant distance on the height of interimplant bone crest, *J Periodontol* 71:546-569, 2000.
21. Degidi M, Misch CE: Vertical and horizontal peri-implant bone loss: relevance of interimplant spacing. Submitted for publication.
22. Bryant SR, MacDonald-Jankowski D, Kwonsik K: Does the type of implant prosthesis affect outcomes for the completely edentulous arch? *Int J Oral Maxillofac Implants* 22:117-139, 2007.
23. Ivanoff CH, Grondahl K, Bergstrom C et al: Influence of bicortical or monocortical anchorage on maxillary implant stability: a 15-year retrospective study of Brånemark System Implants, *Int J Oral Maxillofac Implants* 15:103-110, 2000.
24. Jemt T, Bergendal B, Arvidson K et al: Implant-supported welded titanium frameworks in the edentulous maxilla:

WWW.HIGHDENT.IR

a 5-year prospective multicenter study, *Int J Prosthodont* 15:544-548, 2002.
25. Engfors I, Ortorp A, Jemt T: Fixed implant-supported prostheses in elderly patients: a 5-year retrospective study of 133 edentulous patients older than 79 years, *Clin Implant Dent Relat Res* 6:190-198, 2004.
26. Rasmusson L, Roos J, Bystedt H: A 10-year follow-up study of titanium dioxide-blasted implants, *Clin Implant Dent Relat Res* 7:36-42, 2005.
27. Smedberg JI, Nilner K, Fryholm A: A six-year follow-up study of maxillary overdentures on osseointegrated implants, *Eur J Prosthodont Restor Dent* 7:51-56, 1999.
28. Tarnow D, Eliag N, Fletcher P et al: Vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants, *J Periodontol* 74:1785-1788, 2003.
29. Cox JF, Zarb GA: The longitudinal clinical efficacy of osseointegrated dental implants: a 3-year report, *Int J Oral Maxillofac Implants* 2:91-100, 1987.
30. Palmqvist S, Sondell K, Swartz B: Implant-supported maxillary overdentures: outcome in planned and emergency cases, *Int J Oral Maxillofac Implants* 9:184-190, 1994.
31. Jemt T, Lekholm U: Implant treatment in edentulous maxillae: a 5-year follow-up report on patients with different degrees of jaw resorption, *Int J Oral Maxillofac Implants* 10: 303-311, 1995.
32. Johns RB, Jemt T, Heath MR et al: A multicenter study of overdentures supported by Brånemark implants, *Int J Oral Maxillofac Implants* 7:513-522, 1992.
33. Hutton JE, Heath MR, Chai JY et al: Factors related to success and failure rates at 3-year follow-up in a multicenter study of overdentures supported by Brånemark implants, *Int J Oral Maxillofac Implants* 10:33-42, 1995.
34. Jemt T, Chai J, Harnett J et al: A 5-year prospective multicenter follow-up report on overdentures supported on osseointegrated implants, *Int J Oral Maxillofac Implants* 11:291-298, 1996.
35. Chan MF, Narhito, de Bart C et al: Treatment of the atrophic edentulous maxilla in the implant supported overdentures: a review of the literature, *Int J Prosthodont* 11:7-15, 1998.
36. Kramer A, Weber H, Benzing U: Implant and prosthetic treatment of the edentulous maxilla using a bar supported prosthesis, *Int J Oral Maxillofac Implants* 7:251-255, 1992.
37. Benzing UR, Gail H, Weberr H: Biomechanical aspects of two different implant-prosthetic concepts for the edentulous maxilla, *Int J Oral Maxillofac Implants* 10:188-198, 1995.
38. Seifert E, Runte C, Riebandt M et al: Can dental prostheses influence vocal parameters? *J Prosthet Dent* 81:579-585, 1999.
39. Darley FL: Speech pathology. In Laney WR, Gibilisco JA, editors: *Diagnosis and Treatment in Prosthodontics,* Philadelphia, 1983, Lea & Febiger.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 18

Treatment Planning for the Edentulous Posterior Maxilla

Carl E. Misch

Maxillary posterior partial or complete edentulism is one of the most common occurrences in dentistry. Seven percent of the adult population in the United States (12 million people) are missing all of their maxillary teeth and have at least some mandibular dentition—a condition that occurs 35 times more frequently than complete mandibular edentulism opposing maxillary teeth.[1,2] In addition, 10.5% of the adult population has no teeth. Therefore 30 million people in the United States or 17.5% of the adult population are missing all of their maxillary teeth. In addition, 20% to 30% of the adult partially edentulous population older than 45 years of age is missing maxillary posterior teeth in one quadrant, and 15% of this age group is missing maxillary dentition in both posterior regions.[2] In other words, approximately 40% of adult patients are missing at least some maxillary posterior teeth. Therefore the maxillary posterior region is one of the most common areas to be involved in an implant treatment plan to support a fixed or removable prosthesis.

The maxillary posterior edentulous region presents many unique and challenging conditions in implant dentistry. However, existing proven treatment modalities make procedures in this region as predictable as in any other intraoral region. Most noteworthy surgical methods include sinus grafts to increase available bone height, onlay grafting to increase bone width, and modified surgical approaches to insert implants in poorer bone density.[3] Grafting of the maxillary sinus to overcome the problem of reduced vertical available bone has become a very popular and predictable procedure over the last decades. After the initial introduction by Tatum in the mid-1970s and the initial publication of Boyne and James in 1980, many studies have been published about sinus grafting with results higher than 90%.[4-30]

This chapter addresses the treatment planning concepts specific to the maxillary posterior partial or complete edentulous regions.

ANATOMICAL CONSIDERATIONS FOR THE POSTERIOR MAXILLA

Local anatomical conditions of the edentulous alveolar ridges in the posterior maxilla may be unfavorable for implant placement. The available alveolar bone height is lost in the posterior maxilla as a result of periodontal disease before tooth loss. The maxillary molar regions have distal furcation involvement frequently, because the furca is directly under the distal contact and has no facial or palatal access for hygiene. The furca is also narrower than many dental curettes, and it is difficult to eliminate calculus after it has formed. As a result, periodontal disease is common and is associated with loss of bone height before tooth loss.

The dentate maxilla has a thinner cortical plate on the facial compared with the mandible. In addition, the trabecular bone of the posterior maxilla is finer than other dentate regions. The loss of maxillary posterior teeth results in an initial decrease in bone width at the expense of the labial bony plate. The width of the posterior maxilla decreases at a more rapid rate than in any other region of the jaws.[27] The resorption phenomenon is accelerated by the loss of vascularization of the alveolar bone and the existing fine trabecular bone type. However, because the initial residual ridge is so wide in the posterior maxilla, even with a 60% decrease in the width of the ridge, adequate-diameter root form implants usually can be placed. Moreover, the ridge progressively shifts toward the palate until the ridge is resorbed into a medially positioned narrower bone volume.[31] The posterior maxilla continues to progressively remodel toward the midline as the bone resorption process continues. This will result in the buccal cusp of the final restoration often being cantilevered facially to satisfy esthetic requirements at the expense of biomechanics in the moderate to severe atrophic ridges (Figure 18-1).

WWW.HIGHDENT.IR

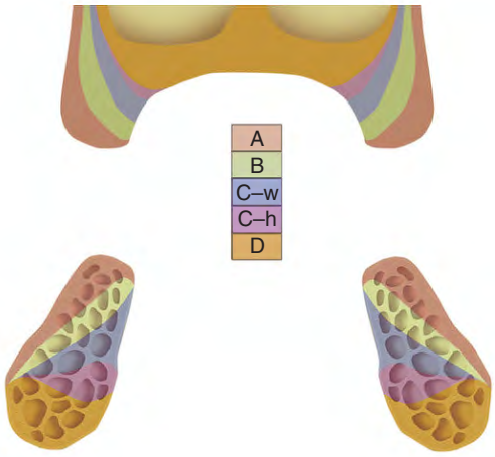


Figure 18-1 The loss of posterior teeth causes resorption of the posterior maxilla. As the edentulous ridge resorbs from Division A to Division D, the crest of the ridge shifts toward the palate. As a consequence, without facial augmentation, the endosteal implant may be placed under the lingual cusp of the original natural tooth position.

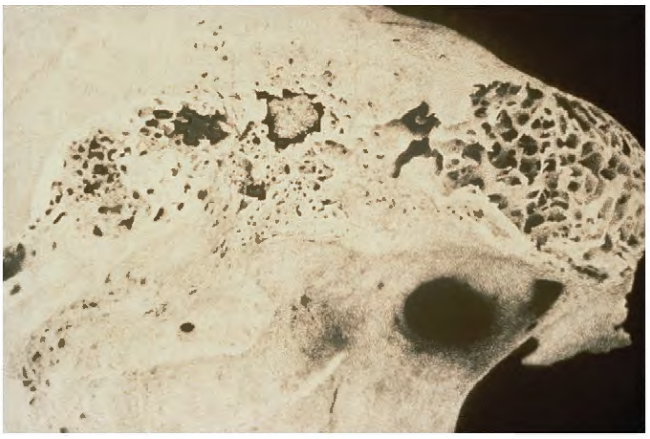


Figure 18-2 The density of the bone in the edentulous posterior maxilla is poorer than in any other region. Because of the lack of cortical bone on the crest, the thin trabecular bone is less strong and has a lower modulus of elasticity.

Special Considerations for the Posterior Maxilla

The crown height space should be evaluated before implant placement. After the occlusal plane is properly restored or modified, the crown height space ideally should be greater than 8 mm. When less clinical space is available for prosthodontic reconstruction, a gingivectomy is first considered, because it is not uncommon for excess tissue thickness to be present in this region. However, if tissue reduction cannot correct the clinical crown height problem, osteoplasty, or vertical osteotomy of the maxillary posterior alveolar process are indicated to restore the correct ridge orientation before surgery.

Poor Bone Density

In general, the bone quality is poorest in the edentulous posterior maxilla compared with any other intraoral region.[32] A literature review of clinical studies from 1981 to 2001 reveals the poorest bone density may decrease implant loading survival by an average of 16% and has been reported as low as 40%.[33] The cause of these failures is related to several factors. Bone strength is directly related to its density, and the poor density bone of this region is often 5 to 10 times weaker in comparison to bone found in the anterior mandible.[34] Bone densities directly influence the percent of implant-bone surface contact, which accounts for the force transmission to the bone. The bone-implant contact is least in D4 bone compared with other bone densities. The stress patterns developed in poor bone density migrate farther toward the apex of the implant. As a result, bone loss is more pronounced and occurs also along the implant body, rather than only crestally as in other denser bone conditions. Type IV (D4) bone also exhibits the greatest biomechanical elastic modulus difference when compared with titanium under load.[34] This biomechanical mismatch develops a higher strain condition to the bone, which may be in the pathologic overload range. As such, strategic choices to increase bone-implant contact are suggested.

In the posterior maxilla, the deficient osseous structures and an absence of cortical plate on the crest of the ridge further compromise the initial implant stability at the time of insertion (Figure 18-2). The labial cortical plate is thin and the ridge is often wide. As a result, the lateral cortical bone-implant contact to stabilize the implant is often insignificant. Therefore initial healing of an implant in Type IV bone is often compromised, and clinical reports indicate a higher initial healing success than with D2 or D3 bone.

ANATOMY

The maxillary sinuses were first illustrated and described by Leonardo Da Vinci in 1489 and later documented by the English anatomist Nathaniel Highmore in 1651.[35] The maxillary sinus—or antrum of Highmore—lies within the body of the maxillary bone and is the largest and first to develop of the paranasal sinuses (Figure 18-3). Adult maxillary sinuses are pyramid-shaped, air-filled cavities that are bordered by the nasal cavity. There is much debate about the actual function of the maxillary sinus. Possible theorized roles of the sinus include weight reduction of the skull, phonetic resonance, participation of warming and humidification of inspired air, and olfaction. A biomechanical adaptation of the maxillary sinus directs forces away from the orbit and cranial cavity when a blow is delivered to the midface.

Expansion of the Maxillary Sinus

A primary pneumatization occurs at about 3 months of fetal development by an outpouching of the nasal

WWW.HIGHDENT.IR

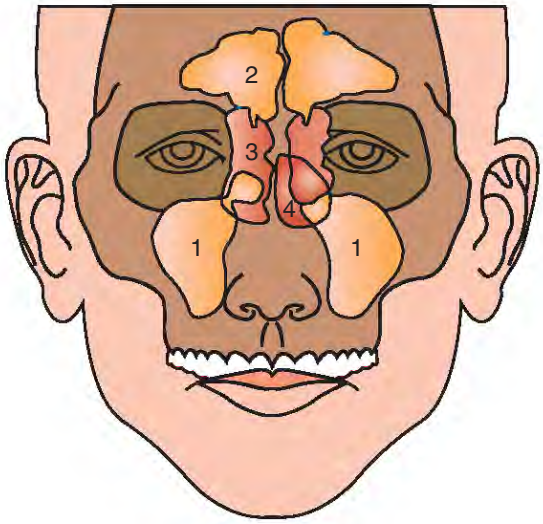


Figure 18-3 The maxillary sinus *(1)* is the largest of the four paranasal sinuses. The initial maxillary sinus formation is completed at age 16 to 18 years. *2,* Frontal sinus; *3,* ethmoid sinus; *4,* sphenoid sinus.

mucosa within the ethmoid infundibulum. At that time, the maxillary sinus is a bud situated at the infralateral surface of the ethmoid infundibulum between the upper and middle meatus.[36] Prenatally, a secondary pneumatization occurs. At birth, the sinus is still an oblong groove on the mesial side of the maxilla just above the germ of the first deciduous molar.[36]

At birth, the sinus cavities are filled with fluid. Postnatally and until the child is 3 months old, the growth of the maxillary sinus is closely related to the pressure exerted by the eye on the orbit floor, the tension of the superficial musculature on the maxilla, and the forming dentition. As the skull matures, these three elements influence its three-dimensional development. At 5 months, the sinus appears as a triangular area medial to the infraorbital foramen.[37,38]

During the child's first year, the maxillary sinus expands laterally underneath the infraorbital canal, which is protected by a thin bony ridge. The antrum grows apically and progressively replaces the space formerly occupied by the developing dentition. The growth in height is best reflected by the relative position of the sinus floor. At 12 years of age, pneumatization extends to the plane of the lateral orbital wall, and the sinus floor is level with the floor of the nose. During later years, pneumatization spreads inferiorly as the permanent teeth erupt. The adult sinus has a volume of approximately 15 mL ($34 \times 33 \times 23$ mm). The main development of the antrum occurs as the permanent dentition erupts and pneumatization extends throughout the body of the maxilla and the maxillary process of the zygomatic bone. Extension into the alveolar process lowers the floor of the sinus about 5 mm. Anteroposteriorly, the sinus expansion corresponds to the growth of the midface and is completed only with the eruption of the third permanent molars when the young person is about 16 to 18 years of age.[35-39]

In the adult, the sinus appears as a pyramid of four bony walls, the base of which faces the lateral nasal wall and the apex of which extends toward the zygomatic bone (Figure 18-4). The floor of the maxillary sinus cavity is reinforced by bony or membranous septa joining the medial or lateral walls with oblique or transverse buttresslike webs. They develop as a result of genetics and stress transfer within the bone over the roots of teeth. These have the appearance of reinforcement webs in a wooden boat and rarely divide the antrum into separate compartments. These elements are present from the canine to the molar region and tend to disappear in the maxilla of the long-term edentulous patient when stresses to the bone are reduced. Karmody found that the most common oblique septum is located in the superior anterior corner of the sinus or infraorbital recess (which may expand anteriorly to the nasolacrimal duct).[40] The medial wall is juxtaposed with the middle and inferior meatus.

Although the maxillary sinus maintains its overall size while the teeth are present, an expansion phenomenon of the maxillary sinus occurs with the loss of posterior teeth.[37] The antrum expands in both inferior and lateral dimensions. This expansion may even invade the canine eminence region and proceed to the lateral piriform rim of the nose. The dimension of available bone height of the posterior maxilla is greatly reduced as a result of dual resorption from the crest of the ridge and pneumatization of the sinus after the loss of teeth. The sinus expansion is more rapid than the crestal bone height changes. As a result of the inferior sinus expansion, the amount of available bone in the posterior maxilla greatly decreases in height (Figure 18-5).

After periodontal disease, tooth loss, and sinus expansion, frequently less than 10 mm remain between the alveolar ridge crest and the floor of the maxillary sinus, resulting in inadequate bone quantity for implant placement. A limited review of the literature reveals implants that were 9 mm or less in height may have a 16% lower survival rate compared with those implants longer than 10 mm.[33] Therefore the height of bone is of primary importance for predictable implant support. This limited dimension is compounded by the decrease in bone density and the problem of the resultant medial posterior position of the ridge after resorption of bone width. As a result, failure and complications in the long term of many endosteal implant systems are reported.

Implant Treatment Plans for the Posterior Maxilla

High Occlusal Forces

The occlusal forces in the posterior region are greater than in the anterior regions of the mouth. Studies have shown that the maximum bite force in the anterior

WWW.HIGHDENT.IR

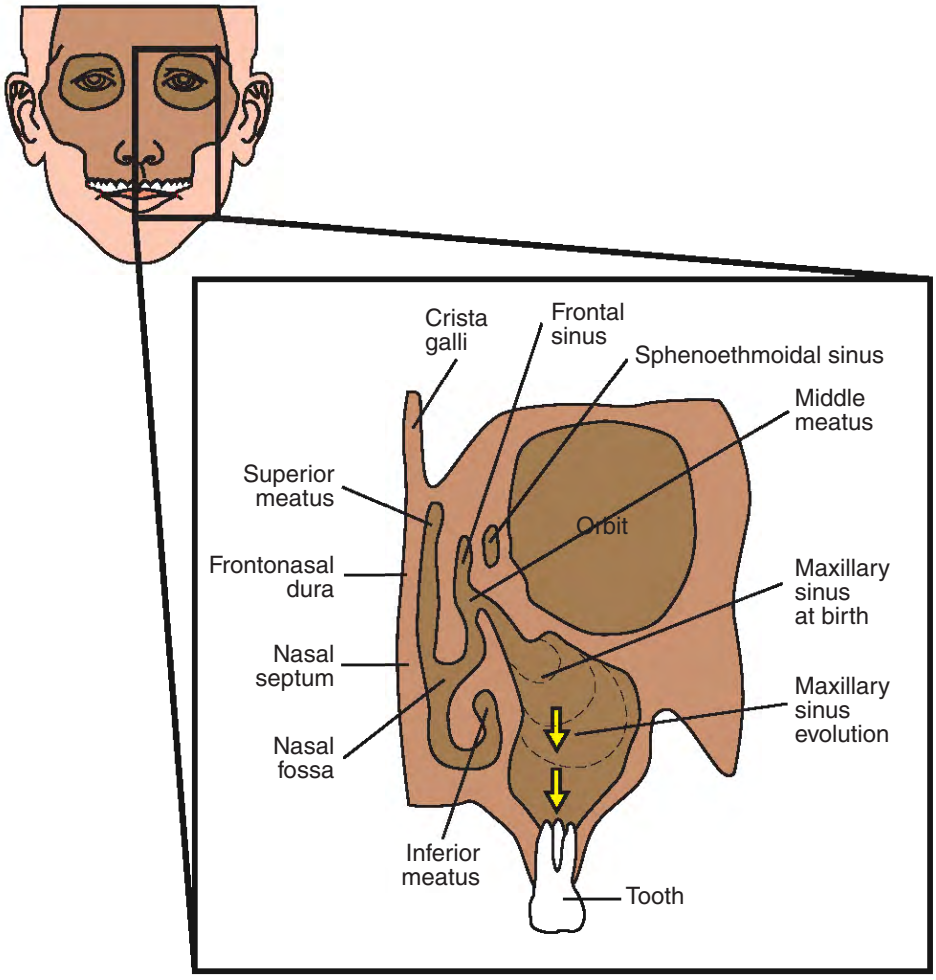


Figure 18-4 The maxillary sinus begins to form in the fetus and by 5 months is the size of a pea, under the eye, and close to the ostium for drainage. By 16 years of age, the maxillary sinus has four thin, bony walls around it. The superior wall separates it from the floor of the orbit. The medial wall contains the ostium to drain the sinus and separates it from the nasal fossa. The lateral wall forms the maxillary bone below the zygomatic arch. The floor of the antrum rests above the roots of the teeth.

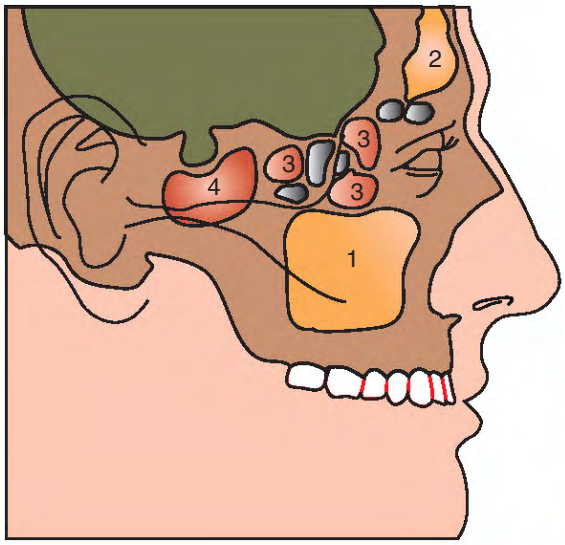
region ranges from 35 to 50 lb/in^2. The bite force in the molar region of a dentate person ranges from 200 to 250 lb/in^2. As a consequence, the maxillary molars of the natural teeth have 200% more surface area than even the premolars and are significantly wider in diameter (Figure 18-6).[3] Both these features reduce the stress to bone, which also reduces the strain of the bone. Following this natural selection, implant support should be greater in the posterior molar region than any other area of the mouth.[3] Therefore the decrease in bone quantity and quality and increased forces should be considered in the treatment plan of this region of the mouth.[41]

Implant Size

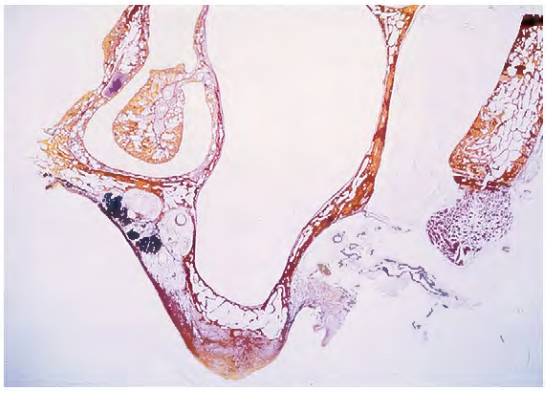
Implant treatment plans should attempt to simulate the conditions found with natural teeth in the posterior maxilla. Because stresses occur primarily at the crestal region, biomechanical designs of implants to minimize their noxious effects should be implemented.[41] Implant diameter is an effective method to increase surface area at the crestal region.[41,42] Ideally, Division B implants are not used in the posterior maxilla. A 12-year retrospective study from 1982 to 1994 of 653 sinus grafts performed by the author revealed 14 implant failures. Eight implant failures were caused by implant fracture at the neck of smaller-diameter implants. Therefore implants of at least 4 mm in diameter are suggested, and 5-mm implants are encouraged in the molar region.

The length of the implant is directly related to the implant width, design, amount of the forces, and bone density. Because implant success after loading is reduced in implants 10 mm and shorter, it is logical to plan for longer implants in the region. In general, 4-mm threaded root form implants should be at least 12 mm in length when the bone density is poor (D3). This usually provides adequate bone-implant contact to dissipate the loads applied to the prosthesis. When the bone density is very poor (D4), 5-mm implants are suggested, also at least 12 mm in length.

WWW.HIGHDENT.IR



A



B

Figure 18-5 **A,** The fourth expansion phenomenon of the maxillary sinus occurs with the loss of the posterior teeth. The anterior portion of the sinus may expand to the piriform rim of the nose. The inferior expansion may approach the crest of the ridge. *1,* Maxillary sinus; *2,* frontal sinus; *3,* ethmoid sinus; *4,* sphenoid sinus. **B,** Coronal section of the posterior region of the edentulous human maxilla. Note expansion of the sinus floor inferiorly far below the level of the floor of the nose. Bone of alveolar ridge is markedly atrophied while the ridge submucosa became fibrotic (×2.4). Stained with Rescorcin Fuchsin stain and counterstained with Ban Gieson. *(Courtesy Mohamed Sharawy, Augusta, Ga.)*

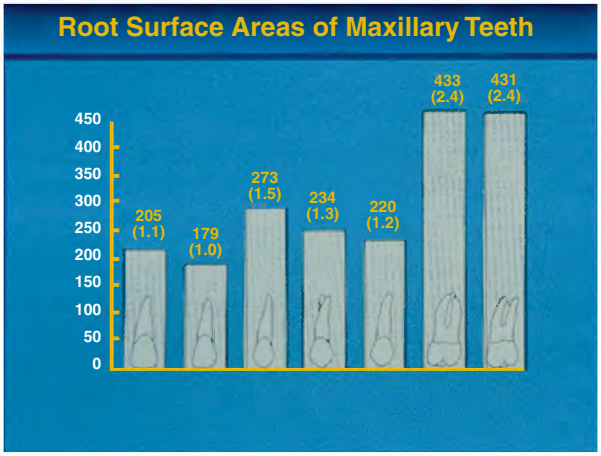


Figure 18-6 The posterior molars have the largest diameter and most surface area of root support compared with any other teeth in the mouth. An implant treatment plan should duplicate this support.

Figure 18-7 The bone-implant contact percentage is lowest in the less dense bone of the posterior maxilla. Therefore implant treatment plans should use a larger and a greater number of implants to compensate for the higher stress conditions.

Implant Number

Implant number is an excellent method to decrease crestal stresses. As a general rule in this area, one implant for each missing tooth is indicated (Figure 18-7). Implants should always be splinted together to reduce stresses to the bone. If stress factors are magnified, two implants for each missing molar are suggested.

Implant Design

Implant design can increase surface area of support. A threaded design implant has 30% to 200% greater surface area compared with a cylinder implant of the same size. Although more difficult to place, the threaded implant in poorer density bone is strongly encouraged. Biomechanical aspects of thread designs affect the total increase in the surface area (i.e., thread pitch, shape, and depth).[41]

Roughened surface conditions or hydroxyapatite coating on the implant have been shown to increase the rate of osseous adaptation to implants, provide greater initial rigid fixation, increase the surface of bone contact

WWW.HIGHDENT.IR

and amount of lamellar bone, and give relative greater strength of the coronal bone around the roughened surface implants when compared with machined or smooth titanium implants.[43-45] Therefore coatings or roughened surfaces are suggested in the compromised D3/D4 bone density.

Dental Contraindications for Implant Treatment of the Posterior Maxilla

A key to long-term success of posterior maxillary implants is the presence of adequate anterior teeth or implants. Therefore the treatment plan should provide for the maintenance or restoration of healthy anterior teeth or Division A bone in the premaxilla for implant placement. A minimum of a healthy natural canine tooth or implant abutments in the canine region for each posterior quadrant are required before posterior implants are considered.

A rule in traditional prosthetics is that a fixed prosthesis is contraindicated when the canine and two adjacent teeth are missing. Therefore when the canine and both premolars are missing, a fixed restoration is contraindicated. A removable prosthesis that has no movement under function is considered a fixed prosthesis and therefore should follow the rules of treatment planning for a fixed prosthesis in relation to implant number and position. The treatment plan of a posterior maxilla should provide for the maintenance or restoration of healthy anterior teeth or adequate bone in the premaxilla for root form implant placement. A minimum of a healthy natural canine tooth or an implant abutment in the canine region is required before posterior implants are considered in the quadrant.

Abnormal intraoral conditions may also compromise the final outcome of sinus grafting procedures or the survival rates of dental implants placed in the grafted sinuses. Therefore relative dental contraindications are similar to those reported in cases of standard implant treatment of edentulous patients.

TREATMENT HISTORY

Treatment of the Posterior Maxilla—Literature Review

Over the years, several strategies have been advocated to restore the posterior maxilla and address the deficiency of bone volume and poor bone quality. The various approaches can be categorized as follows:

- Avoid the sinus and place implants anteriorly, posteriorly, or medially[46-48]
- Place implants and perforate the sinus floor[49,50]
- Use subperiosteal implants[51,52]
- Perform horizontal osteotomy, interpositional bone grafting, and endosteal implants[53,54]
- Elevate sinus floor during implant placement[4,5,7,55-60]
- Perform lateral wall approach sinus graft and simultaneous or delayed implant placement[4-8,15,55,56,61-64]

In the past, implants were inserted in the posterior maxilla without modifying the maxillary sinus topography. Small implants were often placed below the antrum. The decreased surface area compounded by poor bone quality resulted in poor implant stability. Attempts to place larger endosteal implants posterior to the antrum and into the tuberosity and pterygoid plates also resulted in compromised situations. In addition, although feasible from a surgical standpoint, rarely are third- or fourth-molar abutments indicated for proper prosthodontic support. This approach often requires three or more pontics between the anterior and posterior implants. The typical span results with excessive flexibility of the prosthesis, unretained restorations, excess stresses, and implant failure. The thin, porous compact bone often present on the crest of the ridge and on the lateral aspect of the maxilla serves as a poor foundation for subperiosteal implants. Lack of adequate bone height often results in the subperiosteal implant being displaced laterally, off the bony ridge, from occlusal and parafunctional forces.

In the late 1960s, Linkow reported that the blade-vent implant could be blunted and the maxillary sinus membrane slightly elevated to allow implant placement "into" the sinus in the posterior maxilla.[55] This technique required the presence of at least 7 mm of vertical bone height below the antrum. For long-term predictable results, vertical bone height of at least 10 mm for D3 bone in the posterior maxilla has been clinically determined to be necessary when the diameter is 4 mm or more. Because the posterior maxilla often has D3 or D4 bone, implants of traditional design should have even greater height requirements.

Geiger reported that ceramic implants placed through the maxillary sinus floor could heal and stabilize without complication.[49] Brånemark et al. have shown that implants may be placed into the maxillary sinus without consequence if integration occurs between the implants and the bone below the sinus. Yet they also report a higher failure rate (70% success for 5 to 10 years) for this technique.[50] Ashkinazy[48] and others have reported on using tomographic radiographs to determine whether adequate bone exists on the palatal aspect of the maxillary sinus for blade implants. However, Stoler[65] stated that after 25 consecutive computed tomographic scans of maxillae, adequate bone for implant support was not found on the medial aspect of the sinus. Thus it seems that if sufficient bone is present medial to the sinus, it is the rare exception.

In the early 1970s, Tatum[4,5,56,57] began to augment the posterior maxilla with onlay autogenous rib bone to produce adequate vertical bone for implant support. He found that onlay grafts below the existing alveolar

WWW.HIGHDENT.IR

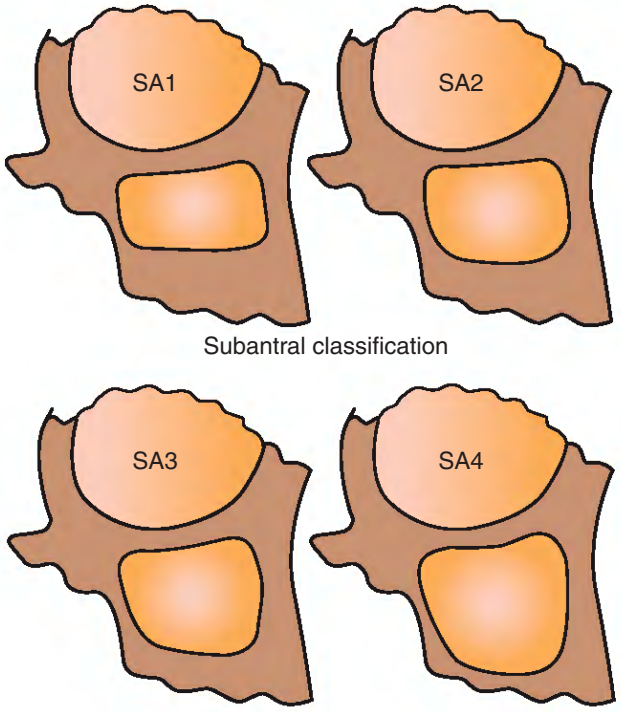


Figure 18-8 In 1987, Misch presented four subantral treatment options based on the amount of bone below the maxillary sinus. Subantral augmentation category 1 (SA1) sinus used traditional implant approaches. Category SA2 used a sinus lift procedure within the osteotomy. For SA3 and SA4 categories, a Tatum sinus graft procedure is performed before implant insertion.

crest would decrease the posterior intradental height significantly, yet very little bone for endosteal implants would be gained. Therefore in 1974 Tatum developed a modified Caldwell-Luc procedure for elevation of the sinus membrane and subantral (SA) augmentation.[4,5] The crestal ridge of the maxilla was infractured and used to elevate the maxillary sinus membrane. Autogenous bone was then added in the area previously occupied by the inferior third of the sinus. Endosteal implants were inserted in this grafted bone after approximately 6 months. Implants were then loaded with final prostheses after an additional 6 months.[4,5]

In 1975, Tatum developed a lateral approach surgical technique that allowed the elevation of the sinus membrane and implant placement in the same surgical appointment. The implant system used was a one-piece ceramic implant, and a permucosal post was required during the healing period. Early ceramic implants were not designed adequately for this procedure and results with the technique were unpredictable. In 1981, Tatum developed a submerged titanium implant for use in the posterior maxilla.[5,56] The advantages of submerged healing, the use of titanium instead of aluminum oxide as a biomaterial, improved biomechanics, and improved surgical technique made this implant modality more predictable.

From 1974 to 1979, the primary graft material for the sinus graft procedure was autologous bone. In 1980 the application of the SA augmentation technique with a lateral maxillary approach was further expanded by Tatum with the use of synthetic bone. The same year, Boyne and James reported on the use of autogenous bone for subantral grafts.[6] Most of the data published in the 1980s were anecdotal or based on very small sample sizes. In 1987, Misch organized a treatment approach to the posterior maxilla based on the amount of bone below the antrum, and in 1989 he expanded the treatment approach to include the available bone width related to surgical approach and implant design (Figure 18-8).[7,63,64] Since then, minor modifications regarding the graft materials or surgical approach have also been proposed.

In the 1990s, the profession developed a much greater interest in the sinus graft technique.[15] Several reports have flourished in the literature, reporting on minor changes in the technique, different materials used in the graft, different origins for the autogenous portion of the graft, histomorphometric data relative to the graft healing, and other retrospective studies relative to the survival rates of implants placed in grafted sinuses with a simultaneous or delayed approach.[62-79]

Long-term results have been reported by Tatum[62] to be above 95%, more than 1500 subantral augmentations performed. The sinus graft procedure has been the most predictable method to grow bone height from 5 to 20 mm compared with any other intraoral bone grafting technique, with a graft success rate and an implant survival rate greater than 95%.

Sinus Graft Options for the Posterior Maxilla

A bone volume classification was published by Misch and Judy in 1987, which followed the bone resorption pattern of edentulous ridges in the maxilla and mandible.[80] The abundant-width ridge for available bone was classified as a Division A ridge. The dimensions of this ridge were described as >5 mm in width, >7 mm in length, >12 mm in height, and an angulation <30 degrees to occlusal load. A residual width of 5 mm was selected for this bone volume because this dimension may adequately support at least a 4-mm-diameter implant, which is the most frequent diameter used as an implant abutment. A Division B edentulous ridge was described as 2.5 to 5 mm in width, 7 mm in mesiodistal length, >12 mm in height, and an angulation to occlusal load of <20 degrees. The decreased width of the bone in all jaw locations primarily occurs from the facial toward the medial aspects of the remaining available bone. Division C–w ridges consisted of 1 to 2.5 mm in width and >12 mm in height. A C–h ridge was inadequate in height (<12 mm) and a crown height space to bone ratio exists greater than 1. A Misch/Judy Division D severely atrophic ridge had significant height resorption and the remaining bone had a crown height to remaining bone ratio >5:1.

WWW.HIGHDENT.IR

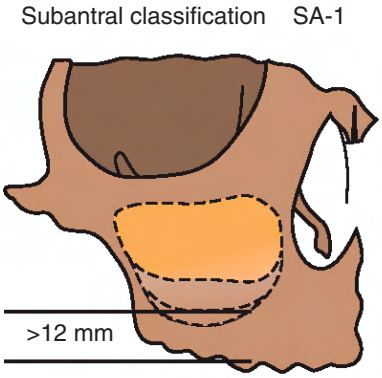


Figure 18-9 Posterior maxilla, with greater than 12-mm height of bone is placed in subantral augmentation option 1. When more than 5-mm width is present, the ridge is Division A; when 2.5 to 5 mm is present, the ridge is Division B and should be augmented in width.

A classification based on a treatment option approach to providing additional prosthodontic abutments in the maxillary posterior edentulous region was presented by Misch in 1987,[7] dependent on the available bone height between the floor of the antrum and the crest of the residual ridge in the region of the ideal implant locations (Table 18-1). This protocol also suggested a surgical approach, bone graft material, and time table for healing before prosthetic reconstruction. In 1988, Cawood and Howell also classified the edentulous posterior maxilla, which included the loss of bone and pneumatization of the maxillary sinus.[79]

In 1995, Misch modified his 1987 classification to include the lateral dimension of the sinus cavity and this dimension was used to modify the healing period protocol, because smaller-width sinuses (0 to 10 mm) form bone faster than larger-width (>15 mm) sinuses. Other classifications of the sinus graft procedure have been proposed by Jensen in 1991[82] and Chiapasco in 2003.[72,83]

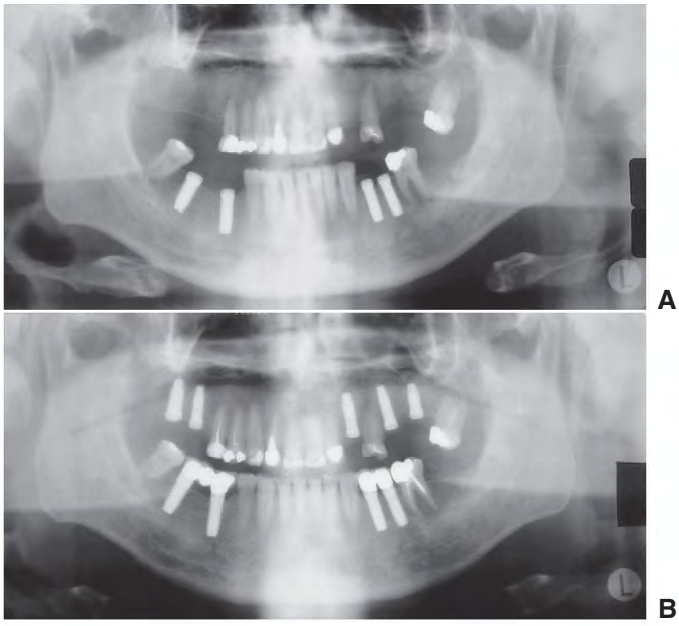


Figure 18-10 **A,** A panoramic radiograph of posterior maxillae with a subantral option 1 condition. **B,** Root form implants, one for each tooth, are the treatment of choice for subantral option 1 conditons.

Misch Maxillary Posterior Classification

Subantral Option 1: Conventional Implant Placement

The first SA treatment option, SA-1, occurs when there is sufficient available bone height to permit the placement of endosteal implants after a usual surgical protocol. In the abundant bone volume (Division A), root form implants are used for prosthetic support. The minimum "ideal" bone height is related to implant design and bone density; however, at least a 12-mm implant in height is suggested for a 4-mm-diameter threaded implant (Figures 18-9 and 18-10).

Patients with narrower bone volume (Division B) may be treated with osteoplasty or augmentation to increase

Table 18-1 Healing Times for Treatment Categories

TIME TREATMENT	HEIGHT (mm)	PROCEDURE	HEALING TIME (MONTHS): GRAFT	HEALING TIME (MONTHS): IMPLANT
SA-1*	>12	Division A root form placement	—	4-6
SA-2	10-12	Sinus lift; simultaneous Division A root form placement	—	6-8
SA-3	5-10	Lateral wall approach sinus graft; delayed Division A root form placement	2-4	4-8†
SA-4	<5	Lateral wall approach sinus graft; delayed Division A root form placement	6-10	4-10†

* Subantral augmentation option.
† Evaluate at implant insertion.

WWW.HIGHDENT.IR

the width of bone. The insertion of smaller surface area implants is not suggested because the forces are greater in the posterior regions of the mouth and the bone density is less than in most regions. In addition, the narrow ridge is often more medial than the central fossa of the mandibular teeth and will result in an offset load on the restoration, which will increase the strain to the bone.

Osteoplasty in the SA-1 posterior maxilla may change the SA category if the height of the remaining bone is less than 12 mm after the bone modification is completed. Augmentation for width may be accomplished with bone spreading, autogenous onlay or appositional grafts. Larger-diameter implants are often required in the molar region, and bone spreading to place wider implants is the most common approach when the bone density is poor. If less than 2.5 mm of width is available in the posterior edentulous region (Division C–w), the most predictable treatment option is to increase width with onlay autogenous bone grafts.[84] After graft maturation, the area is then reevaluated to determine the proper treatment plan classification.

Although a common axiom in implant dentistry is to remain 2 mm or more from an opposing landmark, this is not indicated in the subantral region. As long as the antrum is healthy and instruments do not perforate the thin sinus muscosa lining the sinus floor, no contraindications exist to the preparation or placement of implants at the level of or even through the cortical plate of the sinus floor.

Endosteal implants in the SA-1 category are left to heal in a nonfunctional environment for approximately 4 to 8 months (depending on bone density) before the abutment posts are added for prosthodontic reconstruction. Care is taken to ensure that the implants are not traumatized in any way during the initial healing period. Progressive loading during the prosthetic phases of the treatment are suggested in D3 or D4 bone.

Subantral Option 2: Sinus Lift and Simultaneous Implant Placement

The second subantral option, SA-2, is selected when 10 to 12 mm of vertical bone is present (0 to 2 mm less than the minimum height in SA-1) (Figure 18-11). To obtain the 12 or more millimeters of vertical bone necessary for improved implant survival in ridges of adequate width (Division A), the antral floor is elevated through the implant osteotomy (Figures 18-12 and 18-13). This technique was originally developed by Tatum in 1970 and published by Misch in 1987[7] and many years later by Summers.[4,5,85]

The endosteal implant osteotomy is prepared as determined by the density of bone protocol. The depth of the osteotomy is approximately 1 to 2 mm short of the floor of the antrum. A cupped-shape osteotome of the same diameter as the final osteotomy is selected.[86] It is of a different end shape than osteotomes used for bone spreading.[85] The osteotome is inserted and tapped firmly

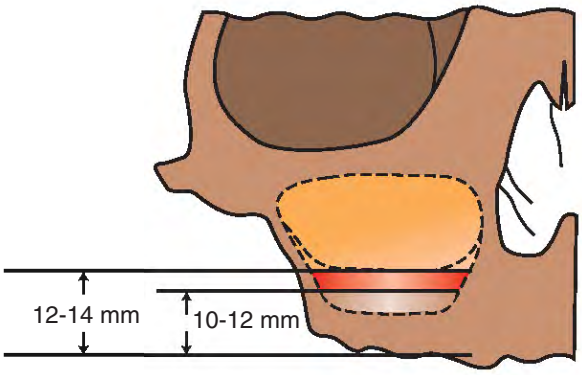


Figure 18-11 A posterior maxilla with 10 to 12 mm of vertical height is treated with a subantral option 2. The sinus floor is raised with the implant osteotomy (sinus lift). When the width of bone is greater than 5 mm, treatment follows Division A with a sinus lift. A width of 2.5 to 5 mm is Division B and should be augmented in width.

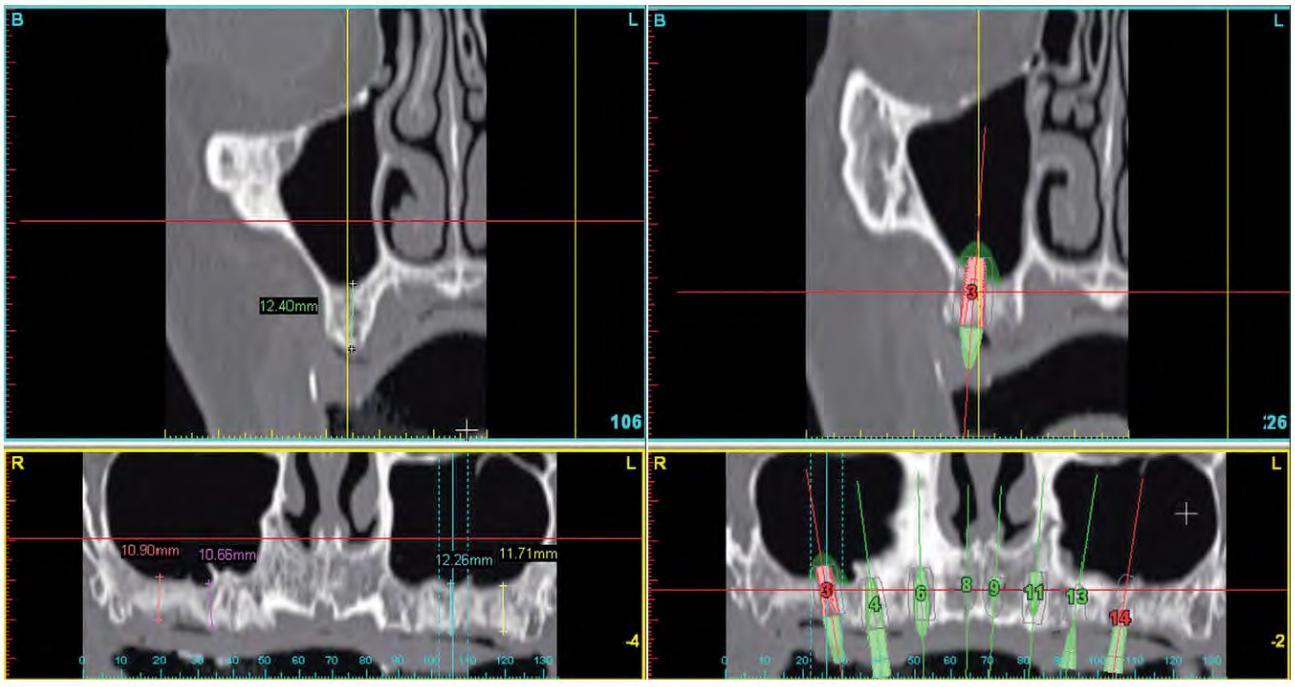
in 0.5- to 1-mm increments beyond the osteotomy until its final position up to 2 mm beyond the prepared implant osteotomy. This surgical approach causes a greenstick-type fracture in the antral floor and slowly elevates the unprepared bone and sinus membrane over the broad-based osteotome.

The implant may be inserted into the osteotomy after the sinus lift and extend up to 2 mm above the floor of the sinus. The implant is slowly threaded into position so that the membrane is less likely to tear as it is elevated. The apical portion of the implant engages the more dense bone on the cortical floor, with bone over the apex, and an intact sinus membrane. The implant may extend up to 2 mm beyond the sinus floor and the 1- to 2-mm bony covering over the apex results in as much as a 4-mm elevation of the sinus mucosa (Figure 18-14). Because of the autologous bone present above the apical portion of the implant, new bone formation is accelerated.

The success of the intact sinus membrane lift cannot be confirmed before or at the time of implant placement. Attempts to feel the elevation of the membrane from within an 8-mm-deep implant osteotomy that is approximately 3 mm in diameter may easily cause tearing of the sinus lining. Four to six months after the surgical procedure, a radiograph may be used to indicate the success of the 0- to 2-mm increased vertical height. The patient's prosthodontic treatment may proceed similar to that in the SA-1 category. If inadequate bone is formed around the apical portion of an implant after initial healing, the progressive loading protocol for D4 bone is suggested.

Some authors have used the SA-2 sinus lift procedure to gain more than 2 mm of implant vertical height or place bone graft materials in the osteotomy site before implant insertion. These blind surgical techniques increase the risk of sinus membrane perforation. When the sinus mucosa is perforated, the graft material may extrude into the antrum and increase the risk of postoperative

WWW.HIGHDENT.IR



A B

Figure 18-12 Treatment plan SA-2 calls for elevation of 0 to 2 mm of the floor of the maxillary sinus at the same time as implant placement. During the treatment plan phase, a computed tomography scan with implant software can be used to visualize the sinus lift and final implant relationship with surrounding bone. *(Courtesy S. Caldwell, El Paso, Tex.)*

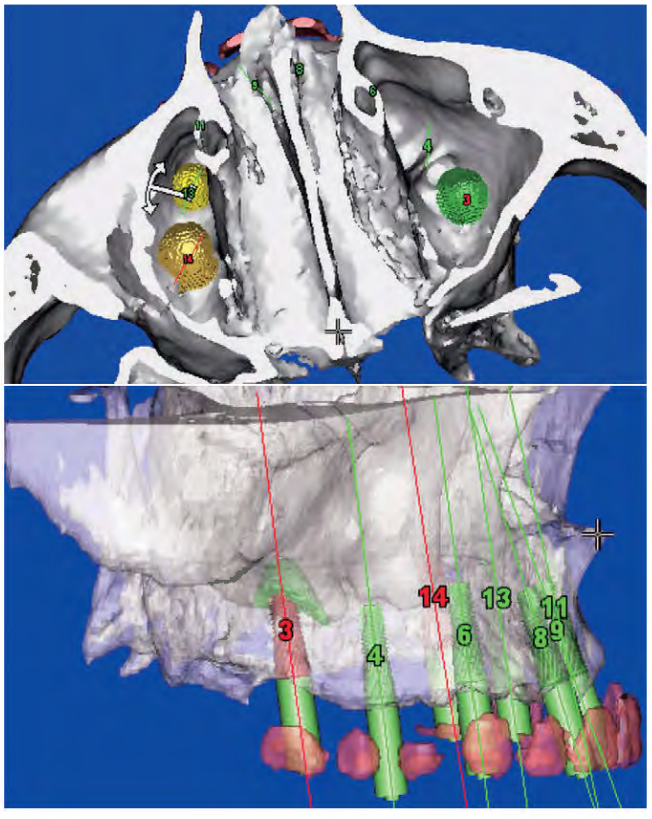


Figure 18-13 **A,** A three-dimensional (3D) view of the maxillary sinus illustrates the membrane being elevated only in the vicinity of the implant apex, creating a halolike image. **B,** A 3D view of the maxillary sinus illustrating implant placement during the treatment planning phase along with the proposed final level of t he sinus floor after SA-2. *(Courtesy S. Caldwell, El Paso, Tex.)*

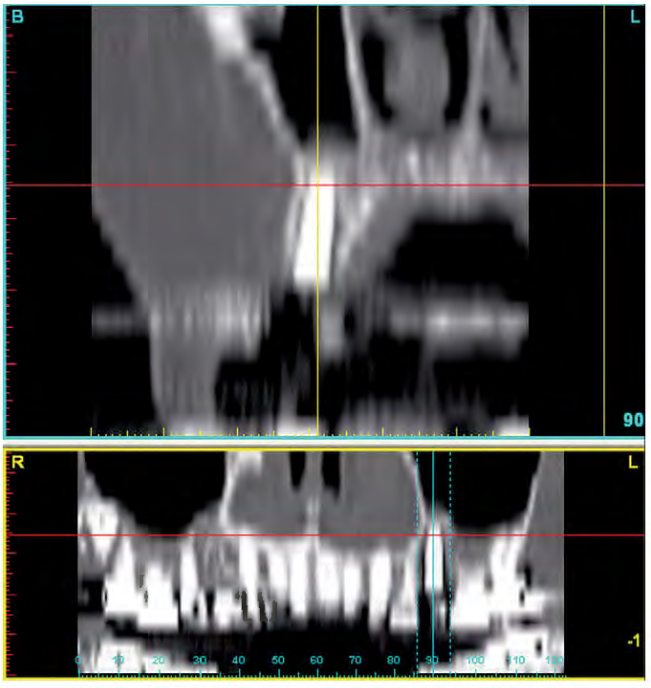


Figure 18-14 After healing, the halo effect at the apex of the implant can be visualized on postoperative imaging. *(Courtesy S. Caldwell, El Paso, Tex.)*

WWW.HIGHDENT.IR

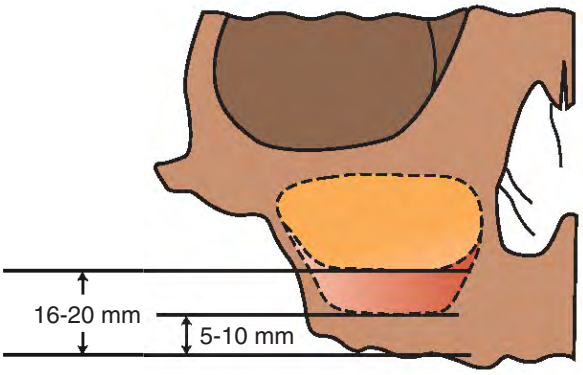


Figure 18-15 A posterior maxilla with 5 to 10 mm between the crest of the ridge and sinus floor is placed in subantral option 3. A Tatum sinus graft is indicated before implant insertion. When greater than 5 mm of width is present, the sinus graft converts the ridge to Division A. When 2.5 to 5 mm of width is present, the Division B ridge should be augmented in width.

infection. If a sinus infection occurs, a bacterial smear layer may accumulate on the implant apex. This prevents future bone-implant contact and may contribute to future sinus infections.

If sinus membrane perforation occurs during the initial implant placement procedure, increased bone height is not likely. This is the primary reason why no more than 2 mm of additional bone height is attempted with this technique. However, even when membrane perforation occurs or no bone grows around the apical end of the implant, the SA-2 technique is of benefit, because the apical end of the implant is surrounded by denser bone. This enhances rigid fixation during healing and increases bone-implant contact, leading to improved loading conditions. As a result of the less predictable outcome, one additional implant may be placed or a larger diameter is suggested in the SA-2 procedure.

Worth and Stoneman have reported a comparable phenomenon of bone growth under an elevated sinus membrane, called *halo formation.*[87] They observed the natural elevation of the sinus membrane around teeth with periapical disease. The elevation of the membrane resulted in new bone formation once the tooth infection was eliminated.

Subantral Option 3: Sinus Graft with Immediate or Delayed Endosteal Implant Placement

The third approach to the maxillary posterior edentulous region, SA-3, is indicated when at least 5 mm of vertical bone and sufficient width are present between the antral floor and the crest of the residual ridge in the area of a needed prosthodontic abutment (Figure 18-15). A Tatum lateral maxillary wall approach is performed just superior to the residual alveolar bone. After the lateral access window and membrane are rotated in and upward to a superior position, a mixture of autogenous bone, alloplast, or allograft material is placed in the space previously occupied by the sinus. When the original ridge is greater than 5 mm in width, the implant may be inserted at the same time as the sinus augmentation under ideal conditions or delayed for a period of 2 or more months before implant insertion[14,60-62,73-75,88-91] (Boxes 18-1 and 18-2). The short delay between graft placement and implant insertion ensures the graft is more stable and is healing without compromise related to postoperative sinus infection. When the original ridge width is Division B or C–w, an onlay graft in conjunction with the sinus augmentation is a possible treatment option.

The 5 to 10 mm of initial bone height in an SA-3 posterior maxilla may have cortical bone on the residual crest, and cortical-like bone on the original antral floor may stabilize an implant that is inserted at the time of the graft and permit its rigid fixation. Therefore an endosteal implant may be inserted at this appointment and has been advocated for some years by this author[7] and others.[14,17,61] However, several advantages tend toward the decision to delay implant placement for approximately 4 months, as follows:

1. The individual rate of healing of the graft may be assessed while the implant osteotomy is being prepared and the implant inserted. The healing time for the implant is no longer arbitrary, but more patient specific.
2. Postoperative sinus graft infection occurs in approximately 3% to 5% of patients, which is greater than the percentage for implant placement surgery.

Box 18-1 Implant Placement (SA-3I)

When the endosteal implants are inserted at the same time as the sinus graft, several conditions are suggested:
1. >5 mm bone height (D3 or better)
2. >6 mm bone width
3. No sinus pathology
4. No history of sinusitis
5. No relative contraindications
6. No parafunction on RPD transitional
7. No sinus membrane perforation

RPD, Removable partial denture.

Box 18-2 No Implant Placement (SA-3)

Implants should not be inserted at the same time as the sinus graft when the following conditions exist:
1. >5 mm bone height (D4)
2. <6 mm bone width
3. Treated sinus pathology
4. History of sinusitis
5. Relative contraindications (smoking, diabetes, periodontal disease)
6. Parafunction on RPD transitional
7. Sinus membrane perforation during surgery

RPD, Removable partial denture.

WWW.HIGHDENT.IR

If the sinus graft becomes infected with an implant in place, a bacterial smear layer may develop on the implant and make future bone contact with the implant less predictable. The infection is also more difficult to treat when the implants are in place, and may result in greater resorption of the graft as a consequence. If the infection can not be adequately treated, the graft and implant must be removed. Therefore there is also a decreased risk of losing the graft and implant if a postoperative infection occurs with a delayed implant insertion. Reports in the literature indeed indicate a higher failure rate of implants when inserted simultaneously compared with a delayed approach.[62,72,83,84]

3. Blood vessels are required to form and remodel bone. An implant in the middle of the sinus graft does not provide a source of blood vessels. It may even make vascular supply more troublesome.
4. Bone width augmentation may be indicated in conjunction with sinus grafts to restore proper maxillomandibular ridge relationships or increase the implant diameter in the molar region. Augmentation may be performed simultaneously with the sinus graft. As a result, larger-diameter implants may be placed with the delayed technique.
5. The bone in the sinus graft is denser with the delayed implant placement. As such, implant angulation and position may be improved because it is not dictated by existing anatomical limitations at the time of the sinus graft.
6. The surgeon may access the sinus graft before implant insertion. On occasion, the sinus graft underfills a region, and the lack of awareness of the condition during implant insertion at the same time results in an implant placed in the sinus proper, rather than the graft site.

The primary disadvantage of delaying the implant placement is the need for an additional surgery. If overall treatment time is a significant factor for the patient, the implant may be inserted after 2 months yet reduce considerably the risk of infection. Otherwise, 4 months or more is typical for implant insertion with the SA-3 technique (Figure 18-16).

Subantral Option 4: Sinus Graft Healing and Extended Delay of Implant Insertion

In the fourth option for implant treatment of the posterior maxilla, SA-4, the subantral region for future endosteal implant insertion is first augmented. This option is indicated when less than 5 mm remains between the residual crest of bone and the floor of the maxillary sinus. There is inadequate vertical bone in these conditions to predictably place an implant at the same time as the sinus graft and less recipient bone to act as a vascular bed for the graft (Figure 18-17). The SA-4 corresponds to a larger antrum and minimal host bone on the lateral, anterior, and distal regions of the graft, because the antrum generally has expanded more aggressively into these regions.

There is also less autologous bone to harvest in the tuberosity, which further delays the bone regeneration in the site. In addition, there are usually fewer septa or webs in the sinus, which increases the bone regeneration, and it typically exhibits longer mediodistal and latero-medial dimensions. Therefore the fewer bony walls, less favorable vascular bed, minimal local autologous bone, and larger graft volume all mandate a longer healing period and slightly altered surgical approach.

The Tatum lateral wall approach for sinus graft is performed as in the previous SA-3 procedure. Most SA-4 regions provide better surgical access than the SA-3 counterparts because the antrum floor is closer to the crest, compared with the SA-3 maxilla. The medial wall of the sinus membrane is elevated at least 16 mm from the crest so that adequate height is available for future endosteal implant placement (Figures 18-18 and 18-19). The combination of graft materials used and their placement are similar to those for the SA-3 technique. However, less autogenous bone is often harvested from the tuberosity, so an additional harvest site may be required, most often from the mandible (i.e., from the ascending ramus).

The augmented region matures for 6 to 10 months before reentry for placement of endosteal implants. The amount of initial healing is related to the antral size (including small, medium, or large lateral-medial size) and the amount of autologous bone used in the inferior one third of the antrum. Typically, the width of crestal bone is wide enough in SA-4 regions for the placement of root form implants after the graft matures.

The implant surgery at reentry for SA-3 or SA-4 is similar to SA-1 with one exception. The periosteal flap on the lateral side is elevated to directly allow inspection of the previous access window of the sinus graft. The previous access window may appear completely healed with bone; soft and filled with loose graft material; or with cone-shaped fibrous tissue ingrowth (with the base of the cone toward the lateral wall); or in any variation states. If the graft site appears clinically as bone, the implant osteotomy and placement follow the approach designated by the bone density.

A hydroxyapatite-coated or roughened surface condition, threaded implant does offer an advantage when the bone density is D4 at implant insertion. Additional time is then allowed until the Stage II implant uncovery is performed and the progressive loading started.

The time interval for stage II uncovery and prosthetic procedures after implant insertion is dependent on the density of bone at the reentry implant placement. The crest of the ridge and the original antral floor may be the only cortical bone in the region for implant fixation. The most common bone density observed is D4, and often it is softer than the region in general.

WWW.HIGHDENT.IR

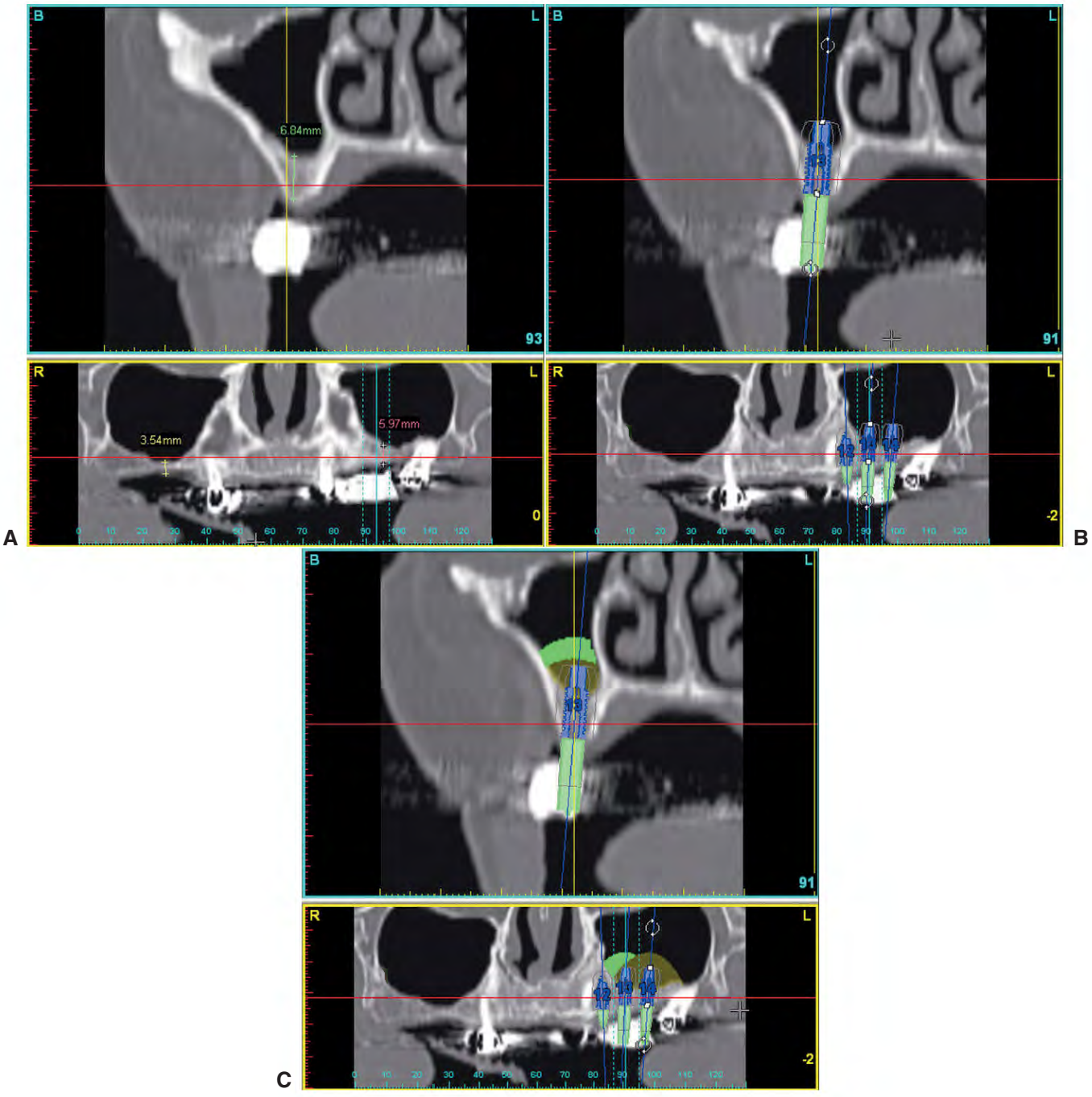


Figure 18-16 **A,** An SA-3 posterior maxilla exhibiting only 5 to 10 mm below the floor of the maxillary sinus. **B,** The implant length required in poor-quality bone such as the posterior maxilla is 12 mm or greater. Therefore a sinus graft to obtain the necessary bone height will be performed before implant placement. **C,** The amount of bone graft necessary can be assessed on computed tomography imaging assisted with a computerized surgery navigation software. *(Courtesy S. Caldwell, El Paso, Tex.)*

Progressive loading after uncovery is most important when the bone is particularly soft and less dense. Inadequate bone formation after the sinus graft healing period of SA-4 surgery is a possible, but uncommon, complication. When observed, the SA-3 technique may be used to place additional subantral graft before the implant placement surgery 4 months later.

The width of the host site for sinus grafts is most often Division A; however, when Division C–w to B exists, an onlay graft for width is indicated. When the graft cannot be secured to the host bone, it is often preferable to perform the sinus graft 6 to 9 months previous to the autogenous graft for width. Then after graft maturation, the implants may be inserted.

SUMMARY

In the past, the posterior maxilla has been reported as the least predictable area for implant survival. Causes cited

WWW.HIGHDENT.IR

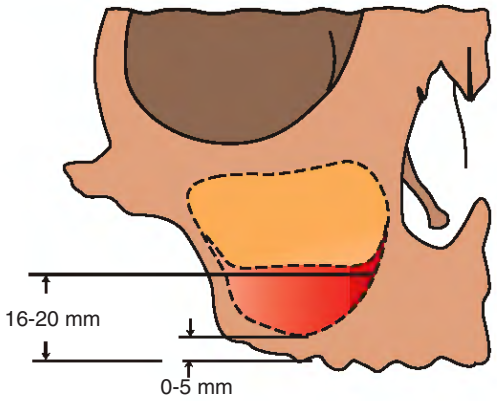


Figure 18-17 A posterior maxilla with 0 to 5 mm of bone below the antrum is subantral augmentation option 4. A Tatum sinus graft is placed and allowed to mature before implant placement. After the sinus graft, width of bone 5 mm or greater is placed into Division A, and 2.5 to 5 mm is placed into Division B and modified into Division A before implant placement.

include inadequate bone height, poor bone density, and high occlusal forces. Past implant modalities attempted to avoid this region, with approaches such as excessive cantilevers when posterior implants are not inserted or excess numbers of pontics when implants are placed posterior to the antrum.

The maxillary sinus may be elevated and subantral bone regenerated to improve implant height. Tatum began to develop these techniques as early as the mid 1970s. Misch developed four options for treatment of the posterior maxilla in 1987 based on the height of bone between the floor of the antrum and the crest of the residual bone. These options were further modified to reflect the width of available bone after adequate height was obtained. Root form implants are indicated under these conditions. When the ridge anatomy is too

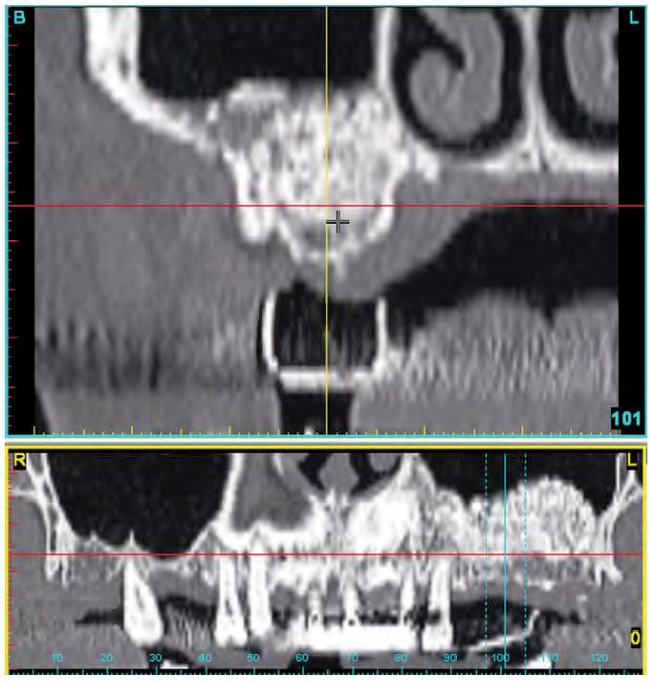
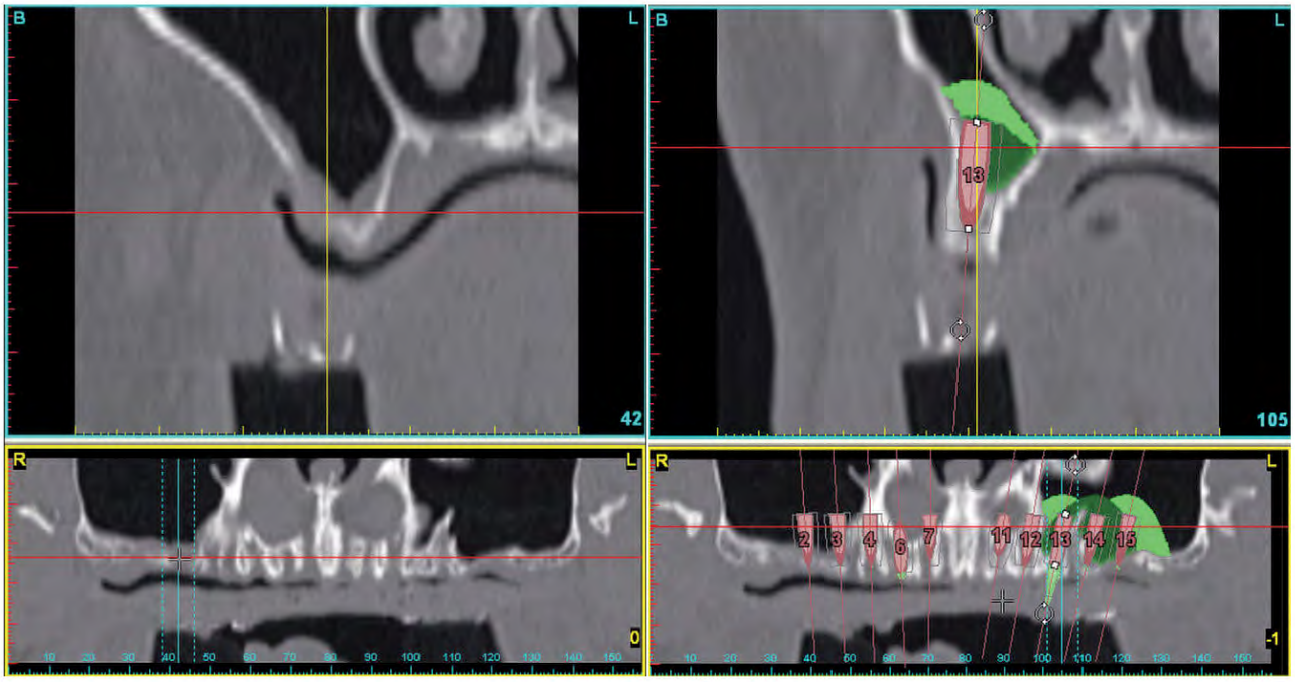


Figure 18-19 Computed tomography (CT) scans can be used after the sinus graft (SA-4) before implant placement to verify proper graft placement. (The medial wall was elevated to permit proper bone graft material placement.) This CT scan image illlustrates the difference in bone height between an SA-4 site before and after grafting. *(Courtesy S. Caldwell, El Paso, Tex.)*



A B

Figure 18-18 **A,** When 0 to 5 mm of bone is available between the crest of the ridge and the floor of the antrum, a sinus graft will be necessary to restore the bone volume prior to implant placement. **B,** The use of three-dimensional imaging and implant software allows the visualization of implants at the treatment planning phase. *(Courtesy S. Caldwell, El Paso, Tex.)*

WWW.HIGHDENT.IR

narrow in width for root form implants, these ridges may be treated by osteoplasty or with autogenous grafts. The higher forces and less dense bone often require larger-diameter implants.

References

1. Marcus SE, Drury JF, Brown LS et al: Tooth retention and tooth loss in the permanent dentition of adults: United States, 1988-1991, *J Dent Res* 75(special issue):684-695, 1996.
2. Meskin LH, Brown LJ: Prevalence and patterns of tooth loss in the U.S. employed adult and senior population, *J Dent Educ* 52:686-691, 1988.
3. Misch CE: Treatment planning for edentulous maxillary posterior region. In Misch CE, editor: *Contemporary implant dentistry*, St Louis, 1993, Mosby.
4. Tatum OH: *Maxillary subantral grafting*. Lecture presented at Alabama Implant Study Group, 1977.
5. Tatum OH: Maxillary and sinus implant reconstruction, *Dent Clin North Am* 30:207-229, 1986.
6. Boyne PJ, James RA: Grafting of the maxillary sinus floor with autogenous marrow and bone, *J Oral Surg* 38:613-616, 1980.
7. Misch CE: Maxillary sinus augmentation for endosteal implants: organized alternative treatment plans, *Int J Oral Implant* 4:49-58, 1987.
8. Smiler DG, Holmes RE: Sinus lift procedure using porous hydroxylapatite: a preliminary clinical report, *J Oral Implantol* 13:2-14, 1987.
9. Chanavaz M: Maxillary sinus: anatomy, physiology, surgery and bone grafting relating to implantology—eleven years of clinical experience, *J Oral Implantol* 16:199-209, 1990.
10. Tidwell JK, Blijdorp PA, Stoelinga PJW et al: Composite grafting of the maxillary sinus for placement of endosteal implants, *Int J Oral Maxillofac Surg* 21:204-209, 1992.
11. Smiler DG, Johnson PW, Lozada JL et al: Sinus lift grafts and endosseous implants: treatment of the atrophic posterior maxilla, *Dental Clin North Am* 36:151-186, 1992.
12. Jensen J, Sindet-Petersen S, Oliver AJ: Varying treatment strategies for reconstruction of maxillary atrophy with implants: results in 98 patients, J *Oral Maxillofac Surg* 52:210-216, 1994.
13. Chiapasco M, Ronchi P: Sinus lift and endosseous implants: preliminary surgical and prosthetic results, *Eur J Prosthodont Rest Dent* 3:15-21, 1994.
14. Blomqvist JE, Alberius P, Isaksson S: Retrospective analysis of one-stage maxillary sinus augmentation with endosseous implants, *Int J Oral Maxillofac Implants* 11: 512-521, 1996.
15. Jensen OT, Shulman LB, Block MS et al: Report of the Sinus Consensus Conference of 1996, *Int J Oral Maxillofac Implants* 13(suppl):11-45, 1998.
16. Valentini P, Abensur DJ: Maxillary sinus grafting with anorganic bovine bone: a clinical report of long-term results, *Int J Oral Maxillofac Impl* 18:556-560, 2003.
17. Lozada JL, Emanuelli S, James RA et al: Root form implants in subantral grafted sites, *J Cal Dent Assoc* 21:31-35, 1993.
18. Wallace SS, Froum SJ: Effect of maxillary sinus augmentation on the survival of endosseous dental implants. A systematic review, *Ann Periodontol* 8:328-343, 2003.
19. Del Fabbro M, Testori T, Francetti L et al: Systematic review of survival rates for implants placed in grafted maxillary sinus, *Int J Periodont Restorative Dent* 24:565-577, 2004.
20. Peleg M, Garg AK, Mazor Z: Predictability of simultaneous implant placement in the severely atrophic posterior maxilla: a 9-year longitudinal experience study of 2132 implants placed into 731 human sinus grafts, *Int J Oral Maxillofac Implants* 21:94-102, 2006.
21. Hising P, Bolin A, Branting C: Reconstruction of severely resorbed alveolar ridge crests with dental implants using a bovine bone mineral for augmentation, *Int J Oral Maxillofac Implants* 16:90-97, 2001.
22. Piattelli M, Favero GA, Scarano A et al: Bone reactions to anorganic bovine bone (Bio-Oss) used in sinus augmentation procedures: a histologic long-term report of 20 cases in humans, *Int J Oral Maxillofac Implants* 14: 835-840, 1999.
23. Valentini P, Abensur D, Wenz B et al: Sinus grafting with porous bone mineral (Bio-Oss) for implant placement: a 5-year study on 15 patients, *Int J Periodontics Restorative Dent* 20:245-253, 2000.
24. Velich N, Nemeth Z, Toth C et al: Long-term results with different bone substitutes used for sinus floor elevation, *J Craniofac Surg* 15:38-41, 2004.
25. Fugazzotto PA, Vlassis J: Long-term success of sinus augmentation using various surgical approaches and grafting materials, *Int J Oral Maxillofac Implant* 13:52-58, 1998.
26. Hallman M, Sennerby L, Lundgren S: A clinical and histologic evaluation of implant integration in the posterior maxilla after sinus floor augmentation with autogenous bone, bovine hydroxyapatite, or a 20:80 mixture, *Int J Oral Maxillo Implants* 17:635-643, 2002.
27. Rodoni LR, Glauser R, Feloutzis A et al: Implants in the posterior maxilla: a comparative clinical and radiologic study, *Int J Oral Maxillofac Implants* 20:231-237, 2005.
28. Maiorana C, Sigurta D, Mirandola A et al: Bone resorption around dental implants placed in grafted sinuses: clinical and radiologic follow-up to 4 years, *Int J Oral Maxillofac Implants* 20:261-266, 2005.
29. Chanavaz M: Sinus grafting related to implantology statistical analysis of 15 years of surgical experience, 1979-1994, *Oral Implantol* 22:119-130, 1996.
30. Aghaloo TL, Moy PK: Which hard tissue augmentation techniques are the most succesful in furnishing bony support for implant placement? *Int J Maxillofac Implants* 22(suppl):49, 2007.
31. Pietrokovski J: The bony residual ridge in man, *J Prosthet Dent* 34:456-462, 1975.
32. Misch CE: Bone character: second vital implant criterion, *Dent Today* 7:39-40, 1988.
33. Goodacre JC, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 2:121-132, 2003.
34. Misch CE, Qu Z, Bidez MW: Mechanical properties of trabecular bone in the human mandible: implications for dental implant treatment planning and surgical placement, *J Oral Maxillofac Surg* 57:700-706, 1999.
35. Blitzer A, Lawson W, Friedman WH, editors: *Surgery of the paranasal sinuses*, Philadelphia, 1985, WB Saunders.
36. Lang J, editor: *Clinical anatomy of the nose, nasal cavity and paranasal sinuses*, New York, 1989, Thieme.
37. Anon JB, Rontal M, Zinreich SJ: *Anatomy of the paranasal sinuses*, New York, 1996, Thieme.
38. Stammberger H: History of rhinology: anatomy of the paranasal sinuses, *Rhinology* 27:197-210, 1989.
39. Takahashi R: The formation of the human paranasal sinuses, *Acta Otolaryngol* 408:1-28, 1984.

WWW.HIGHDENT.IR

40. Karmody CS, Carter B, Vincent ME: Developmental anomalies of the maxillary sinus, *Trans Am Acad Ophthalmol Otol* 84: 723-728, 1977.
41. Fanuscu MI, Iida K, Caputo AA et al: Load transfer by an implant in a sinus-grafted maxillary model, I*nt J Oral Maxillofac Implants* 18:667-674, 2003.
42. Herzberg R, Doley E, Schwartz-Arad D: Implant marginal bone loss in maxillary sinus grafts, *Int J Oral Maxillofac Implants* 21:103-110, 2006.
43. Trisi P, Marcato C, Todisco M: Bone-to-implant apposition with machined and MTX microtextured implant surfaces in human sinus graft, *Int J Periodont Restorative Dent* 23: 427-37, 2003.
44. Cochran DL, Schenk RK, Lussi A et al: Bone response to unloaded and loaded titanium implants with sandblasted and acid-etched surface: a histometric study in the canine mandible, *J Biomet Mater Res* 40:1-11, 1998.
45. Xie J, Baumann MJ, McCabe LE: Osteoblasts respond to hydroxyapatite surfaces with immediate changes in gene expression, *J Biomet Mater Res* 1:71:108-117, 2004.
46. Linkow LI: Tuber blades, *J Oral Implant* 9:190-216, 1980.
47. Tulasne JF: Implant treatment of missing posterior dentition. In Albrektsson T, Zarb G, editors: *The Branemark osseointegrated implant*, Chicago, 1989, Quintessence.
48. Ashkinazy LR: Tomography in implantology, *J Oral Implant* 10:100-118, 1982.
49. Geiger S, Pesch HJ: Animal experimental studies of the healing around ceramic implants in bone lesions in the maxillary sinus region, *Deutsche Zahnarztl Z* 32:396-399, 1977.
50. Branemark PI, Adell R, Albrektsson T et al: An experimental and clinical study of osseointegrated implants penetrating the nasal cavity, *J Oral Maxillofac Surg* 42:497-505, 1984.
51. Linkow LI: Maxillary pterygoid extension implants: the state of the art, *Dent Clin North Am* 24:535-551, 1980.
52. Cranin AN, Satler N, Shpuntoff R: The unilateral pterygohamular subperiosteal implant evolution of a technique, *J Am Dent Assoc* 110:496-500, 1985.
53. Keller EE, van Roekel NB, Desjardins RR et al: Prosthetic surgical reconstruction of severely resorbed maxilla with iliac bone grafting and tissue integrated prostheses, *Int J Oral Maxillofac Impl* 2:155, 1987.
54. Sailer HF: A new method of inserting endosseous implants in totally atrophic maxillae, *J Cranio Maxillofac Surg* 17: 299-305, 1989.
55. Linkow LI: *Maxillary implants: a dynamic approach to oral implantology,* North Haven, Conn, 1977, Glarus.
56. Tatum OH: Omni Implant Systems, S Series Implants, St Petersburg, Fla, 1981 Omni.
57. Tatum OH: The Omni implant system. In Hardin JF, editor: *Clarke's clinical dentistry*, Philadelphia, 1984, Lippincott.
58. Feigel A, Maker M: The significance of sinus elevation for blade implantology: report of an autopsy case, *J Oral Implant* 15:232-247, 1989.
59. Jensen J, Simonsen EK, Sindet-Pedersen S: Reconstruction of the severely resorbed maxilla with bone grafting and osseointegrated implants: a preliminary report, *J Oral Maxillofac Surg* 48:27-32, 1990.
60. Wood RM, Moore DL: Grafting of the maxillary sinus with intraorally harvested autogenous bone prior to implant placement, *Int J Oral Maxillofac Impl* 3:209-214, 1988.
61. Raghoebar GM, Brouwer TJ, Reintsema H et al: Augmentation of the maxillary sinus floor with autogenous bone for the placement of endosseous implants: a preliminary report, *J Oral Maxillofac Surg* 51:1198-1203, 1993.
62. Tatum OH, Lebowitz MS, Tatum CA et al: Sinus augmentation: rationale, development, long term results, *N Y St Dent J* 59:43-48, 1993.
63. Misch CE: Treatment plans for implant dentistry, *Dent Today* 12:56-61, 1993.
64. Misch CE: Maxillary posterior treatment plans for implant dentistry, *Implantodontie* 19:7-24, 1995.
65. Stoler A: *The CAT-scan subperiosteal implant,* Hong Kong, 1986, International Congress of Oral Implantologist World Meeting.
66. Boyne PT: Analysis of performance of root form endosseous implants placed in the maxillary sinus, *J Long Term Effects Med Implants* 3:143-159, 1993.
67. Small SA, Zinner ID, Panno FV et al: Augmenting the maxillary sinus for implants, report of 27 patients, *Int J Oral Maxillofac Impl* 8:523, 1993.
68. Gonzalez-Garcia R, Naval-Gias L, Munoz-Guerra MF et al: Preprosthetic and implantological surgery in patients with severe maxillary atrophy, *Med Oral Patol Oral Cir Bucal* 10:343-354, 2005.
69. Merkx, MA, Maltha JC, Steolinga PJ: Assessment of the value of anorganic bone additives in sinus floor augmentation: a review of clinical reports, *Int J Oral Maxillofac Surg* 32:1-6, 2003.
70. Tong DC, Rioux K, Drangsholt M et al: A review of survival rates for implants placed in grafted maxillary sinuses using meta-analysis, *Int J Oral Maxillofac Implants* 13:175-182, 1998.
71. Hallman M, Sennerby I, Zetterqvist L et al: A 3-year prospective follow-up study of implant-supported fixed prostheses in patients subjected to maxillary sinus floor augmentation with a 80:20 mixture of deproteinized bovine bone and autogenous bone: clinical, radiographic and resonance frequence analysis, *Int J Oral Maxillofac Surg* 34:273-280, 2005.
72. Chiapasco M: Tecniche ricostruttive con innesti e/o osteotomie. In: Chiapasco M, Romeo E, editors: *Riabilitazione implanto-protesica dei casi complessi*, Torino, Italy, 2003, UTET ed.
73. Loukota RA, Isaksson SG, Linner EL et al: A technique for inserting endosseous implants in the atrophic maxilla in a single stage procedure, *Br J Oral Maxillofac Surg* 30:46-49, 1992.
74. Jensen OT, Greer R: Immediate placement of osseointegrated implants into the maxillary sinus augmented with mineralized cancellous allograft and Gore-Tex, 2nd stage surgical and histological findings. In Laney WR, Tolman DE, editors: *Oral orthopedic and maxillofacial reconstruction*, Chicago, 1992, Quintessence.
75. Hirsch JM, Ericsson I: Maxillary sinus augmentation using mandibular bone grafts and simultaneous installation of implants—a surgical technique, *Clin Oral Impl Res* 2: 91-96, 1991.
76. Hurzeler MB, Kirsch A, Ackermann KL et al: Reconstruction of the severely resorbed maxilla with dental implants in the augmented maxillary sinus—a 5 year clinical investigation, *Int J Oral Maxillofac Impl* 11: 466-475, 1996.
77. Chiapasco M, Romeo E, Vogel G: Tridimensional reconstruction of knife-edge edentulous maxillae by sinus elevation, onlay grafts, and sagittal osteotomy of the anterior maxilla: preliminary surgical and prosthetic results, *Int J Oral Maxillofac Implants* 13:394-399, 1998.
78. Widmark G, Andersson B, Andrup B et al: Rehabilitation of patients with severely resorbed maxillae by means of

WWW.HIGHDENT.IR

implants with or without bone grafts: a 1-year follow-up study, *Int J Oral Maxillofac Implants* 13:474-482, 1998.
79. Yildirin M, Spiekermann H, Biesterfeld S et al: Maxillary sinus augmentation using xenogenic bone substitute material Bio-Oss in combination with venous blood. A histologic and histomorphometric study in humans, *Clin Oral Implants Res* 11:217-229, 2000.
80. Misch CE, Judy KW: Classification of the partially edentulous arches for implant dentistry, *Int J Oral Implant* 4: 7-12, 1987.
81. Cawood JI, Howell R: A classifications for the edentulous jaws, *Int J Oral Maxillofac Surg* 17:232-236, 1998.
82. Jensen O: *The sinus bone graft,* Carol Stream, Ill, 1999, Quintessence.
83. Misch CE, Chiapasco M: Identification for and classification of sinus bone grafts. In Jensen O, editor: *The sinus bone graft,* ed 2, Carol Stream, Ill, 2006, Quintessence.
84. Misch CE, Dietsh F: Bone grafting materials in implant dentistry, *Impl Dent* 2:158-167, 1993.
85. Summers RB: Maxillary implant surgery: the osteotome technique, *Compend Cont Educ Dent* 15:152-162, 1994.
86. Zaninari A: Rialzo del Seno Mascellare Prima parte, *Tam Tam Dentale* 2:8-12, 1990.
87. Worth HM, Stoneman DW: Radiographic interpretation of antral mucosal changes due to localized dental infection, *J Can Dent Assoc* 38:111, 1972.
88. Nystrom E, Lundgren S, Gunne J et al: Interpositional bone grafting and Le Fort I osteotomy for the reconstruction of the atrophic edentulous maxilla: a two-stage technique, *Int J Oral Maxillofac Surg* 26:423-427, 1997.
89. Lundgren S, Nystrom E, Nilem H et al: Bone grafting to the maxillary sinuses, nasal floor and anterior maxilla in the atrophic edentulous maxilla: a two-stage technique, *Int J Oral Maxillofac Surg* 26:428-434, 1997.
90. Triplett RG, Schow SR: Autologous bone grafts and endosseous implants: complementary techniques, *J Oral Maxillofac Surg* 54:486-494, 1996.
91. Misch CE, Dietsh F: Endosteal implants and iliac crest grafts to restore severely resorbed totally edentulous maxillae, a retrospective study, *J Oral Implantol* 20:100-110, 1994.

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Chapter 19

Treatment Plans for Partially and Completely Edentulous Arches in Implant Dentistry

Carl E. Misch

PARTIALLY EDENTULOUS ARCHES

To organize treatment plans in a consistent approach, a classification of patient conditions is necessary. Because more than 65,000 possible combinations of teeth and edentulous spaces exist in a single arch, no universal agreement exists regarding the use of any one classification system. Numerous classifications have been proposed for partially edentulous arches. Their use allows the profession to visualize and communicate the relationship of hard and soft structures. This chapter reviews a classification for diagnosis and treatment planning for patients who are partially or completely edentulous and require implant prostheses. By using this classification, which the author first presented in 1985, the doctor is able to convey the dimensions of the bone available in the edentulous area and also indicate the strategic position of the segment to be restored.[15]

HISTORY

Cummer,[1] Kennedy,[2] and Bailyn[3] originally proposed the classifications of partially edentulous arches that are most familiar to the profession. These classifications were developed to organize removable partial denture (RPD) designs and concepts. Other classifications have also been proposed[4-14] (including one by the American College of Prosthodontists), none of which has been universally accepted. The Kennedy classification, however, has been taught in most American dental schools.

The Kennedy classification divides partially edentulous arches into four classes.[2] Class I has bilateral posterior edentulous spaces, Class II has a unilateral posterior edentulous space, Class III has an intradental edentulous area, and Class IV has an anterior edentulous area that crosses the midline.

The Kennedy classification is difficult to use in many situations without certain qualifying rules. The eight Applegate rules are used to help clarify the system.[11] They may be summarized in three general principles. The first principle is that the classification should include only natural teeth involved in the final prosthesis and follow rather than precede any extractions of teeth that might alter the original classification. This concept, for example, considers whether second or third molars are to be replaced in the final restoration. The second rule is that the most posterior edentulous area always determines the classification. The third principle is that edentulous areas, other than those determining the classification, are referred to as *modifications* and are designated only by their number. The extent of the modification is not considered.

CLASSIFICATION OF PARTIALLY EDENTULOUS ARCHES

The implant dentistry bone volume classification developed by Misch and Judy in 1985 may be used to build on the four classes of partial edentulism described in the Kennedy-Applegate system.[15,16] This facilitates communication of teeth positions and the primary edentulous sites among the large segment of practitioners already familiar with this classification, and it enables the use of common treatment methods and principles established for each class. The implant dentistry classification for partially edentulous patients by Misch and Judy also includes the same four available bone volume divisions previously presented for edentulous areas. Other intradental edentulous regions that are not responsible for the Kennedy-Applegate class determination are not specified within the available bone section, should implants not be considered in the modification region. However, if the modification segment is also included in the treatment plan, then it is listed, followed by the available bone division it characterizes (Box 19-1).

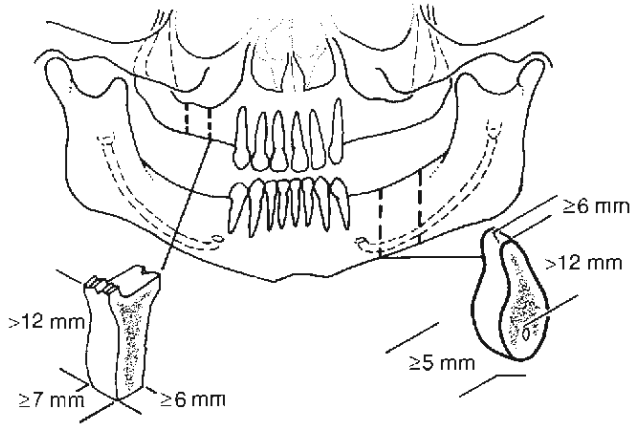
WWW.HIGHDENT.IR

Box 19-1 Implant Dentistry Classification of Partially Edentulous Arches

Class I: Partially Edentulous Arch with Bilateral Edentulous Areas Posterior to Remaining Natural Teeth

Division A

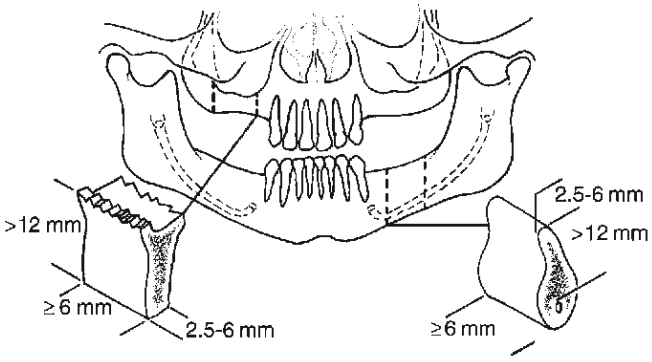
1. Edentulous areas have abundant bone width (>6 mm), height (>12 mm), and length (>7 mm) for endosteal implant(s).



2. Direction of load is within 30 degrees of implant body axis.
3. Crown height <15 mm.
4. Root form implants and independent prostheses often are indicated.

Division B

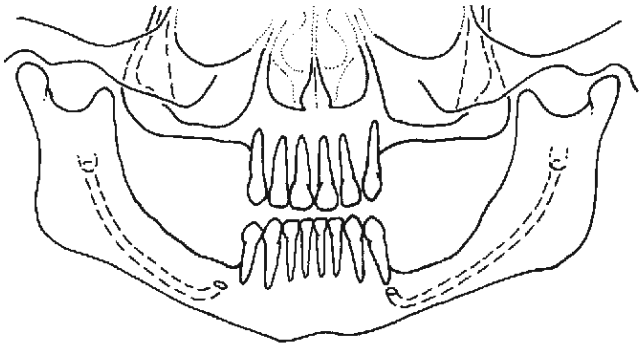
1. Edentulous areas have moderate available bone width (2.5 to 6 mm) and at least adequate bone height (>12 mm) and length (>6 mm).



2. Direction of load is within 30 degrees of implant body axis.
3. Crown height <15 mm.
4. Surgical options include osteoplasty, small-diameter implants, and/or augmentation.

Division C

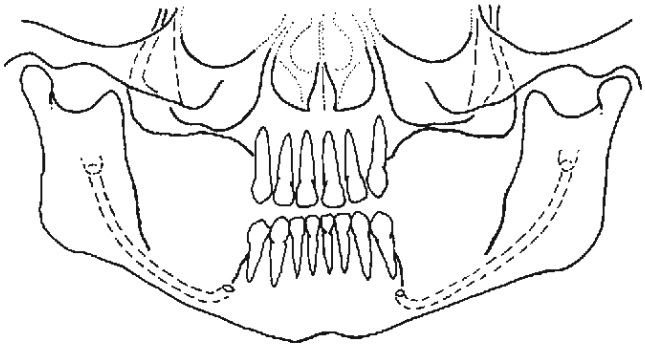
1. Edentulous areas have inadequate available bone for endosteal implant (or implants) with a predictable result, because of too little bone width (C–w), length, height (C–h), or angulation of load.



2. Crown height >15 mm.
3. Surgical options for C–w include osteoplasty or augmentation; for C–h, options include subperiosteal or disc implants or augmentation.
4. Root forms may be considered with augmentation and/or nerve repositioning.

Division D

1. Edentulous areas have severely resorbed ridges, involving a portion of the basal or cortical supporting bone.



2. Crown height >20 mm.
3. Surgical options usually require augmentation before implants can be inserted.

Note: If the bilateral edentulous areas are not within the same division, then the right side is described first (e.g., Class I, Division A, B).

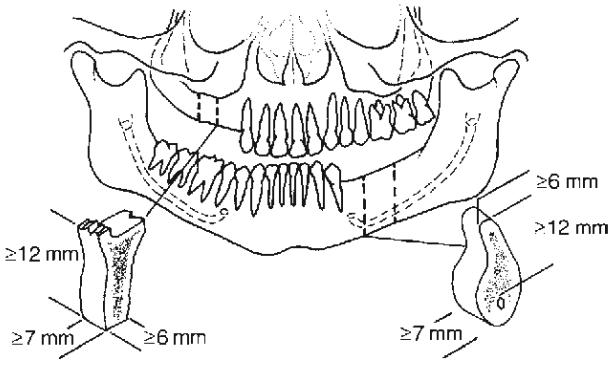
Continued

WWW.HIGHDENT.IR

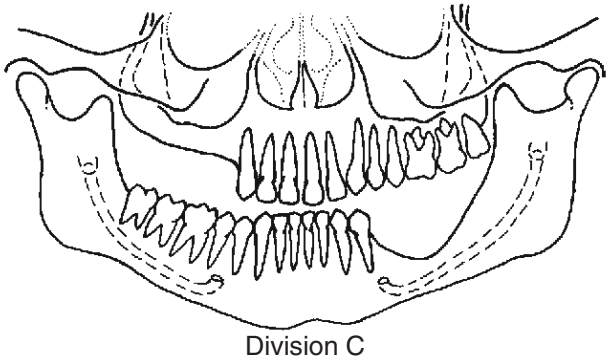
Box 19-1 Implant Dentistry Classification of Partially Edentulous Arches—cont'd

Class II: Partially Edentulous Arch with Unilateral Edentulous Area Posterior to Remaining Teeth

Divisions A to D (Same as for Class I)



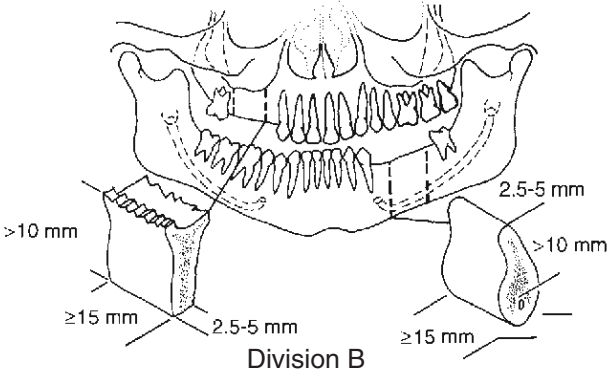
Division A



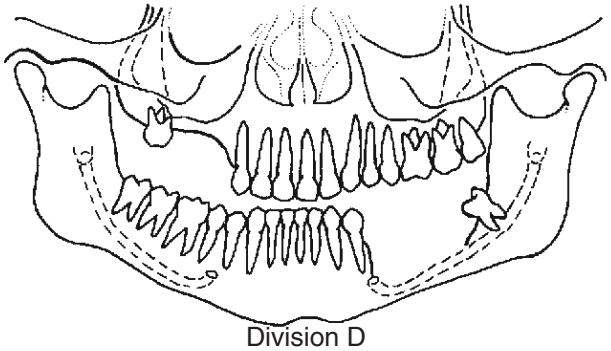
Division C

Class III: Partially Edentulous Arch with Unilateral Edentulous Area with Natural Teeth Remaining Anterior and Posterior

Divisions A to D (Same as for Class I)



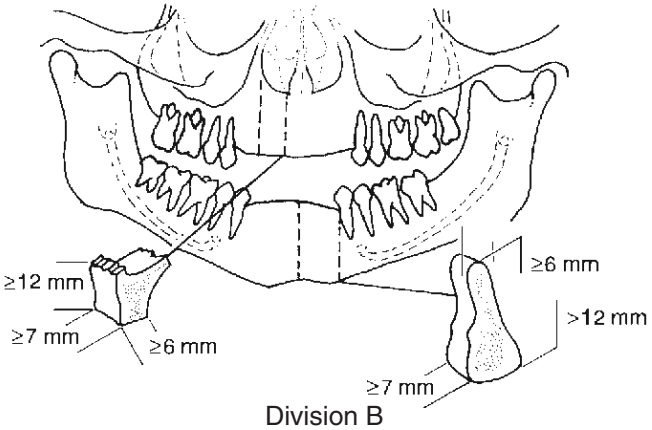
Division B



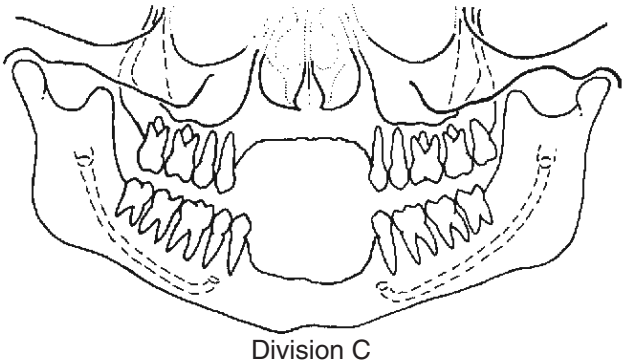
Division D

Class IV: Partially Edentulous Arch with Edentulous Area Anterior to Remaining Natural Teeth and Crossing the Midline

Divisions A to D (Same as for Class I)



Division B



Division C

WWW.HIGHDENT.IR

Treatment Planning: Class I

In Class I patients, distal edentulous segments are bilateral and natural anterior teeth are present. The majority of these arches are only missing molars, and almost all have retained at least the anterior incisors and canines.[17] Therefore, once restored to proper occlusal vertical dimension, the natural anterior teeth contribute to the distribution of forces throughout the mouth in centric relation occlusion. More importantly, when opposing natural teeth or in fixed implant prosthesis, they also permit excursions during mandibular movement to disclude the posterior implant-supported prostheses and protect them from lateral forces. However, many of these mandibular Class I patients oppose a maxillary denture, in which case bilateral balance is more appropriate.

The Class I patient is more likely to wear a RPD than Class II or III patients because mastication and/or support of an opposing removable prosthesis is more difficult when not wearing a mandibular prosthesis. The posterior soft tissue–supported Class I partial dentures are designed to either primary load the edentulous regions or the natural anterior teeth. The clasp design, which places less force on the tooth (e.g., bar clasp including t, y rpi), will place more force on the bone. The RPDs, which place more force on the abutment teeth (e.g., precision partial dentures), will place less force on the bone. In either case, the removable prosthesis often accelerates the posterior bone loss. In addition, a partial denture that is not well designed or maintained distributes additional loads to abutment teeth and may even contribute to poor periodontal health. The combinations of these conditions lead to bone loss in the edentulous regions and poorer adjacent natural abutments.[18-20] As a result, it is this author's observation that long-term Class I patients who have been wearing an RPD often exhibit Division C ridges and mobile abutment teeth.

Class I patients often have mobile anterior teeth, because long-term lack of bilateral posterior support caused by the wearing of a poorly fitting RPD, or none at all, has resulted in an overload to the remaining dentition. Therefore these patients often require a posterior implant prosthesis to be independent from the mobile natural teeth. In addition, the occlusal scheme must accommodate the specific conditions of mobile anterior teeth. This requires increased implant support in the posterior segments when compared with most Class II or III patients, as well as greater attention and frequency for occlusal adjustments.

The treatment plan must consider the factors of force previously identified and relate them to the existing bilateral edentulous condition. Osteoplasty cannot be as aggressive in the Class I patient to increase bone width, compared with the Class IV or fully edentulous patient with implants primarily in the anterior regions, because of the opposing anatomical landmarks (maxillary sinus or mandibular canal). Augmentation procedures are often required to improve posterior bone volume, increase the implant surface area, and permit the fabrication of an independent implant restoration.

Financial concerns may require the staging of treatment over years. The posterior region with the greatest volume of bone usually is restored first, if no bone grafting is required. In this manner, implants of greater size and surface area can resist the unilateral posterior forces while the patient awaits future treatment. If many years pass before implants are to be inserted in the lesser available bone, then continued resorption may require augmentation before reconstruction.

If both posterior segments require bone grafting, the patient is encouraged to have both posterior segments augmented at the same time. In this way, the autologous portion of the graft may be harvested and distributed to both posterior regions, decreasing the number of surgical episodes for the patient.

Division A Treatment Plans

When patients are placed in a Class I, Division A category, an independent implant-supported fixed prosthesis is usually indicated. Two or more endosteal root form implants are required to replace molars with independent prostheses. The greater the number of teeth missing, the larger the size and/or number of implants required. Posterior available bone is limited in height by the mandibular canal in the mandible or the maxillary sinus in the maxilla. The first premolar-positioned implants must avoid encroachment on the apex of the canine root and yet avoid the anterior loop of the mandibular canal or maxillary sinus.

Division B Treatment Plans

Class I, Division B patients have narrow bone in posterior edentulous spaces and anterior natural teeth. A fixed prosthesis is also indicated in these categories. Available bone height is restricted by the mandibular canal or maxillary sinus. Therefore osteoplasty to increase bone width has limited applications. Endosteal small-diameter root form implants may be placed in the mandibular posterior Division B edentulous ridge. If narrow-diameter root forms are used, then a greater number than for the Division A ridge is indicated, and the use of one implant for every missing tooth root with no cantilever is recommended.

The patient missing molars and both premolars requires additional implant support. Four Division B root forms may be the foundation of an independent fixed partial denture (FPD) in the mandible , depending on the other stress factors. If stress factors are too great (as a result of parafunction) or bone density is poor (as in the maxilla), then the Division B bone should be augmented to Division A before larger-diameter implant insertion. The anterior teeth in Class I patients

WWW.HIGHDENT.IR

should provide disclusion of the posterior implants during all excursions when opposing natural teeth or a fixed prosthesis. Molar endosteal implants should not be rigidly cross-splinted to each other in the Class I patient. Flexure of the mandible during opening may cause a rigid splint to exert lateral forces on the posterior implants. Therefore independent restorations are indicated.

Division C Treatment Plans

When inadequate bone exists in height, width, length, or angulation, or if crown/implant ratios are equal to or greater than 1, the practitioner must consider several options. The first treatment option is to not use implant support, but rather to orient the patient toward a conventional removable partial prosthesis. However, although this condition is easiest to treat with a traditional soft tissue–borne restoration, bone loss will continue and can eventually compromise any restorative modality.

The second option is to use bone augmentation procedures. If the intent of the bone graft is to change a Division C to a Division A or B for endosteal implants, then at least some autogenous bone is indicated. Augmentation is used most often in the Class I maxilla, where sinus grafts with a combination of allografts and autogenous bone are a predictable modality. Implants may be placed after the graft has created a Division A ridge, and the treatment plan follows the options previously addressed.

In the mandible, the third option for the Class I, Division C patient is to place unilateral subperiosteal implants or disc implants above the canal (Figure 19-1). The disc implants support independent posterior fixed prostheses bilaterally.

The fourth treatment option in the mandible is nerve repositioning and endosteal implants in Class I patients who are poor candidates for bone augmentation or subperiosteal implants. Risks of long-term paresthesia exist that may include hyperesthesia and pain. Reports in the literature concern dysesthesia and fracture of the severely atrophic mandible.[21,22] In addition, the gain of height in the C–h mandible may only permit the placement of implants 10 mm high, still insufficient to compensate for the increased crown height and resultant unfavorable crown/implant ratio.

Division D Treatment Plans

Class I, Division D usually occurs most often in the long-term edentulous maxilla. A sinus graft is usually performed before implant placement. Class I, Division D ridges are rarely found in the mandibular partially edentulous patient. When observed, the most common causes are from trauma or surgical excision of neoplasms. These patients often need autogenous bone onlay grafts to improve implant success and prevent pathologic fracture before prosthodontic reconstruction. After the graft is mature and the available bone improved, the patient is evaluated and treated in a manner similar to other patients with favorable bone volume.

Treatment Planning: Class II

Kennedy-Applegate Class II partially edentulous patients are missing teeth in one posterior segment (see Box 19-1). These patients are often able to function without a removable restoration and are less likely to tolerate or overcome the minor complications of wearing the prosthesis. As a result, they are not as likely to wear a removable restoration. The available bone is therefore often adequate for endosteal implants, even when long-term edentulism has been present. However, the local bone density may be decreased. Endosteal implants with minimum osteoplasty are a common modality in these patients, who are more often Class II, Division A or B types.[23,24]

Because the patient is less likely to wear the RPD, the opposing natural teeth have often extruded into the posterior edentulous region. The occlusal plane and tipped or extruded teeth should be closely evaluated and restored as indicated to provide a favorable environment in terms of occlusion and forces distribution. It is not unusual to require extraction of the second molar, endodontics, crown lengthening and a crown of the first molar, and enameloplasty for the second premolar.

Division A Treatment Plans

When patients are placed in a Class II, Division A category, an independent implant-supported fixed prosthesis is usually indicated. Two or more endosteal root form

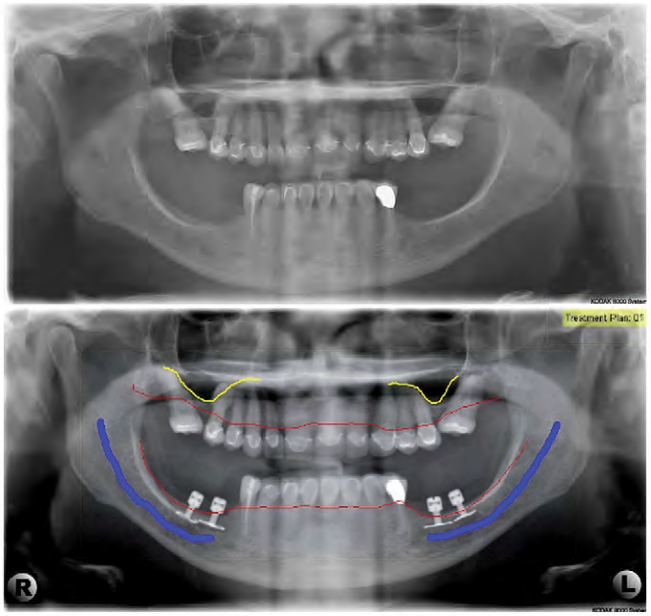


Figure 19-1 In Class I, Division C, options include small-dimension implants such as disc implants, which can be placed in minimal heights of bone above the mandibular canal. Two independent fixed prostheses are supported by implants.

WWW.HIGHDENT.IR

implants are required to replace molars with independent prostheses. The greater the number of teeth missing, the larger the size and/or number of implants required. Posterior available bone is limited in height by the mandibular canal in the mandible or the maxillary sinus in the maxilla. The first premolar-positioned implants must not encroach on the apex of the canine root while still avoiding the anterior loop of the mandibular canal or maxillary sinus (Figure 19-2).

Division B Treatment Plans

Class II, Division B patients have narrow bone in posterior edentulous spaces and anterior natural teeth. A fixed prosthesis is also indicated in these categories. Available bone height is restricted by the mandibular canal or maxillary sinus. Therefore osteoplasty to increase bone width has limited applications. Endosteal small-diameter root form implants may be placed in the mandibular posterior Division B edentulous ridge. If narrow-diameter root forms are used, then a greater number than for the Division A ridge is indicated, and the use of one implant for every missing tooth root with no cantilever is recommended.

The patient missing molars and both premolars requires additional implant support. Four Division B root forms may be the foundation of an independent FPD in the mandible, depending on the other stress factors. If stress factors are too great (as a result of parafunction) or bone density is poor (as in the maxilla), then the Division B bone should be augmented to Division A before larger-diameter implant insertion. The anterior teeth in Class II patients should provide disclusion of the posterior implants during all excursions.

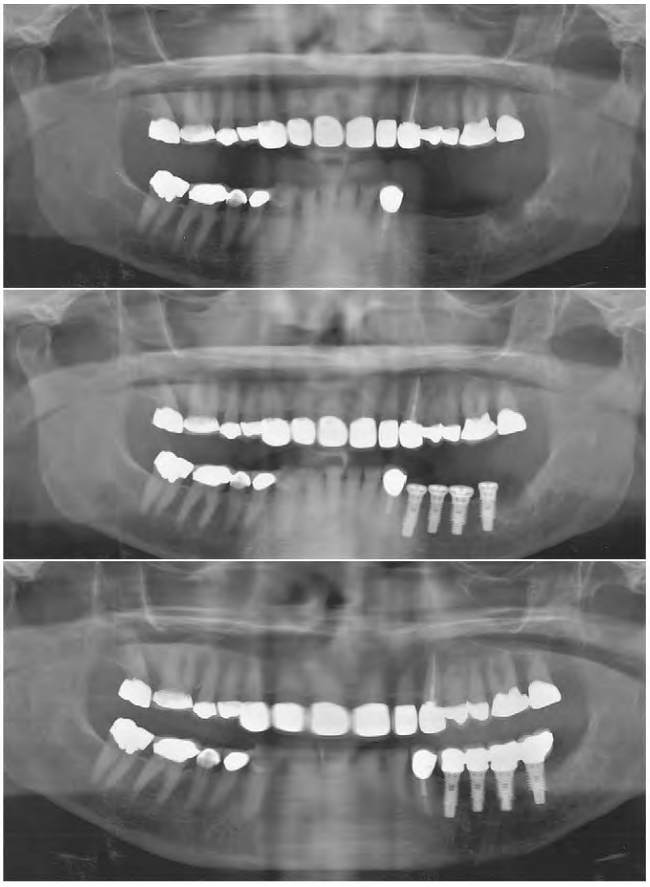


Figure 19-2 **A,** After socket grafting and ridge preservation in the posterior mandible, sufficient bone exists for the area to be classified Division A. **B,** Class II, Division A patients can be treated with endosteal implants (often one implant is treatment planned for each mandibular root). **C,** A final independent fixed cemented restoration is the treatment of choice.

Division C Treatment Plans

When inadequate bone exists in height, width, length, or angulation, or if crown/implant ratios are equal to or greater than 1, then the practitioner must consider several options. In the mandible, the first treatment option is to not use implant support but to consider a posterior cantilevered FPD replacing one premolar-sized crown, using two or three anterior teeth as abutment support. This is the easiest option and is strongly recommended when only molars are missing.

The second option is to use bone augmentation procedures. If the intent of the bone graft is to change a Division C to a Division A or B for endosteal implants in the mandible, then autogenous bone is indicated. Augmentation is used most often in the Class II maxilla as the first choice, where sinus grafts with a combination of allografts and autogenous bone are a predictable modality. Implants may be placed after the graft has created a Division A ridge, and the treatment plan follows the options previously addressed.

The third option for the Division C mandibular patient is to place a Class II unilateral subperiosteal implant or a disc implant above the canal.

The fourth treatment option in the mandible is nerve repositioning and endosteal implants in Class II patients who are poor candidates for bone augmentation. Risks of long-term paresthesia exist that may include hyperesthesia and pain. Reports in the literature concern dysesthesia and fracture of the severely atrophic mandible.[21,22] In addition, the gain of height in the C–h mandible may only permit the placement of implants 10 mm high, which is still insufficient to compensate for the increased crown height and resultant unfavorable crown/implant ratio.

Division D Treatment Plans

Class II, Division D usually occurs most often in the long-term edentulous maxilla. A sinus graft is usually performed before implant placement. Class II, Division D ridges are rarely found in the mandibular partially edentulous patient. When observed, the most common causes are from trauma or surgical excision of neoplasms. These patients often need autogenous bone onlay grafts to improve implant success and prevent pathologic fracture before prosthodontic reconstruction. After the

WWW.HIGHDENT.IR

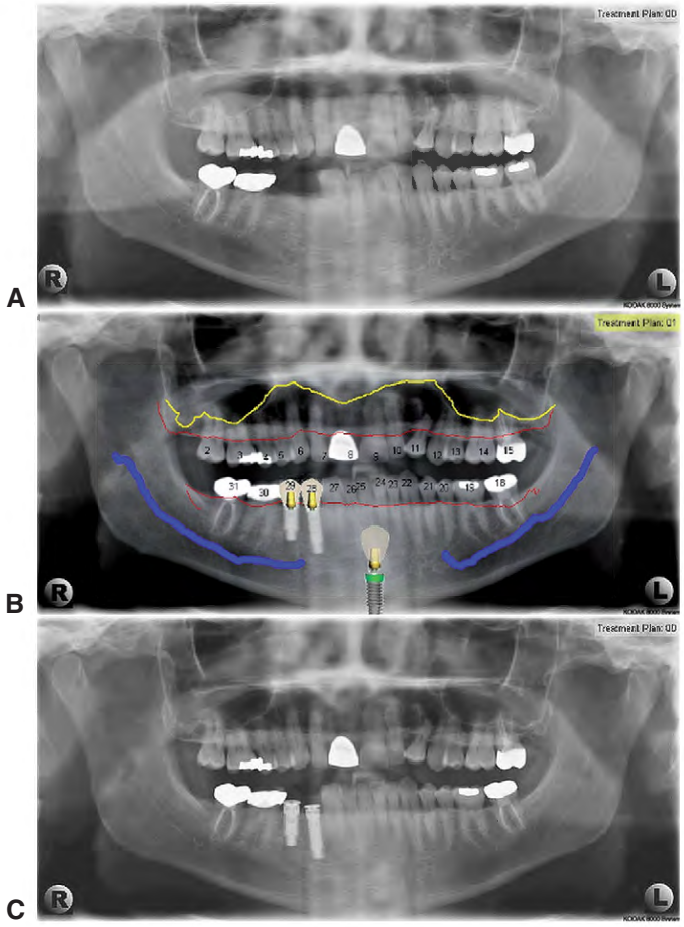


Figure 19-3 **A,** Typically, patients with Class III edentulous segments are missing a single tooth or a posterior space. **B,** The ideal treatment plan can be studied with computer-assisted programs that help visualize proper positioning and the contours of the final restoration (XCPT, Naples, Fla.). **C,** The implant surgical phase replicates the computer-assisted programmed surgery. (**B,** *Courtesy N. Levine, Chicago, Ill.)*

graft is mature and the available bone improved, the patient is evaluated and treated in a manner similar to other patients with favorable bone volume.

Treatment Planning: Class III

Typically, the two most common Class III patients consulting for implants are either missing a single tooth or have a long posterior edentulous span. A multiple-tooth posterior edentulous region most often can be restored as an independent restoration, but it may on occasion need to be joined to a posterior natural abutment. The posterior tooth usually exhibits less mobility compared with the anterior teeth, and disclusion of the posterior segments can be achieved. A review of the literature demonstrates joining implants in teeth in the same prosthesis under those conditions is possible. On the other hand, an anterior tooth abutment exhibits greater mobility and sustains greater lateral forces during excursions and should not be joined to an implant, unless it is a "living pontic" or a part of a restoration using a splinted arch concept to distribute lateral forces.

A single-tooth implant is the treatment of choice when the bone and soft tissues are within normal range before or during implant treatment. Fixed prostheses increase the risk of decay, pulpal involvement, and periodontal disease on the natural abutment teeth.[25] Both the traditional prosthesis and the abutment teeth have a poorer survival rate than implant prostheses. As a result, single-tooth implants are often indicated.[26,27]

Division A Treatment Plans

Class III, Division A patients are good candidates for endosteal root form implant placement in the edentulous space. This allows the restoration of natural teeth to be independent and allows the fabrication of shorter span restorations. It is easier to obtain maximum available height of bone for implant placement anterior to the mandibular foramen or maxillary sinus (Figure 19-3). As a general rule, the final prosthesis should be completely implant supported, and two implants should support each section of three missing tooth roots (not three missing crowns). Mobile natural teeth adjacent to the edentulous span cause greater loads on the implants; therefore one implant for each missing root may be indicated. If the adjacent teeth are mobile, then the implant must support both the missing teeth and mobile teeth during occlusion.

Division B Treatment Plans

In Class III, Division B patients, narrow-diameter endosteal implants may be placed in the mandibular long-span edentulous space. This treatment plan is primarily used for a fixed prosthesis when the span is too long or occlusal forces are too great for the natural abutments to act as sole support for the final prosthesis. The final implant prosthesis should be independent of these teeth.

Division C or Division D Treatment Plans

When Division C is found in Class III patients, the most common treatment plan in the maxilla is bone augmentation before implant insertion and an independent implant prosthesis. Sinus grafting in the posterior Division C ridge is very predictable. In the mandible, Division C bone volume for Class III patients should often consider a traditional fixed prosthesis, because bone grafting for height is less predictable than in the maxilla.

Treatment Planning: Class IV

In the Class IV patient, the anterior edentulous space crosses the midline. In the past, traditional FPDs were often the treatment of choice when the canines were present. Today, an independent implant prosthesis is often warranted. However, a lack of anterior bone volume in the maxilla is common, and bone grafts before

WWW.HIGHDENT.IR

implant placement are typically necessary to prevent the implants from being placed palatally in relation to the natural roots. A cantilever is often created off the implant bodies to place the maxillary incisor edge in proper position for esthetics and speech. The moment force generated is greater than when found in the mandibular counterpart. This, added to other factors, makes the premaxilla one of the more difficult regions of the mouth to treat successfully. As a general rule, one implant for each tooth is considered in the premaxilla; in the mandible, two implants can often be used to replace the four anterior incisors.

Division A Treatment Plans

Class IV, Division A patients are good candidates for endosteal root form implant placement in the edentulous space. This allows the restoration of teeth independently should they need it and allows the fabrication of shorter-span restorations. This situation is also indicated when occlusal forces are too great for the natural abutment teeth to act as support for a fixed prosthesis but are not mobile. If the adjacent teeth are mobile, then the implant must support both the missing teeth and the mobile teeth during occlusion. As a general rule, the final prosthesis should be completely implant supported, and two implants should support each section of three missing tooth roots (not three missing crowns). Mobile natural teeth adjacent to the edentulous span cause greater loads on the implants. Therefore one implant for each missing root may be indicated.

Division B Treatment Plans

The Class IV, Division B patient is most often treated with augmentation before implant placement. If the ridge is Division B and inadequate in width for Division A root form implants, then the narrow-diameter root forms compromise esthetics and oral hygiene procedures. Bone augmentation is more often used in anterior edentulous regions with narrow bone, and Division A implants are indicated to improve the final crown contour, esthetic appearance, and daily maintenance. Implant and tooth replacement should remain independent. The canine is an important natural abutment. When the canine and two adjacent teeth are missing, a fixed prosthesis is contraindicated. In other words, an implant should replace a canine whenever multiple teeth are missing, which includes the canine.[29] A hydroxyapatite (HA) graft is often placed on the labial aspect of the Division B edentulous ridge for enhanced soft tissue contour, proper emergence profile, and improved lip support for esthetics when pontics are used in the region.

Division C and D Treatment Plans

The first option for a Class IV patient is to use bone augmentation procedures. If the intent of the bone graft is to change a Division C or D to a Division A or B for endosteal implants, then autogenous bone is indicated. Implants may be placed after the graft has created a Division A ridge, and the treatment plan follows the options previously addressed.

CLASSIFICATION OF COMPLETELY EDENTULOUS ARCHES

Completely edentulous classifications include the classification of Kent and the Louisiana Dental School.[30] The classification was for ridge augmentation with HA and a conventional denture. This classification treats all regions of an edentulous arch in similar fashion and does not address regional variation. Likewise, the classification of Lekholm and Zarb only addressed the anterior maxilla and mandible, always resulted in root form implants without regard for bone grafting, and always used a fixed prosthesis, regardless of biomechanical considerations.[30a] The divisions of bone previously presented by Misch and Judy are the basis of the classification of the completely edentulous patient presented in this chapter.[15,16] Its purpose is to allow communication of not only the volume of bone but also its location. It organizes the most common implant options of prosthodontic support for the completely edentulous patient.

The edentulous jaw is divided into three regions and described according to the Misch-Judy classification.[30b] In the mandible, the right and left posterior sections extend from the mental foramen to the retromolar pad, and the anterior area is located between the mental foramina. The anterior section usually extends from first premolar to first premolar because of the foramen's most common location (i.e., between the two premolar teeth). The right and left posterior regions of the edentulous maxilla also start from the second premolar site, where the maxillary sinus most often determines the height of available bone. The anterior section of the maxilla consists of the region between the first premolars and is usually anterior to the maxillary sinus (Figure 19-4). The division of bone in each section of the edentulous arch then determines the classification of the edentulous jaw. The three areas of bone are evaluated independently from each other. Therefore one, two, or three different divisions of bone may exist. The term *type* is used in the completely edentulous classification, rather than *class*, as in the partially edentulous classification.

Type 1

In the Type 1 edentulous arch, the division of bone is similar in all three anatomical segments. Therefore four different categories of Type 1 edentulous arches are present. In the Type 1, Division A ridge, with abundant bone in all three sections, as many root forms as needed may be inserted (and the locations of implants are not

WWW.HIGHDENT.IR

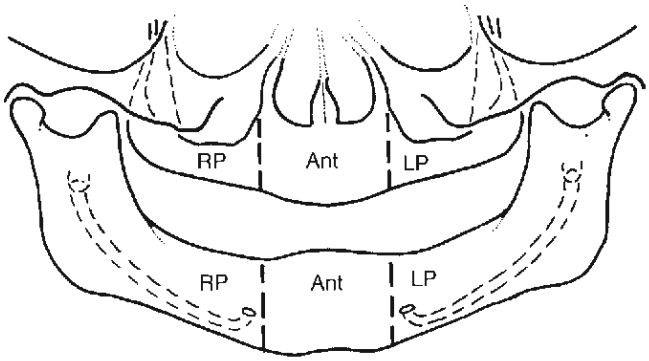


Figure 19-4 The completely edentulous jaw is divided into three segments. The anterior component *(Ant)* is between the mental foramina or in front of the maxillary sinus. Right *(RP)* and left *(LP)* posterior segments correspond to the patient's right and left sides.

limited) to support the final prosthesis. As a general rule, the range of five to nine implants may be used in the mandible and 7 to 10 implants in the maxilla for a fixed prosthesis.

The Type 1, Division B edentulous ridge presents adequate bone in all three sections in which to place narrow-diameter root form implants. It is common practice to modify the anterior section of bone in the mandible by osteoplasty to a Division A and to place full-size root form implants in this region. It is more unusual to have sufficient height in either the posterior maxilla or mandible to permit osteoplasty to improve the division. Therefore several narrower implants are often indicated in the mandible if posterior implants are inserted without grafting. One implant is used for every tooth root to compensate for the decrease in surface area of implant support. Augmentation by bone spreading may be indicated in the maxilla, if the patient desires a fixed prosthesis, especially when opposing natural teeth. If stress factors are great, then lateral augmentation may also be necessary in the posterior regions to increase implant diameter.

Type 1, Division C–w edentulous arches present adequate height of available bone but have inadequate width. If the patient desires an implant-supported removable prosthesis, then an osteoplasty may be used to convert the ridge to C–h. The treatment plan then follows a Type 1, Division C–h formula. When a fixed restoration is desired, an autogenous onlay graft in the C–w arch is usually warranted to restore the ridge to Division A before implant insertion.

Type 1, Division C–h edentulous arches often do not present all the essential requirements for predictable long-term implant support for fixed prostheses. An implant-supported RP-4 or RP-5 removable prosthesis is often indicated to reduce occlusal loads. The mandibular arch may be treated with a complete subperiosteal implant and/or root form implants in the anterior section. The prosthesis should be completely implant supported (RP-4) to halt the continued bone loss in the posterior regions of the mouth. When only Division C anterior root form implants are inserted, posterior soft tissue support (RP-5) may be required.

The edentulous maxilla is often treated with a conventional removable prosthesis until the mandible is completely restored. If this denture needs additional retention or stability, then HA can be used to augment the premaxilla. This squares the ridge shape and provides resistance to occlusal excursions during function. Intramucosal inserts may also be used to increase the retention of the removable complete denture. However, the patient and doctor should realize that bone loss will continue and will make future implant placement even more difficult.

The C–h maxilla should often consider subnasal augmentation combined with root form implants in the canine eminence region and sinus graft with root form implants with an RP-4 prosthesis.[31,32] Additional surgical training is required for these last two alternatives, and they have a greater incidence of complication.

Fixed prostheses may need autogenous iliac crest grafts to change the anterior division of bone and improve long-term success and esthetics. Sinus grafts are also indicated in these situations (Figure 19-5).

The edentulous arches classified as Type 1, Division D are the most challenging to traditional and implant dentistry. If an implant fails in a Type 1, Division D patient, then pathologic fractures or almost unrestorable conditions may result; yet these are the patients who need the most help for support of their prostheses. The benefits versus risks must be weighed carefully for each patient. Endosteal implants may be placed in the anterior mandible. However, the unfavorable crown height of greater than 20 mm and mandibular fracture during implant placement or after implant failure may result in significant complications.[33]

Often the best solution is to change the division with autogenous grafts, then reevaluate the improved conditions and appropriately alter the treatment plan. The Type 1, Division D ridges most often use autogenous iliac crest grafts. After 6 months a total of five to nine implants may be placed in the anterior and posterior regions.[34-35]

Type 2

In the Type 2 completely edentulous arch, the posterior sections of bone are similar but differ from the anterior segment. The most common arches in this category present less bone in the posterior regions, under the maxillary sinus, or over the mandibular canal than in the anterior segment. These edentulous ridges are described in the completely edentulous classification with two division letters following Type 2, with the anterior segment being listed first because it often determines the overall treatment plan. Therefore a mandible with Division A between the foramina and

WWW.HIGHDENT.IR

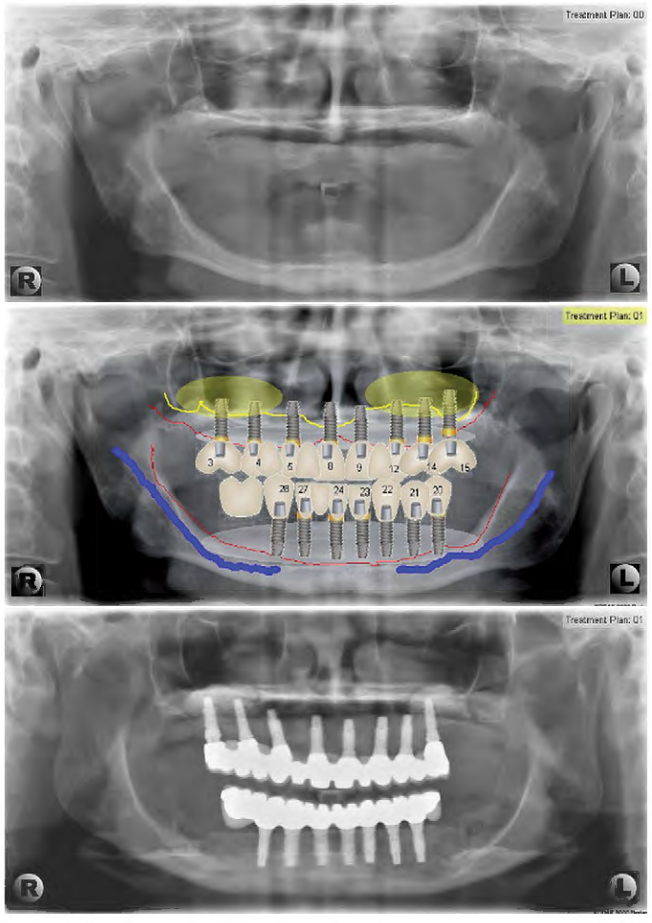


Figure 19-5 **A,** Type I division C–h mandible and maxilla should be restored to Division A with autogenous grafts when a fixed prosthesis is desired. **B,** Computer-assisted treatment plans with five to nine implants placed in the grafted bone volumes can be developed before treatment (XCPT, Naples, Fla.). **C,** Panoramic radiograph of a patient with Division C–h maxilla and in mandible—restored to Division A arches with iliac crest grafts, endosteal implants, and fixed maxilla and mandibular prostheses.

Division C distal to the mandibular foramen is a Type 2, Division A, C arch. This condition is common in the mandible, because the posterior regions resorb four times faster than the anterior regions. Because onlay grafts in the posterior mandible are more difficult to perform predictably, the anterior region is often the only segment used for implant support.

In the Type 2, Division A, B arch, the posterior segments may be treated with narrow-diameter implants, whereas the anterior section is adequate for larger-diameter root form implants to support the prosthesis (Figure 19-6). When possible, the posterior Division B section is changed into Division A. Synthetic onlay augmentation is not as predictable or timely as Division B endosteal implants, which may be used when stress factors are low. Autogenous grafts are more debilitating and require extended healing periods but may be indicated for the benefit of increased posterior bone width when stress factors and patient desires are high. Smaller segments can be augmented with intraorally harvested block grafts.

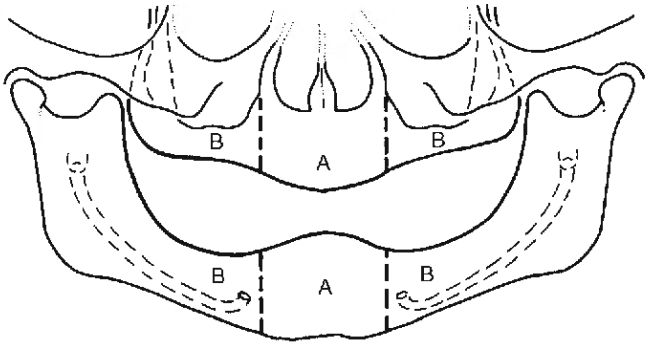


Figure 19-6 A Type 2, Division A, B arch has an anterior section classified as *Division A (A)* and posterior sections as *Division B (B)*. The anterior region dominates the overall treatment plan in all edentulous arches and usually has a greater volume of bone than the posterior.

In the posterior maxilla, bone spreading and Division A root forms should be considered. The softer the bone is, the easier it is to spread.

Two primary modules exist to restore the Type 2, Division A, C edentulous ridge. In the mandible, the most common option is the use of the anterior section only for implant-supported root form implants (Figure 19-7). The maxillary arch may be treated with a combination of sinus graft and endosteal implants if additional posterior support is required for the prosthesis. Because the bone density of the mandible is usually superior to that of the maxilla, and the moment forces remain directed within the arch form, rarely does the mandible require additional posterior support with grafts or circumferential subperiosteal implants. However, for a patient with a square arch form or high masticatory dynamics such as opposing natural teeth, posterior support may be required for an RP-4 or fixed prosthesis.

An edentulous ridge with severe posterior bone loss and abundant bone in the anterior region is uncommon and occurs more frequently in the maxilla. The Type 2, Division A, D patient is treated in a similar manner to the patient with a Type 2, Division A, C arch. Sinus grafts and endosteal implants in the maxilla or only anterior implants with or without an autogenous graft in the mandible are most often the treatment of choice.

The Type 2, Division B, C edentulous arch can be treated with two main treatment options. The anterior section may be changed to Division A by osteoplasty if anatomical conditions permit. These patients are then treated exactly as the previously described Type 2, Division A, C. When the ridge does not present sufficient height after osteoplasty to upgrade the division, the posterior segments may be improved by sinus grafts and the whole arch treated in the same manner as Type 1, Division B or Type 2, Division B, A. Onlay grafts are less predictable than sinus grafts; therefore the anterior mandible may be changed to a Division C by osteoplasty, and a mandibular complete subperiosteal implant and RP-4 restoration or anterior root forms and

WWW.HIGHDENT.IR

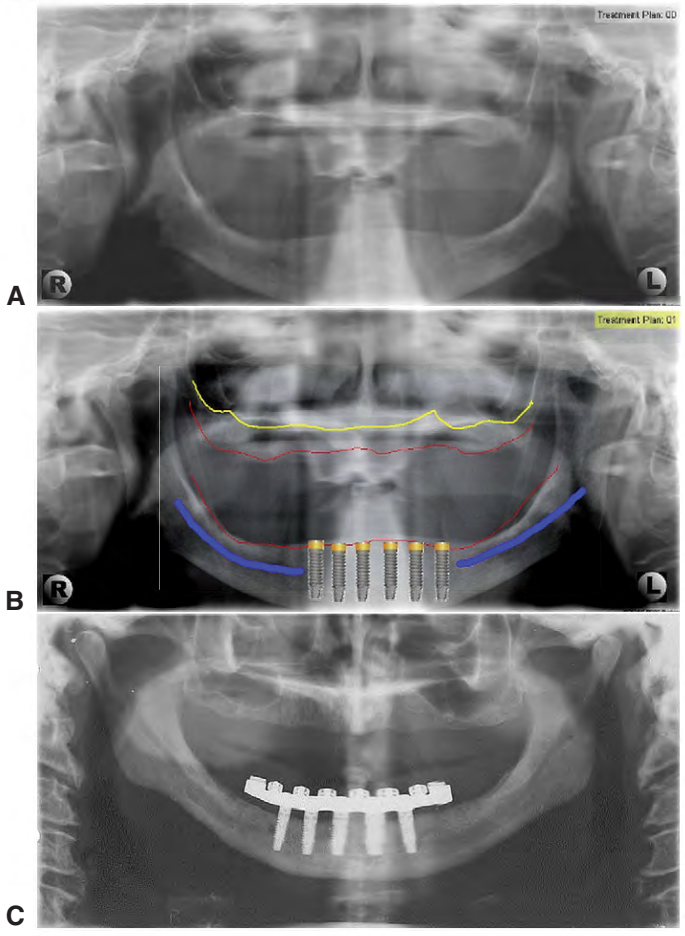


Figure 19-7 **A,** A treatment option for Type 2, Division A, C. **B,** A computer-generated treatment plan with favorable biomechanics places implants in the anterior section of the mandible and restore the arch with cantilevered posterior sections (XCPT, Naples, Fla.). **C,** In a situation with higher masticatory dynamics, this type of arch may mandate the fabrication of a RP-4 prosthesis.

RP-5 prosthesis may be selected for Type 1, Division C mandibular patients.

Patients who have advanced atrophy in the posterior segments and adequate ridge width and height in the anterior may be described as Type 2, Division B, D. This condition almost never occurs in the mandible, but it can be found on occasion in the maxilla. These patients are treated in a manner similar to patients with Type 2, Division B, C, as previously described. The primary difference is that the posterior graft is more extensive and requires additional months for healing before implant insertion and prosthodontic reconstruction. In the mandible, Type 2, Division C, D patients may be treated similar to a Type 1, Division D mandible with autogenous bone grafts before implant placement (Figure 19-8).

Type 3

In Type 3 edentulous arches, the posterior sections of the maxilla or mandible differ from each other. This condition is less common than the other two types and is found more frequently in the maxilla than the mandible. The anterior bone volume is listed first, then the right posterior, followed by the left posterior segment. Therefore the edentulous maxilla with no bone available for implants in the left posterior section, abundant bone in the anterior section, and adequate bone in the right posterior segment is a Type 3, Division A, B, D edentulous arch (Figure 19-9).

The patient with a mandible that has adequate bone in the right posterior segment and inadequate bone on the other side, but abundant bone in the anterior is a Type 3, Division A, B, C edentulous ridge. A narrow-diameter implant may be placed in the right posterior segment, as well as root forms in the anterior section as indicated by the prosthesis. If additional prosthetic support is needed in the left mandibular region, then in most cases additional anterior root forms are placed and splinted to the posterior implants and the teeth or bar cantilevered without implant support on the left posterior region. The Type 3, Division A, C, B patient is treated as a mirror image of Type 3, Division A, B, C.

The Type 3, Division A, D, C (or Type 3, Division A, C, D) patient receives a treatment plan similar to the plans discussed under Type 2, Division A, C. Endosteal root form implants are placed in the anterior section; if the prosthesis needs additional posterior support, then grafts are considered, especially in the posterior maxilla. Patients with Type 3 arches with anterior Division B or C are treated similar to the corresponding Type 2 patients with an anterior Division B or C. In the maxilla, it is not unusual that the premaxilla presents insufficient bone volume and one posterior quadrant requires a sinus graft (Type 3, Division C, A, D). In that case and if appropriate bone volume is present in the cuspid area with favorable force factors, then a full-arch fixed prosthesis can be fabricated after sinus graft and implant placement in the posterior regions, bypassing the premaxilla (Figure 19-10).

The arch is Type 3, even when the anterior region is similar to one of the posterior sections. For example, the Type 3, Division C, D, C ridge has Division C in the anterior, severe atrophy on the right section, and moderate atrophy in the left section. In a mandibular arch of this type, implant placement in the anterior section only may be sufficient to restore the patient, although a subperiosteal implant may be indicated. The maxilla usually requires sinus grafts and subnasal elevation because of the poor biomechanics and bone quality.

The anterior section usually determines the treatment plan. Rarely are posterior implants inserted without any anterior implant support. In traditional prosthetics, Kennedy-Applegate Class I, modification I patients with anterior missing teeth are often restored with an anterior FPD and posterior RPD. This limits rocking of the prosthesis and decreases the forces transmitted to the

WWW.HIGHDENT.IR

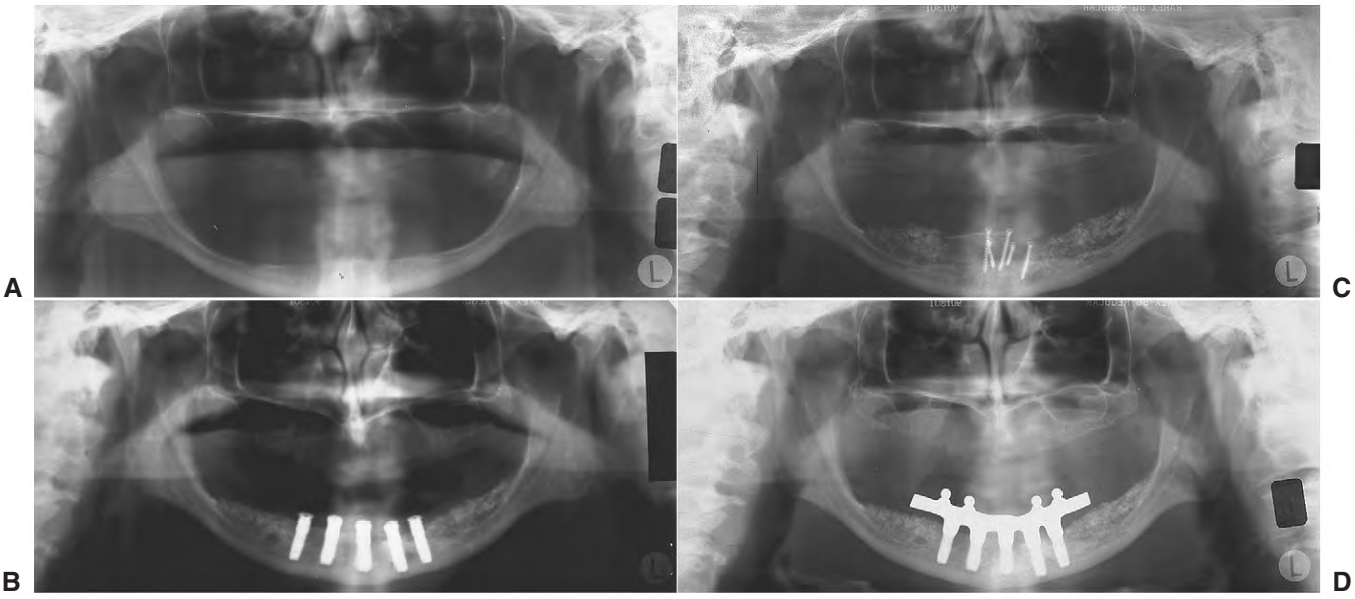


Figure 19-8 **A,** Patients with Type 2, Division C, D mandibles may be treated in a similar fashion as patients with Type I, Division D arches. Iliac crest bone grafting is necessary to restore proper volume of bone to Division A **(B)** before implant placement **(C)** and final restoration fabrication **(D)**.

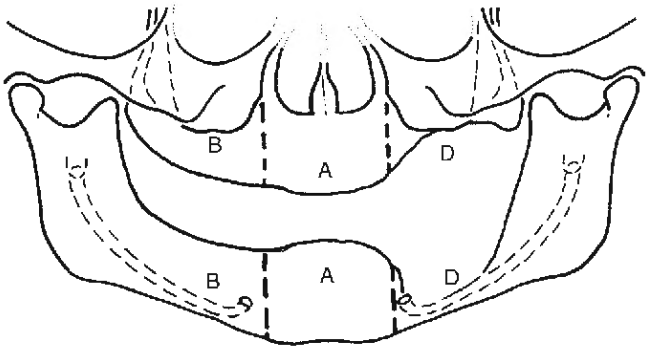


Figure 19-9 This Type 3, Division A, B, D arch has abundant anterior bone *(A)*, moderate bone in the posterior right *(B)*, and severe atrophy in the left posterior segment *(D)*. Sinus grafting is a common treatment if posterior implants are required in the maxilla. However, bone augmentation in the posterior mandible is more unusual, and additional anterior implants with a cantilever are more typical.

Figure 19-10 A maxilla with Type 3, Division C, D, E, may require bilateral sinus grafts and implants in the canine with nasal elevation to support a fixed prosthesis.

abutments. Conventional prosthetics also dictate that a FPD is not indicated when the canine and two adjacent teeth are missing. This applies also when the anterior six teeth are missing and implants cannot be inserted. These time-tested, traditional prosthodontic axioms indicate that posterior implants alone should not be placed without any anterior implant or natural tooth support. However, this concept is often ignored in the maxilla, where practitioners often rely solely on sinus grafts and implants in the posterior segments. If no canine implants are inserted, then the lack of anterior support can cause rotation of the prosthesis and accelerate posterior implant loss. The two posterior sections are not connected because the span between the first premolars is too great, and the posterior implants are placed almost in a straight line with little biomechanical advantage. Anterior rocking and posterior lateral forces on these straight-line implants increase implant failure. The patient's condition is then often worse than before any implant therapy. It is usually far more prudent to convince the patient to be treated with an anterior onlay graft and anterior implants so that a full-arch restoration (RP-4 or fixed) may be fabricated.

SUMMARY

An implant dentistry classification has been outlined that permits visualization of teeth and bone in partially edentulous arches. The foundation of this classification is the Kennedy-Applegate system, which is the most-used classification in prosthodontics. A classification for the completely edentulous arch based on available bone also has been developed.

WWW.HIGHDENT.IR

References

1. Cummer WE: Possible combinations of teeth present and missing in partial restorations, *Oral Health* 10:421, 1920.
2. Kennedy E: *Partial denture construction,* Brooklyn, NY, 1928, Dental Items of Interest.
3. Bailyn M: Tissue support in partial denture construction, *Dent Cosmos* 70:988, 1928.
4. Neurohr F: *Partial dentures: a system of functional restoration,* Philadelphia, 1939, Lea & Febiger.
5. Mauk EH: Classification of mutilated dental arches requiring treatment by removable partial dentures, *J Am Dent Assoc* 29:2121, 1942.
6. Godfrey RJ: Classification of removable partial dentures, *J Am Coll Dent* 18:5, 1951.
7. Beckett LS: The influence of saddle classifications on the design of partial removable restoration, *J Prosthet Dent* 3:506, 1953.
8. Friedman J: The ABC classification of partial denture segments, *J Prosthet Dent* 3:517, 1953.
9. Austin KP, Lidge EF: *Partial dentures: a practical textbook,* St Louis, 1957, Mosby.
10. Skinner CNA: Classification of removable partial dentures based upon the principles of anatomy and physiology, *J Prosthet Dent* 9:240-246, 1959.
11. Applegate OC: *Essentials of removable partial denture prosthesis,* ed 3, Philadelphia, 1965, WB Saunders.
12. Avant WE: A universal classification for removable partial denture situations, *J Prosthet Dent* 16:533-539, 1966.
13. Parameters of care for the American College of Prosthodontists, *J Prosthodont* 5:3-71, 1996.
14. Garry TJ, Nimmo A, Skiba JF et al: Classification system for partial edentulism, *J Prosthodont* 11:181-193, 2002.
15. Misch CE: *Available bone improved surgical concept in implant dentistry.* Paper presented at the Alabama Implant Study Group, Congress XI, Birmingham, Ala, May 1985.
16. Misch CE, Judy WMK: Classifications of the partially edentulous arches for implant dentistry, *Int J Oral Implantol* 4:7-12, 1987.
17. Marcus SE, Drury TF, Brown LJ et al: Tooth retention and tooth loss in the permanent dentition of adults: United States 1988-1991, *J Dent Res* 75(special issue):684-695, 1996.
18. Laney WR, Gibilisco JA: *Diagnosis and treatment in prosthodontics,* Philadelphia, 1983, Lea & Febiger.
19. Cecconi B, Asgtar K, Dootz E: The effect of partial denture clasp design on abutment tooth movement, *J Prosthet Dent* 25:44, 1971.
20. Thompson W, Kratochvil F, Caputo A: Evaluation of photoelastic stress patterns produced by various designs of bilateral distal-extension removable partial dentures, *J Prosthet Dent* 38:261, 1997.
21. Davis WH: Neurologic complications in implant surgery. In the *American Association of Oral and Maxillofacial Surgeons Congress, Clinical Study Guide,* 1992.
22. Fonseca RJ, Davis HW: *Reconstructive preprosthetic oral and maxillofacial surgery,* ed 2, Philadelphia, 1995, WB Saunders.
23. Naert I, Quirynen M, van Steenberghe D et al: A six year prosthodontic study of 509 consecutively inserted implants for the treatment of partial edentulism, *J Prosthet Dent* 67:236-245, 1992.
24. van Steenberghe D, Lekholm U, Bolender C et al: The applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study on 558 fixtures, *Int J Oral Maxillofac Implants* 5:272-281, 1990.
25. Walton JN, Gardner MF, Agar JR et al: A survey of crown and fixed partial denture failures: length of service and reasons for replacement, *J Prosthet Dent* 56:416-420, 1986.
26. Andersson B, Odman P, Lidvall AM et al: Single tooth restorations supported by osseointegrated implants, *Int J Oral Maxillofac Implants* 10:702-711, 1995.
27. Henry PJ, Laney WR, Jemt T et al: Osseointegrated implants for single tooth replacement. A prospective 5-year multicenter study, *Int J Oral Maxillofac Implants* 11:450-455, 1996.
28. Misch CE: Maxillary anterior single tooth implant esthetic-health compromise, *Int J Dental Symp* 3:4-9, 1995.
29. Misch CE, Bidez MW: Implant protected occlusion, a biomechanical rationale, *Compend Cont Educ Dent* 15: 1330-1344, 1994.
30. Kent JN: Correction of alveolar ridge deficiencies with non-resorbable hydroxylapatite, *J Am Dent Assoc* 105:99-100, 1982.

30a. Lekhalm U, Zarb GA: Patient selection and preparation. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue integrated prostheses: osseointegration in clinical dentistry,* Chicago, 1985, Quintessence.

30b. Misch CE: Classification of partially and completely edentulous arches in implant dentistry. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.

31. Misch CE: Available bone influences prosthodontic treatment, *Dent Today* 7:44-75, 1988.
32. Misch CE: Maxillary sinus augmentation for endosteal implants: organized alternative treatment plans, *Int J Oral Implantol* 4:49-58, 1987.
33. Tolman DE, Keller EE: Management of mandibular fractures in patients with endosseous implants, *Int J Oral Maxillofac Implants* 6:427-436, 1991.
34. Misch CE: Iliac crest grafts and endosteal implants to restore 35 severely resorbed totally edentulous maxillae: a retrospective study. In *Proceedings of the Second World Congress of Osseointegration,* Rome, October 1996.
35. Li KK, Stephens WL, Gliklich R: Reconstruction of the severely atrophic edentulous maxilla using the Lefort I osteotomy with simultaneous bone graft and implant placement, *J Oral Maxillofac Surg* 54:542-547, 1996.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 20

Medical Evaluation of the Dental Implant Patient

Carl E. Misch, Randolph R. Resnik

The medical evaluation of patients considering implant therapy is an important aspect to consider for every patient. A retrospective analysis of Veterans' Administration Registry data found that the medical status of patients (i.e., medical history, American Society of Anesthesiologists [ASA] category, and medication history) correlated with implant failure.[1] This chapter is specific for an implant candidate and is developed in three sections. The first section focuses on the importance of the patient interview, with primary emphasis on the medical history questionnaire and the physical examination. The medical history includes those medical conditions most likely to influence implant treatment decisions. The physical examination consists of a hands-on evaluation and recording of the patient's vital signs. The second section (Laboratory Evaluation) reviews those laboratory tests of interest to implant dentistry. The evaluation includes a complete blood cell count (CBC), basic metabolic panel (BMP), comprehensive metabolic panel (CMP), or bleeding disorder tests. The third section relates the medical and dental implications of the most common systemic diseases found in implant patients.

MEDICAL EVALUATION

The medical evaluation remains of paramount importance in implant dentistry, perhaps more so than in other disciplines of dentistry.[2] Implant treatment is primarily a surgical, prosthetic, and maintenance discipline for an older segment of the population. The need for implant-related treatment increases with the age of the patient; as a result, the implant dentist treats more elderly patients than do other specialists in dentistry.[3,4]

An estimated 12% of the U.S. population is 65 years of age or older; this number is expected to reach 21% (64.6 million) in the year 2030.[5] A 65-year-old person has a life expectancy of another 16.7 years, and an 80-year-old person can expect to live an additional 8 years.[6] These patients often request implant support for their failing fixed restorations or to improve the conditions of their removable prostheses. An increased life span means the number of elderly patients in the dental practice is likely to increase. Therefore it is important to design the medical and physical evaluations to accommodate the special conditions of these patients.

Physiologic Changes in the Elderly

Physiologic changes associated with aging and their pharmacologic counterparts modify the physical, social, and economic life of the patient. Although important individual variations exist, the biologic systems of the elderly patient must cope with a decrease in function and physiologic reserve. These physiologic changes may predispose or increase the aging patient's susceptibility to disorders.

In general, a healthy older person demonstrates only half the lung function of a healthy young adult. The blood flow of an older patient is 80% that of the healthy 30 year old, the cardiac output is only 70%, the renal plasma flow is only 50%, and the glomerular filtration is only 60%. A decrease in the elasticity of the arterial system is illustrated by an increase in systolic blood pressure. The vital capacity is reduced to 70% of a 17-year-old patient and corresponds to a decrease in the arterial partial pressure of oxygen.[7] Gastric motility and intestinal absorption are also decreased.

The total body weight of the patient is often reduced, especially if masticatory deficiency from lack of teeth and bone is present; however, there is an increase in body fat. Consequently, any medications administered will follow modified pharmacokinetics and dynamics. Drug kinetics are modified, especially in the distribution phase, as a result of the increased water weight, decreased plasma albumin, and decreased cardiac output. The decreased plasma albumin concentration causes a greater percentage of the drug to remain free and active. The decreased ability to metabolize drugs, related to the decreased renal function, is responsible

WWW.HIGHDENT.IR

for the decreased excretion of the drugs. Therefore the intervals between drug administration should be longer and dosages should be decreased, except for liposoluble drugs and antibiotics, to compensate for the increase in body fat and the reduced immune response.

The decreased gastric motility of the elderly patient affects the use of oral analgesics such as codeine. Pharmacodynamic alterations include an increased sensitivity to central nervous system (CNS) depressant drugs. The individual variations are greater than in other segments of the population; the dosage should be assessed for each patient.

Chronic illness and multiple disease states are characteristics of aging. Patient surveys indicate that 80% of the elderly have at least one chronic disease. Half of the people older than 65 years have arthritis, 42% have hypertension, 34% have other cardiovascular problems, and more than 40% have hearing impairments. Other conditions associated with aging are the increased frequency of diabetes (8.5%), immune response problems, orthopedic (osteoporosis) problems (17%), and sensory deficiencies as well as degenerative diseases.[8,9] The influence of chronic disease states doubles for people older than 65 years of age and affects pathologic processes; surgery may increase individuals' surgical risks and affect prognoses.

Medications and the number of drugs usually increase with age; women usually take more medications than men. At least 75% of patients 65 and older take medications.[10-12] Elderly patients receive 25% of all prescription drugs, although they represent only 12% of the total population. They receive an average of 13 prescriptions a year, in addition to numerous over-the-counter drugs, mainly analgesics. The drug categories most often prescribed among the top 50 drugs prescribed in 2004 include: (1) cardiovascular drugs (13 of the top 50), (2) central nervous system agents (10 of the top 50), (3) gastrointestinal and respiratory agents (7 of the top 50), and (4) endocrine agents (6 of the top 50).[13]

The cardiovascular drugs most prescribed are digitalis (digoxin; Lanoxin), anticoagulants (warfarin sodium; Coumadin), angiotensin-converting enzyme inhibitors (Vasotec, Zestril, Capoten, Prinivil), diuretics (triamterene, furosemide, Lasix), calcium channel blockers (Procardia, Cardizem, Norvasc), and cholesterol-reducing agents (Lipitor, Zocor). Psychotropics and anticonvulsants are mostly represented by benzodiazepines (Xanax), antidepressants (Prozac, Zoloft), and anticonvulsants (Dilantin, Klonopin). Among gastrointestinal and respiratory drugs are antacids (Zantac), beta bronchodilators (Proventil), and antihistamines (Claritin, Seldane).

Many of these drugs are often the cause of adverse drug reactions. A study showed that more than 70% of drugs taken by elderly patients have potential adverse effects in the dental practice. The most common side effects of prescription drugs are xerostomia (especially antidepressants and antianxiety drugs), gastroesophageal irritation and reflux, and abnormal bleeding.

Among the 13 broad categories of oral side effects of the 200 most frequently prescribed drugs reported, xerostomia was first. This may lead to frequent *Candida* spp. infections, increased periodontal and peri-implant diseases, caries, and bacterial infections caused by the loss of protection from saliva. Xerostomia also decreases the valve seal of soft tissue–borne removable prostheses and increases the risk of abrasions and sore spots. Suggested management includes saliva substitutes, salivary stimulants, frequent glasses of water throughout the day, strict control of the diet to decrease cariogenicity, and avoidance of tobacco or alcohol products.

Side effects of cardiovascular drugs depend on the drug category. Digitalis can cause nausea, anorexia, chromatopsia, and arrhythmias. Diuretics cause dehydration, xerostomia, electrolyte imbalance (potassium depletion), and hyperglycemia and predispose to sialoadenitis. Calcium channel blockers may be responsible for edema, constipation, and gingival hyperplasia. Nifedipine, a calcium channel blocker, is known to exacerbate gingival enlargement around both natural teeth and dental implants. To date, the best management is still frequent professional cleanings combined, if necessary, with surgical removal of the hyperplastic tissue to ease daily care and improve esthetics. Angiotensin-converting enzyme inhibitors may produce cough, angioedema, and taste alteration. Anticoagulants and potential risk of hemorrhage are well documented. Care should be taken not to administer erythromycin with terfenadine (Seldane) because of potential cardiotoxic drug interaction. The most common risk of patients on insulin is hypoglycemia.[14-17] Therefore dental practitioners may have to address a variety of drug-related adverse effects, the most common being:

- Abnormal bleeding (aspirin, nonsteroidal antiinflammatory drugs [NSAIDs])
- Altered hematopoiesis (barbiturates, phenothiazines)
- Altered host resistance (antibiotics, insulin)
- Systemic corticosteroids
- Decreased stress tolerance (beta blockers, calcium channel blockers)
- Gastrointestinal irritation (aspirin, antibiotics)
- Gingival hyperplasia (nifedipine, diltiazem, phenytoin, valproic acid, verapamil)
- Mucosal reaction (barbiturates)
- Respiratory depression (narcotics, barbiturates)
- Salivary gland swelling/pain (clonidine, insulin)
- Phenothiazines
- Sialorrhea (cholinergics, anticonvulsants, antianxiety)
- Taste alteration (ACE inhibitors, benzodiazepines, metronidazole)
- Xerostomia (nearly all drugs)

In older patients, 30% of drug-induced illnesses are due to interactions between medications. Antimicrobials and

WWW.HIGHDENT.IR

analgesics accounted for 40% of these.[17] These interactions account for 20% of hospital admissions of senior citizens. Notably, asthmatic patients taking NSAIDs may experience acute bronchospasm. Terfenadine (Seldane) may cause life-threatening ventricular arrhythmias in patients taking ketoconazole or erythromycin.[18] Erythromycin can cause nausea, vomiting, and cardiac dysrhythmias in patients on carbamazepine treatment. Patients receiving antiparkinsonian treatment with selegiline (Eldepryl)[18-20] may suffer fatal interaction if used with opioids (especially meperidine).[18-20] Patients following antihypertensive therapy should not receive long-term NSAIDs therapy because the latter may significantly lower the efficacy of the antihypertensive agents.

Management of Elderly Implant Patients

The elderly patient's compliance is often decreased. Aging patients have a tendency to forget to take medications or to mistake dosage and frequency. Because of impaired hearing and vision, they may become confused more easily. Many follow an inadequate diet that can impair their condition and slow healing after surgery.

Even healthy older implant candidates (older than 60 years) should be considered as patients with mild systemic disease, just because of their age.[21] As a result of a decrease in physiologic adaptability, the aged patient has less reserve to react to stress. If the limits of homeostatic reserve are reached, the patient can reach a critical condition.[22] The elderly patient can follow regular implant therapy provided a typical stress reduction protocol is implemented. Monitoring of vital signs, modified dosage of medications, and special care during sedation because of an increased sensitivity to CNS depressants are indicated. An increased dosage of antibiotics compensates for the less competent immune system and the increased susceptibility to viral and bacterial diseases. An increased dosage of liposoluble drugs is also suggested. Elderly patients are reportedly less sensitive to pain, so a reduced dosage of narcotic analgesics is recommended, especially because gastric motility is reduced in these patients. The doctor should be aware of the eventual adverse drug reactions to the medications already taken by these patients when combined with those later prescribed for implant surgical procedures.

MEDICAL HISTORY

A written medical history should be obtained for every implant candidate. The review of the patient's medical history is the first opportunity for the dentist to talk with the patient. The time and consideration taken at the onset will set the tone for the entire subsequent treatment. This first impression should reflect a warm, caring practitioner who is highly trained to help patients with complex treatments. A sincere interest and active note-taking process are beneficial. The practitioner should not underestimate the value of the medical history interview. Asking questions that show an understanding of listed medical conditions and related common problems offer several benefits.

The two basic categories of information addressed during the review of the medical history include the medical history and a review of the patient's systemic health. The dental office uses a medical evaluation form to obtain most of this information (Figure 20-1).[23] Of particular note is medication usage within the preceding 6 months, allergies, and a review of the systems of the body. The pathophysiology of the systems, the degree of involvement, and the medications being used to treat the conditions are evaluated. It is important to review this form with the patient to ensure that comprehension is adequate to answer all questions. The form should include all medical areas of interest to the implant dentist.

Extraoral and Intraoral Examinations

After the medical history is reviewed, the medical physical examination begins. This is the first physical contact the office staff has with the patient. A gentle, caring approach should continue throughout the examination. A complete evaluation of the head and neck is important initially and at all subsequent preventive maintenance (recall) appointments. The patient is informed of the need for periodic examination for cysts and benign or malignant tumors, because 26,000 cases of cancer are diagnosed in the head and neck region each year.

The extraoral and intraoral examinations are similar to those addressed in any oral diagnosis textbook. A few specific areas are mentioned because complications specific to implant treatment have been observed. The extraoral examination is performed first. Features and facial symmetry are observed, including the ears, nose, and eyes. If the midline, occlusal plane, or smile line of the natural teeth or existing prosthesis is not harmonious, the cause should be determined. Patients are very receptive to critical evaluation and treatment limitations relating to facial esthetics before reconstruction begins. However, a similar discussion at the end of treatment is considered an excuse.

The submental, submandibular, parotid, and cervical areas are palpated for lymphadenopathy or unusual swelling. Sialoliths may be blamed on implant surgery, when they were actually previously present. The area between the cricoid notch and the suprasternal notch is palpated for hypertrophy of the thyroid gland because its physiology influences bone metabolism and implant management.

Intraoral examination of the lips, labial and buccal mucosa, hard and soft palate, tongue, and oral pharynx is then performed. Any lesions or disease states must be further evaluated before implant procedures commence.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

About Your Medical History

For the following questions, check YES or NO, whichever applies. Your answers are for our records only, and will be confidential.
THESE FACTS HAVE A DIRECT BEARING ON YOUR DENTAL HEALTH

Sex:_____ Height:_____ Weight:_____ Race:_____

		Date ___/___/___	Date ___/___/___
General Medical History	1. Are you in good general health?	❑ Yes ❑ No	❑ Yes ❑ No
	2. Has there been ANY change in your general health in the past year?	❑ Yes ❑ No	❑ Yes ❑ No
	3. My last physical examination was on, approximate date		
	4. Are you PRESENTLY under a physician's care?	❑ Yes ❑ No	❑ Yes ❑ No
	If YES, what condition?		
	5. The physician's name and address		
	6. Have you had any serious illness or operation?	❑ YES ❑ NO	❑ YES ❑ NO
	If YES, please list		
	7. Have you ever been hospitalized or had a serious illness within the past 5 years?	❑ Yes ❑ No	❑ Yes ❑ No
	If YES, reason		
Cardiovascular System	CV1. Do you have or have you ever had any of the following: Please Check ❑ Heart trouble ❑ Heart attack ❑ Coronary insufficiency ❑ Mitral Valve Prolapse ❑ Stroke ❑ Damaged heart valves ❑ Congenital heart disease		
	CV2. Rheumatic heart disease, heart murmur?	❑ Yes ❑ No	❑ Yes ❑ No
	CV3. Chest pain after exertion?	❑ Yes ❑ No	❑ Yes ❑ No
	CV4. Shortness of breath after mild exercise?	❑ Yes ❑ No	❑ Yes ❑ No
	CV5. Do your ankles swell?	❑ Yes ❑ No	❑ Yes ❑ No
	CV6. Do you use extra pillows to sleep?	❑ Yes ❑ No	❑ Yes ❑ No
	CV7. Do you have a cardiac pacemaker?	❑ Yes ❑ No	❑ Yes ❑ No
	CV8. Do you have any blood pressure problems?	❑ Yes ❑ No	❑ Yes ❑ No
	If YES,	❑ High ❑ Low	❑ High ❑ Low
Central Nervous System	CN1. Do you have or have you ever had:		
	CN1A. Epilepsy?	❑ Yes ❑ No	❑ Yes ❑ No
	CN1B. Fainting spells?	❑ Yes ❑ No	❑ Yes ❑ No
	CN1C. Seizures?	❑ Yes ❑ No	❑ Yes ❑ No
	CN1D. Emotional disturbances?	❑ Yes ❑ No	❑ Yes ❑ No
	CN2. Do you follow any treatment for a nervous disease?	❑ Yes ❑ No	❑ Yes ❑ No
Respiratory System	RE1. Do you have a persistent cough or cold?	❑ Yes ❑ No	❑ Yes ❑ No
	RE2. Do you have or have you ever had tuberculosis?	❑ Yes ❑ No	❑ Yes ❑ No
	RE3. Is there ANY history of tuberculosis in your family?	❑ Yes ❑ No	❑ Yes ❑ No
	RE4. Do you have any sinusitis, sinus trouble?	❑ Yes ❑ No	❑ Yes ❑ No
	RE5. Do you have emphysema, chronic bronchitis, asthma?	❑ Yes ❑ No	❑ Yes ❑ No
Digestive System	GI1. Do you have ANY stomach ulcers?	❑ Yes ❑ No	❑ Yes ❑ No
	GI2. Do you have or have you ever had:		
	GI2A. Acic Reflux (GERD)?	❑ Yes ❑ No	❑ Yes ❑ No
	G12B. Hepatitis?	❑ Yes ❑ No	❑ Yes ❑ No
	GI2C. Jaundice?	❑ Yes ❑ No	❑ Yes ❑ No
	GI2D. Liver disease?	❑ Yes ❑ No	❑ Yes ❑ No
	GI3. Have you ever vomited blood?	❑ Yes ❑ No	❑ Yes ❑ No
	GI4. Do you have ANY diarrhea?	❑ Yes ❑ No	❑ Yes ❑ No

About Your Medical History

		Date ___/___/___	Date ___/___/___
Endocrine System	EN1. Do you have diabetes? controlled/uncontrolled	❑ Yes ❑ No	❑ Yes ❑ No
	EN2. Does anyone in your family have diabetes?	❑ Yes ❑ No	❑ Yes ❑ No
	EN3. Do you urinate more than six times a day?	❑ Yes ❑ No	❑ Yes ❑ No
	EN4. Are you thirsty very often or do you have a dry mouth?	❑ Yes ❑ No	❑ Yes ❑ No
	EN5. Do you have hypothyroidism or hyperthyroidism?	❑ Yes ❑ No	❑ Yes ❑ No
Hematogenic System	HB1. Do you have anemia, Sickle Cell disease, blood disorder?	❑ Yes ❑ No	❑ Yes ❑ No
	HB2. Is there ANY family history of blood disorders?	❑ Yes ❑ No	❑ Yes ❑ No
	HB3. Are you hemophilic?	❑ Yes ❑ No	❑ Yes ❑ No
	HB4. Have you had abnormal bleeding after any surgery, extraction, or trauma?	❑ Yes ❑ No	❑ Yes ❑ No
	HB5. Have you ever had a blood transfusion?	❑ Yes ❑ No	❑ Yes ❑ No
	HB6. Immunodeficiency problem?	❑ Yes ❑ No	❑ Yes ❑ No
Allergies	AL1. Are you allergic to or have you reacted adversely to:		
	AL1A. Local anesthetic	❑ Yes ❑ No	❑ Yes ❑ No
	AL1B. Antibiotics, Penicillin, Sulfa Drugs?	❑ Yes ❑ No	❑ Yes ❑ No
	AL1C. Barbiturates, sedatives, or sleeping pills?	❑ Yes ❑ No	❑ Yes ❑ No
	AL1D. Aspirin?	❑ Yes ❑ No	❑ Yes ❑ No
	AL1E. Iodine?	❑ Yes ❑ No	❑ Yes ❑ No
	AL1F. Codeine or other narcotics?	❑ Yes ❑ No	❑ Yes ❑ No
	AL1G. Latex?	❑ Yes ❑ No	❑ Yes ❑ No
	AL1H. Others? Please Specify		
	AL2. Do you have asthma, hay fever or seasonal allergies?	❑ Yes ❑ No	❑ Yes ❑ No
	AL3. Do you have or have you ever had hives or skin rash?	❑ Yes ❑ No	❑ Yes ❑ No
System	UR1. Do you have or have you ever had:		
	UR1A. Kidney trouble?	❑ Yes ❑ No	❑ Yes ❑ No
	UR1B. Dialysis?	❑ Yes ❑ No	❑ Yes ❑ No
	UR1B. Syphilis, gonorrhea?	❑ Yes ❑ No	❑ Yes ❑ No
Bones & Joints	BJ1. Do you have or have you ever had:		
	BJ1A. Arthritis?	❑ Yes ❑ No	❑ Yes ❑ No
	BJ1B. Inflammatory rheumatism?	❑ Yes ❑ No	❑ Yes ❑ No
	BJ1C. Bone infection?	❑ Yes ❑ No	❑ Yes ❑ No
	BJ1D. Osteoporosis?	❑ Yes ❑ No	❑ Yes ❑ No
	BJ1E. Artificial Joint Replacement?	❑ Yes ❑ No	❑ Yes ❑ No
	BJ1F. 1. Have you received or are you currently receiving the intravenous medication known as biphosphonate for example, Zomata (IV) (zoledronic acid) or Aridia (IM) (pamidronate)	❑ Yes ❑ No	❑ Yes ❑ No
	2. Are you taking or have you taken the oral medication known as biphosphonate for osteoporosis or another medical condition for example, Fosamax (alendronate), Actonal (risedronate) or Boniva (ibandronate sodium)	❑ Yes ❑ No	❑ Yes ❑ No
	If yes to 1 or 2		
	3. Have you noticed any changes in your mouth or jaws?	❑ Yes ❑ No	❑ Yes ❑ No
	4. Have you had any jaw pain or toothache(s)?	❑ Yes ❑ No	❑ Yes ❑ No
	5. Have you noticed any foul smell, swelling or discharge in your mouth?	❑ Yes ❑ No	❑ Yes ❑ No
Other	TR1. Do you have or have you ever had:		
	TR1A. Tumor or malignancy?	❑ Yes ❑ No	❑ Yes ❑ No
	TR1B. Chemotherapy or radiation therapy?	❑ Yes ❑ No	❑ Yes ❑ No
	Do you have or have you ever had ANY disease, condition or problem NOT listed above that you think we should know about?		
	If so, please explain		
	TR2. Are you regularly exposed to x-rays or ANY other ionizing radiation or toxic substances?	❑ Yes ❑ No	❑ Yes ❑ No
	TR3. Do you have glaucoma?	❑ Yes ❑ No	❑ Yes ❑ No
	If so,	❑ Wide ❑ Close	❑ Wide ❑ Close

Figure 20-1 Medical history form.

continued

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

About Your Medical History

		Date ___/___/___	Date ___/___/___
TR4.	Are you wearing or do you wear contact lenses?	❑ Yes ❑ No	❑ Yes ❑ No
TR5.	Do you drink alcohol?	❑ Yes ❑ No	❑ Yes ❑ No
	If so, how much and how often?		
TR6.	Do you smoke tobacco?	❑ Yes ❑ No	❑ Yes ❑ No
TR7.	Do you use oral tobacco?	❑ Yes ❑ No	❑ Yes ❑ No
	If so, how much and how often?		

Your Medications

		Date ___/___/___	Date ___/___/___
ME1.	Are you taking any of the following medications:		
ME1B.	Anticoagulants, blood thinning agents?	❑ Yes ❑ No	❑ Yes ❑ No
ME1C.	Medicine for high blood pressure?	❑ Yes ❑ No	❑ Yes ❑ No
ME1D.	Tranquilizers?	❑ Yes ❑ No	❑ Yes ❑ No
ME1E.	Iodine?	❑ Yes ❑ No	❑ Yes ❑ No
ME1F.	Aspirin?	❑ Yes ❑ No	❑ Yes ❑ No
ME1G.	Codeine or other narcotics?	❑ Yes ❑ No	❑ Yes ❑ No
ME1H.	Steroids?	❑ Yes ❑ No	❑ Yes ❑ No
ME1I.	Other?	❑ Yes ❑ No	❑ Yes ❑ No
	If so, please explain		

MEDICATION LIST

Please provide a list of **any type of medication** you are presently taking as well as the dosage.
(Prescription or Over the Counter)

Name/Type of drugs	Dosage	How many times per day?

About Your Medical History

For Women

	Date ___/___/___	Date ___/___/___
Are you pregnant?	❑ Yes ❑ No	❑ Yes ❑ No
Are you nursing?	❑ Yes ❑ No	❑ Yes ❑ No
Do you have any problems associated with your menstrual period?	❑ Yes ❑ No	❑ Yes ❑ No
Are you taking oral contraceptives?	❑ Yes ❑ No	❑ Yes ❑ No
Are you undergoing hormonal therapy?	❑ Yes ❑ No	❑ Yes ❑ No

Dental History

What is your chief dental complaint?

Are you experiencing any discomfort or pain at this time?	❑ Yes ❑ No	❑ Yes ❑ No
Are you satisfied with the appearance of your teeth?	❑ Yes ❑ No	❑ Yes ❑ No
Are you able to eat and chew foods satisfactorily?	❑ Yes ❑ No	❑ Yes ❑ No
Do you have headaches, ear aches, or neck pain?	❑ Yes ❑ No	❑ Yes ❑ No
Do you frequently experience sinus problems?	❑ Yes ❑ No	❑ Yes ❑ No
Have you had ANY serious trouble associated with ANY previous dental treatment?	❑ Yes ❑ No	❑ Yes ❑ No
If YES please explain		

Other Conditions Not Listed

General Dental Responsibility And Consent Statement

I hereby authorize and request the performance of dental services for myself or for: _____

I also give my consent to ANY advisable and necessary dental procedures, medications or anesthetics to be administered by the attending dentist or his supervised staff for diagnostic purposes or dental treatment. These records may include study models, photographs, x-rays and blood studies. I understand and acknowledge that I am financially responsible for the services provided for myself and or the above named, regardless of insurance coverage. Treatment plans involving extended credit circumstances are subject to a credit check. I also understand that the treatment estimate presented to me is only an estimate. Occasionally, the need may arise to modify treatment. In such a case, I will be informed of the need for additional treatment, and any fee modification.

To the best of my knowledge the information in this form is accurate.

Signature of Patient or Guardian	Date
Signature of Witness	Date
Signature of Doctor	Date

Figure 20-1, cont'd Medical history form.

WWW.HIGHDENT.IR

Vital Signs

The recording of vital signs (blood pressure, pulse, temperature, respiration, weight, and height) is also part of the physical examination. This information can often be gathered by trained dental auxiliary personnel before the patient's history is reviewed by the dentist. If any findings are unusual, the doctor can repeat the evaluation as needed.

Blood Pressure

Approximately 10% of dental offices record the patient's blood pressure.[24] This proves worthwhile for the implant dentist because surgery and long prosthodontic procedures are frequently required. The importance of obtaining and recording the blood pressure in every implant patient is twofold. First, the initial recording may serve as a baseline measurement, which if too high may contraindicate a surgical procedure. Second, when in an acceptable range, the initial blood pressure acts as a baseline measurement specific for that patient. If the patient has a future problem during treatment, the blood pressure difference between baseline and the current situation may alter the medical risk of the patient.

The blood pressure is measured within the arterial system. The maximum pressure is the systolic, and the minimum pressure is the diastolic. Blood pressure is influenced by the cardiac output, blood volume, viscosity of the blood, condition of blood vessels (especially the arterioles), and heart rate. There is a direct and an indirect determination of blood pressure. The dentist will use only the indirect method. This technique was first developed by the Italian physician Riva-Rocca in the nineteenth century.[25] The sphygmomanometer consists of an inflatable bag covered by a cuff and a manometer to register the force and rate of air within the bag. The two most common manometer systems use mercury gravity or aneroid gauges. The mercury system is more accurate in changing climates; after it is calibrated, it is consistent for many years. The aneroid manometer is as accurate as the gravity type, but requires regular calibration.

Blood pressure monitoring should only be completed by trained medical personnel. The operator should be trained and retrained on a regular basis in the standardized technique. Patients should ideally be seated for 5 minutes, with their feet on the floor and arm supported at the level of the heart. Caffeine intake, smoking, and strenuous exercise should be avoided for at least 30 minutes before measurement. At least two measurements should be taken, with the average recorded. An appropriately sized cuff (cuff bladder encircling 80% of the arm) should be used for accuracy.[26] The inflatable cuff is positioned over the bare upper arm at the level of the patient's heart, with the patient's palm supine. The brachial or radial artery is palpated and the bag is inflated to obliterate the vessel, about 30 mm Hg above the estimated systolic pressure. The cuff is deflated 2 to 4 mm Hg at every heartbeat. Using a stethoscope over the brachial artery, the systolic pressure is recorded at the first tapping sound heard. When the sounds become muffled or inaudible the diastolic pressure is noted.

Pulse

Only 3% of dentists record the pulse of their patients, yet much pertinent information is available from this simple procedure. The pulse represents the force of the blood against the aortic walls for each contraction of the left ventricle. The pulse wave travels through the arteries and reaches the wrist 0.1 to 0.2 seconds after each contraction. The actual blood flow takes longer to travel this distance. The usual location to record pulse is the radial artery in the wrist. However, other locations such as the carotid artery in the neck and the temporal artery in the temporal region are convenient to use during implant surgery or dental treatment. Pulse monitors are easy to use and are beneficial during surgery or long prosthetic appointments.

Pulse Rate

The pulse rate should be evaluated for a minimum of 30 seconds, and 1 minute is suggested. The normal pulse rate varies from 60 to 90 beats/min in a relaxed, nonanxious patient. The beats are both strong and regular. The normal cardiac rhythm originates in the sinoatrial node; the pulse reflects the ventricular contractions. The upper limit of normal is considered 100 beats/min; people in excellent physical condition may have a pulse rate of 40 to 60 beats/min. A pulse rate less than 60 beats/min or higher than 110 beats/min in the nonathlete is suspect and warrants a medical consultation.

A decreased pulse rate of normal rhythm (less than 60 beats/min) signals sinus bradycardia. It is natural for some patients and may reach as low as 40 beats/min, although most patients become unconscious below this rate. An adult pulse rate lower than 60 beats/min in a nonathlete mandates medical evaluation before surgery. During implant surgery, inappropriate bradycardia may indicate impending sudden death. If the pulse rate of the patient decreases to less than 60 beats/min and is accompanied by sweating, weakness, chest pain, or dyspnea, the implant procedure should be stopped, oxygen administered, and immediate medical assistance obtained.

An increased pulse rate of regular rhythm (more than 100 beats/min) is called *sinus tachycardia*. This rate is normal if experienced during exercise or anxiety. However, a medical consultation is suggested when a nonanxious patient has a resting pulse rate higher than 100 beats/min. In patients with anemia or severe hemorrhage, the heart rate increases to compensate for the depletion of oxygen in the tissues. Therefore, when increased bleeding during surgery is observed, evaluate

WWW.HIGHDENT.IR

the pulse rate and blood pressure. When elevated, an increase in bleeding is readily observed.

Pulse rate and temperature are also related, with the pulse rate increasing 5 beats/min for each degree that the body temperature rises. Hyperthyroidism and acute or chronic heart disease also may result in sinus tachycardia. A condition called *paroxysmal atrial tachycardia* is characterized by episodes of very fast heartbeats that may last a few minutes or several weeks. All of these conditions affect the surgery or may increase postoperative swelling. The increased swelling favors the occurrence of infections and complications during the first critical weeks after implant placement. This can compromise the subsequent years of implant service to the patient.

Pulse Rhythm

Two types of abnormal pulse rhythm are noted: regular and irregular. A regular irregularity increasing during exercise can signal atrial fibrillation. This may be a consequence of hyperthyroidism, mitral stenosis, or hypertensive heart disease and should be evaluated before implant surgery. Stress-reduction protocols can be implemented and implants may even be contraindicated if the causal conditions are severe.

An irregular abnormal pulse rhythm includes premature ventricular contractions (PVCs), which are noticed as a distinct pause in an otherwise normal rhythm. This condition may be associated with fatigue, stress, or excessive use of tobacco or coffee, but it is also observed during myocardial infarction and as a precursor to cardiac arrest. If during implant surgery, five or more PVCs are recorded within 1 minute, especially when accompanied by dyspnea or pain, the surgery should be stopped, oxygen administered, the patient placed in a supine position, and immediate medical assistance obtained. If the health history includes cardiovascular disease, including hypertension, the pulse rhythm should be recorded. Sudden death in persons older than 30 years with PVC is six times more frequent than in younger persons.[22]

Pulse Strength

The patient's pulse rate and rhythm may be normal, yet the blood volume can affect the character of the pulse. The pulse may alternate between strong and weak beats that can indicate pulsus alternans, which is frequently observed in left ventricular failure, severe arterial high blood pressure, and advanced coronary disease. Implant surgery is contraindicated, and medical consultation with an electrocardiographic examination is needed to obtain a diagnosis. After a diagnosis of pulsus alternans, a patient may have less than 2 years to live.

Temperature

The thermometer was first used clinically by Santorio of Padua, Italy, in the seventeenth century. Normal body temperature ranges from 96.8° to 99.4° F in a healthy individual. It is usually lowest in the morning and highest during late afternoon or evening. An oral temperature of 99.5° F or higher is considered in the febrile range. For every degree of fever, the pulse rate rises 5 beats/min and the respiratory rate increases 4 beats/min. If the patient's temperature is 104° F or higher, medical consultation is suggested.

The usual cause of elevated body temperature is bacterial infection and its toxic by-products. Other causes can be exercise, hyperthyroidism, myocardial infarction, congestive heart failure, and tissue injury from trauma or surgery. Dental conditions causing an elevated temperature include severe dental abscess, cellulitis, and acute herpetic stomatitis. Elective dental treatment (such as implant surgery or bone grafting) is contraindicated when the patient is febrile. The cause of the fever may complicate the postsurgical phase of healing. In addition, because elevated temperature increases the patient's pulse rate, the risks of hemorrhage, edema, infection, and postoperative discomfort are greater. Special attention must be given to a prolonged, sustained fever after surgery because sepsis or possible brain abscess could be present. Low body temperature can be found in hypothyroidism.

Respiration

Respiration is noted while the patient is at rest. The normal rate in the adult varies between 16 and 20 breaths per minute and is regular in rate and rhythm. If the patient uses accessory muscles in the neck or shoulders for inspiration, whether before or during surgery, dyspnea is suspected. Many intravenous drugs, including narcotics, cause dyspnea. Congestive heart failure, bronchial asthma, and advanced pulmonary emphysema also impair breathing. If dyspnea occurs during surgery, the pulse should immediately be evaluated to rule out the presence of PVC or myocardial infarction.

Hyperventilation is the result of both an increased rate and depth of respiration and may be preceded by frequent sighs such as is seen in the anxious patient. A respiratory rate greater than 20 breaths per minute requires investigation. Anxiety may increase this rate, in which case sedatives or stress-reduction protocols are indicated before implant surgery. Other causes for an increased respiration rate are severe anemia, advanced bronchopulmonary disease, and congestive heart failure. All three can affect the surgical procedure or healing response of the implant candidate.

LABORATORY EVALUATION

Routine laboratory screening of patients in a general dental setting who previously reported a normal health history have found that 12% to 18% have undiagnosed systemic diseases.[27,28] Many of these disorders may influence implant surgery protocol or long-term success rates. The percentage of implant patients with unreported systemic illnesses is most likely higher because

WWW.HIGHDENT.IR

Table 20-1 Normal Laboratory Values

Hemoglobin	12-18 g/100 mL
Hematocrit	35%-50%
Red blood cell count	4-6 million/mm^3
White blood cell count	4500-13,500/mm^3
Neutrophils	54%-62%
Lymphocytes	25%-30%
Eosinophils	1%-3%
Basophils	1%
Monocytes	0%-9%
Prothrombin time	1-18 sec
Partial thromboplastin time	By laboratory control
Platelets	140,000-340,000/mL
Bleeding time	1-6 min
International normalized ratio	Without anticoagulant therapy: 1 Anticoagulant therapy range: 2–3
Sodium	135-147 mEq
Potassium	3.5-5 mEq
Bicarbonate	24-30 mEq
Chloride	100-106 mEq
A1c (glycosylated hemoglobin)	6% controlled diabetic
Fasting blood sugar	70-100 mg/dL
Oral glucose tolerance test	<140 mg/dL

the average implant patient is older than those in these general studies. Implant therapy comprises an elective surgery that involves a considerable investment of time and money by the patient. Therefore clinical laboratory tests are often used to supplement diagnosis and treatment planning. Laboratory screening is also of benefit in recognizing oral manifestations of systemic diseases. However, preoperative testing should not be considered mandatory for all patients. Justification of laboratory procedures should relate to the specific type of surgery and the patient's condition. On the other hand, any patient older than age 50 years should have laboratory evaluations as part of a routine physical every 3 years or less. If the patient older than 50 years of age has not had a physical and laboratory tests, a physician referral is warranted before advanced implant surgery.

The most common clinical laboratory evaluation for implant dentistry is obtained from venous blood samples and may include a CBC, BMP, CMP, and bleeding disorder tests.[29] The dentist should select the tests needed in the diagnosis of systemic diseases affecting implant treatment.

Urinalysis

In the healthy adult, approximately 180 L of fluid is filtered through the glomeruli of the kidneys each day. The glomeruli filter the plasma, and the tubules selectively reabsorb water and substances useful to the body. They leave undesirable substances behind or secrete them into the urine. The urinary constituents can be altered in the presence of systemic disease, infection, or focal urinary tract infection.

Urinalysis has more qualitative than quantitative information. It is not indicated as a routine procedure for all dental patients and is rarely used in implant dentistry. It is primarily a screening test for diabetes (glucose/ketone), deficiencies or irregularities in metabolism, renal disease, protein in blood, liver function (bilirubin), or suspected infection. The most frequent use of urinalysis by the dentist concerns suspicion of or screening for diabetes mellitus, which is the most common cause of glucosuria. However, other conditions such as pregnancy, Cushing's disease, Graves' disease, intracranial tumor, and coronary thrombosis can modify this level. In addition, a diabetic patient with high glucose blood levels may not spill glucose into the urine. Examination of the blood, on the other hand, indicates more precisely the patient's glucose metabolism and is a more reliable test. Therefore urinalysis is rarely indicated specifically for oral implant surgery.

Complete Blood Cell Count

The CBC is one of the most common blood tests and consists of the calculation of the cellular components of blood. These calculations are determined by specifically designed machines that analyze the different components of blood in a very short period. These include testing the number of erythrocytes (red blood cells) and leukocytes (white blood cells), leukocytic differential, cellular morphology and maturity, hemoglobin determination, hematocrit, and platelet count (Table 20-1). Indications for a CBC are suspected dyscrasia, glucocorticoid therapy within 1 year, chemotherapy, renal diseases, or expected major blood loss. Asymptomatic patients do not require a CBC unless major blood loss is possible with surgery. Evaluation of the CBC can be limited to three clinical situations for implant dentistry: erythrocytic disorders, leukocytic disorders, and bleeding disorders.[30]

White Blood Cell Count

The normal total white blood cell (WBC) count ranges from 4500 to 13,500 per mm^3 and often varies with each disease. An increase in WBCs, or leukocytosis, is not specific to one white blood cell type. A decrease in number of WBCs is referred to as *leukopenia*. One or all the WBC elements may be decreased.

The determination of an inflammatory process is important for the implant dentist and may occur without leukocytosis. An increased count of band neutrophils indicates inflammation. Segmented neutrophils phagocytize bacteria and increase in times of infection. Therefore if a WBC count is ordered to determine whether infection around an implant is affecting the patient's overall health, a differential of WBC types is evaluated, not

WWW.HIGHDENT.IR

just the number of leukocytes present. WBC counts are critical to the dental outpatient, particularly for patients with immune diseases or undergoing chemotherapy. The counts can indicate infections, leukemic disease (myoproliferative), immune diseases, and toxicity of drugs (especially chemotherapeutic drugs). The two most critical groups are granulocytes (e.g., neutrophils, which fight infection) and lymphocytes (immune response). Absolute neutrophil count includes segs and bands and must be greater than 2000 for normal dental treatment without antibiotic prophylaxis. Levels of 1000 to 2000 need antibiotic coverage. Levels lower than 1000 mandate physician referral. Lymphocytes are necessary to evaluate the immune response potential of the patient. Many immunodeficient patients, including those with human immunodeficiency virus (HIV), may have no systemic symptoms, yet have deficient lymphocytes.[31]

Red Blood Cell Count

Red blood cells (RBCs) are responsible for the transport of oxygen and carbon dioxide throughout the body and for control of the blood pH. The cells represent the largest segment of the formed elements of the blood. The normal RBC count is higher in men than in women. Increases may result from polycythemia, congenital heart disease, or Cushing's syndrome. The most common finding is a decreased cell count usually indicating anemia.

Hemoglobin

Almost 95% of the dry weight of an RBC is hemoglobin. It is responsible for the oxygen-carrying capacity of the blood. The normal level of hemoglobin is 13.5 to 18 g/dL in men and 12 to 16 g/dL in women. The preoperative threshold of 10 g/dL is often used as a minimum baseline for surgery. However, many patients can undergo surgical procedures safely at 8 g/dL. The threshold is related to the underlying condition of the patient and the anticipated blood loss.[25]

Hematocrit

The hematocrit represents the packed cell volume and indicates the percentage of RBCs in a given volume of whole blood (RBCs/100 mL of blood). It is the prime indicator of anemia or blood loss. Therefore the hematocrit is evaluated if one of these conditions is suggested. Values within 75% to 80% of normal are required before sedation or general anesthesia.

Bleeding Tests

Bleeding disorders are one of the most critical conditions encountered in surgery. The platelet count may reflect this complication, but it does not provide enough information to determine potential bleeding disorders. The patient's medical history is a better detector (see Figure 20-1).

Understanding the normal clotting process determines which bleeding test to evaluate. Whenever the integrity of a vessel wall is altered surgically, hemostasis is achieved by vascular spasm, blood coagulation, and, finally, growth of fibrous tissue to close the defect in the vessel. For hemostasis to be maintained or achieved, the blood vessels must be normal, functional platelets must be present in sufficient number, and the coagulation mechanism of the blood must be intact. The three phases necessary for hemostasis involve the vessels, the platelets, and coagulation—both extrinsic (outside blood vessels) and intrinsic (inside blood vessels).

In a normal, healthy individual, coagulation is initiated within 20 seconds of blood vessel damage. This is accomplished by two phases of hemostasis: primary and secondary. Primary hemostasis is initiated when platelets adhere to collagen fibers in the vascular endothelium. The platelets are then activated, which results in additional platelet activation. The platelets undergo a change in shape, which exposes a phospholipid surface for platelets to link together via glycoprotein. The secondary phase consists of a coagulation cascade that has two pathways (Figure 20-2): contact activation pathway (formerly the intrinsic pathway) and the tissue factor pathway (formerly the extrinsic pathway). These pathways involve a series of enzymatic reactions in which an inactive coagulation factor is converted to an active form, which then activates the next coagulation factor in the series. The process involves a series of reactions in which the enzyme and substrate are localized on the activated platelet surface. In addition to the clotting factors, cofactors such as calcium and vitamin

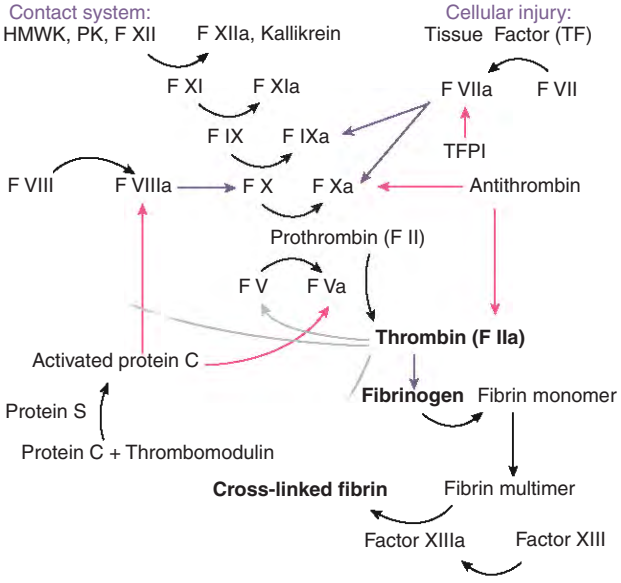


Figure 20-2 The secondary phase of hemostasis is a coagulation cascade.

WWW.HIGHDENT.IR

K are involved that are necessary for the synthesis of additional clotting factors. The tissue factor pathway main role is for the generation of thrombin via various reactions precipitated by various coagulation factors. The contact activation pathway uses various coagulation factors that form the primary complex on collagen. The end result is the formation of thrombin, which helps to convert fibrinogen to fibrin, which is the building block of the hemostatic plug.[32]

The tissue factor pathway (extrinsic system) and the contact activation pathway (intrinsic system) lead to completion of hemostasis along a common pathway. Both systems are necessary for normal coagulation. The extrinsic system is activated outside the blood vessels; the intrinsic system is activated within the blood vessels.

Three ways to detect potential bleeding problems are: (1) check the medical history, (2) review the physical examination, and (3) screen the clinical laboratory tests.[33] More than 90% of bleeding disorders can be diagnosed on the basis of the medical history alone. The history should include the following questions covering five topics entered on the medical history form:

1. Familial history of bleeding disorders
2. Spontaneous bleeding from the nose, mouth, or other apertures
3. Bleeding problems/bruising after operations, tooth extractions, or trauma
4. Use of medications that may cause bleeding disorders
5. Past or present illness associated with bleeding disorders

Medical History

Bleeding problems in relatives are significant because they indicate inherited coagulation disorders. Hemophilia and Christmas factor disease are the most common. Spontaneous bleeding from the nose, gingiva, or joints may indicate either inherited disease or illness with bleeding disorders (e.g., acute leukemia). If there were no bleeding problems in a previous surgery, no acquired disorders were present at that time, and no significant inherited disorder is present now. Medications that cause increased bleeding usually do so because of platelet disorders. These iatrogenic factors are seen primarily with three types of drugs: anticoagulants, aspirin, and long-term antibiotics. Past or present illnesses that may present associated bleeding disorders include leukemia, anemia, thrombocytopenia, hemophilia, and hepatic diseases. Approximately half of the patients with liver disease have a decrease in platelets secondary to hypersplenism, and therefore have potential bleeding disorders.[34]

Physical Examination

The second method by which the implant dentist can detect a patient with a bleeding disorder is the physical examination. The exposed skin and oral mucosa must be examined for objective signs. Petechiae, ecchymoses, spider angioma, or jaundice may be observed in liver disease patients with bleeding complications. Intraoral petechiae, bleeding gingiva, ecchymoses, hemarthroses, and hematomas may be present in patients with genetic bleeding disorders. Patients with acute or chronic leukemia show signs of oral mucosa ulceration, hyperplasia of the gingiva, petechiae or ecchymoses of the skin or oral mucosa, or lymphadenopathy.

Clinical Laboratory Testing

The third option to detect a bleeding disorder is clinical laboratory testing. If a patient's health history and physical examination do not reveal potential bleeding disorders, routine screening with a coagulation profile is not indicated. However, if extensive surgical procedures are expected, a coagulation profile is indicated. Four tests are used to screen patients for bleeding disorders and measure both pathways of the coagulation cascade as well as platelet function and capillary fragility. These include the platelet count, bleeding time, partial thromboplastin time (PTT), and international normalized ratio (INR) (formerly prothrombin time [PT]). A thrombin clotting time and fibrinogen level may be added on occasion.

International Normalized Ratio and Prothrombin Time

The most appropriate tests to evaluate patients that are on anticoagulant therapy are the INR and PT. These tests measure the effectiveness of the tissue factor pathway (extrinsic system) and common pathways of coagulation. In the past, PT values were used exclusively; however, because of the variability in laboratory reporting, the World Health Organization developed a more standardized system, the INR. In a normal individual, the INR value should be 1.0. The recommended therapeutic range of continuous anticoagulation is an INR between 2.0 and 3.0 for all conditions except artificial heart valves, for which the INR should be between 2.5 and 3.5 (Table 20-2).[35]

Partial Thromboplastin Time

Another test for coagulation is the PTT, which measures the contact activation pathway (intrinsic system) and

Table 20-2 Target International Normalized Values for Specific Medical Conditions

PATIENT CONDITION	INR VALUE
Normal	1.0
Prevention of myocardial infarction	2.0-3.0
Treatment of pulmonary embolism	2.0-3.0
Treatment of atrial fibrillation	2.0-3.0
Pulmonary embolism	2.0-3.0
Prosthetic heart valves	2.5-3.5
Prevention of venous thrombosis	2.5-3.5

INR, International normalized ratio.

WWW.HIGHDENT.IR

common pathways. A normal test time is 25 to 40 seconds. This test is usually used to measure the effectiveness of the anticoagulant heparin. It may also be used to evaluate the long-term affects of daily aspirin ingestion.

Bleeding Time

The bleeding time test is used to evaluate platelet function. The Ivy method is the standardized test usually used. In this test, a blood pressure cuff is placed on the upper arm and inflated to 40 mm Hg. A lancet or scalpel blade is used to make a cut on the underside of the forearm, and the time is measured until bleeding has stopped. Normal values fall between 1 and 6 minutes depending on the method used. The bleeding time measures both coagulation pathways and platelet function and capillary activity.

Platelet Count

The platelet count is part of the CBC and is usually in range of 140,000 to 340,000/µL of blood. This test identifies the number of platelets (thrombocytes), which are vital to the formation of the blood clot. If the count falls below 100,000/µL, the patient is said to have thrombocytopenia, which can be significant for bleeding problems in implant patients. If the count level is below 20,000/µL, spontaneous bleeding may occur, which can be life-threatening. Low blood counts may be caused by stomach ulcers, autoimmune diseases, alcohol, and chemotherapeutic medications.[36]

The PTT is used to determine the ability of the blood to coagulate within the vessels. Therefore it tests the intrinsic and common pathways of coagulation (factors VII through XII). The PT/INR determines the ability of the blood to coagulate outside the vessels and therefore tests the extrinsic and common pathways of coagulation. Both systems are necessary for normal coagulation. The patient's history and physical examination can now be related to the appropriate clinical laboratory tests.[37,38]

Heparin is the anticoagulant usually prescribed for renal dialysis. A PTT test should be scheduled for the day of surgery. Heparin is a short-acting anticoagulant; therefore this test and the surgery should be scheduled 24 hours or more after heparin administration. Four to six hours without heparin are usually adequate to return coagulation to near normal levels. Kidney dialysis patients often experience healing and maintenance complications with their natural teeth; implants are usually contraindicated in these patients. However, the dentist may have to treat a dialysis patient who has previously had implant therapy. If the PTT is more than 1.5 times the normal value, surgery should be postponed.

Long-term antibiotic therapy can affect the intestinal bacteria that produce the vitamin K necessary for prothrombin production in the liver. Therefore if long-term administration of antibiotics has been used by the implant patient, a PT should be obtained to evaluate possible bleeding complications.

Bleeding disorders should be suspected in a patient who is an alcoholic or shows signs of liver dysfunction. The liver is the primary site of synthesis of the vitamin K–dependent clotting factors II, VII, IX, and X. Alcoholism, independent of liver disease, has been shown to decrease platelet production by megakaryocytes and increased platelet destruction. Most coagulation factors are produced in the liver; 50% of patients with liver disease have hypersplenism resulting from the destruction of platelets. The PT is the single most useful test used to evaluate impaired hepatocyte synthesis of prothrombin complex factors and to assess hemostasis in patients with liver disease. Factor VII has the shortest half-life and is the first to decrease. Factor VIII and von Willebrand factor have a tendency to increase in chronic hepatic disease patients. The bleeding time and PT therefore should be evaluated.

The PT and PTT may be used together to determine coagulation factor defects. A normal PT and abnormal PTT suggest hemophilia. An abnormal PT and a normal PTT suggest factor VII deficiency. If both PT and PTT are longer, a deficiency of factors II, V, or X or fibrinogen are considered.

No surgical procedures should be performed on a patient suspected of having a bleeding problem based on history, examination, and clinical laboratory tests without proper preparation, understanding, and concerted management by the dentist and the physician. If the bleeding disorder has been previously undiagnosed, the underlying cause should be addressed before the elective implant surgical procedures begin.

Biochemical Profiles (Serum Chemistry)

The tenets of laboratory diagnosis should be understood, particularly as they relate to implant dentistry. The interpretation of biochemical profiles and the ability to communicate effectively with medical consultants will enhance the treatment of many patients.

The decision to proceed with oral implant treatment may be affected by the results of biochemical profiles by contraindicating the procedure completely, altering the type of implant surgery and reconstruction, postponing the treatment until therapy controls the disease entity, or simply changing the sequence of medications normally used during treatment. Biochemical sanguine profiles are a more necessary part of the medical evaluation for an implant candidate in the presence of systemic diseases or advanced surgical procedures.[39] They are not indicated for every potential implant patient.

1. The BMP, which has replaced the tests SMA 7 and Chem 7, is a group of seven to eight tests that gives the current status of the kidneys, electrolyte and acid/base balance, blood sugar, calcium levels, and monitoring of hypertension.

WWW.HIGHDENT.IR

Table 20-3 Laboratory Evaluation of Disease Indicators

CHEMISTRY	DISEASE
Glucose	Diabetes, steroid dysfunction
Calcium	Renal disease, diet, bone diseases, (carcinoma, parathyroid disease, Paget's disease)
Inorganic phosphorus	Renal disease, endocrine (parathyroid, thyroid, steroids), antacids
Alkaline phosphatase	Liver disease, bone diseases (Paget's disease, metastases, fractures, hyperparathyroidism)
Lactic dehydrogenase	Hemolytic disorders, liver disorders, myocardial infarction
Creatinine	Renal function
Bilirubin	Liver disease

2. The CMP (also Chem 12, SMA 12, or SMA 20) is a group of 14 specific tests that is used as a broad screening tool to evaluate organ function and conditions such as diabetes, liver disease, kidney disease, and hypertension.

Dentists have been trained to take a careful medical history, perform a thorough dental examination, and, on occasion, order laboratory tests that will either confirm or exclude a provisional diagnosis. However, with biochemical profiles such as the BMP and CMP, there is the opportunity to evaluate different biochemical parameters reflecting the patient's health.

To be comfortable in interpreting the biochemical profile, some time must be spent in learning the BMP/CMP pattern of systemic diseases. This pattern recognition is similar to the tissue patterns a pathologist looks at during a biopsy. The BMP/CMP profile has been described as a "biochemical biopsy" of the blood. It includes normal and abnormal values that have interrelationships in the diagnosis of systemic diseases. It is not wise to single out one value to establish a diagnosis. The data should be related with other values obtained in the profile before further determinations are rendered.

More than 20 different tests are available for the selection of this profile from which each laboratory and its referring physicians choose. Therefore the use of different laboratories makes it difficult to develop interpretative skills. The dentist should use the same laboratory consistently, highlighting the abnormal values for an individual test. The dentist is not responsible for treating the many systemic diseases that the biochemical profile reveals. However, it is necessary to know the effects of a disease on the implant treatment.

Normal Range

The normal values found on the BMP/CMP represent a statistical norm. Any population characteristically shows a bell-shaped curve for a particular measurement. It has been shown that 56% of a sample fall within one standard deviation of the mean and 95% are within two standard deviations. The normal value in the biochemical profile represents two standard deviations. Thus "normal" in the statistical sense does not necessarily mean healthy; the word merely describes the typical range of values expected in any given population. Approximately 1 in 20 results will be outside the two standard deviation ranges. The further from the average value a particular value falls, the more certain its clinical significance. Different laboratories can have different normal results.

As biochemical profiles are compiled for an individual patient over several years, the deviation in a given test may indicate a radical change for that individual, although the result never deviates from the normal population range. The implant dentist should remember that the healthy patient of today may have a systemic disease in the future. Therefore when evaluating long-term complications, it is of interest to relate a recent biochemical profile to the one first reviewed before the initial surgery.

The patient should fast before the blood is collected to avoid artificial elevations of blood glucose and depressed inorganic phosphorus. Most of the other elements of the profile will not be affected. This chapter will limit discussion to the factors of most benefit to the implant dentist (Table 20-3): glucose, calcium, inorganic phosphorus, alkaline phosphatase, lactic dehydrogenase, creatinine, and bilirubin.

Serum Glucose

The normal range of glucose found in the blood, 70 to 100 mg/mL, is maintained within fairly narrow limits. Hyperglycemia is a relatively common finding; the most common cause of this condition is diabetes mellitus. If high glucose values are found, referral to a physician for glucose tolerance tests after a glucose loading dose is recommended, except when inorganic phosphorus values are depressed. Depressed inorganic phosphorus usually indicates the patient has eaten before the blood was drawn and the elevated glucose is then related to digestion. Cushing's disease and other conditions related to excess production of adrenal corticoids are also considered in the differential diagnosis with hyperglycemia. Hypoglycemia is unusual and is related to varied causes (e.g., Addison's disease, bacterial sepsis, excessive insulin administration).

Serum Calcium

The implant dentist may be the first to detect diseases affecting the bones. Chemical confirmation is dependent on the patient's serum levels of calcium, phosphorus,

WWW.HIGHDENT.IR

and alkaline phosphatase. In the body, more than 98% of the calcium is stored in the skeleton and teeth. Calcium ions are responsible for neuromuscular excitability, normal blood coagulation, and the activation of several enzymes. Serum calcium levels are influenced by the parathyroid hormone and calcitonin. Serum calcium levels are increased by bone resorption, intestinal absorption, and renal reabsorption of calcium.

Decreased levels of calcium are primarily seen in hypoproteinemic conditions and in renal disease. Renal disease is much more common, but the diet of the potential implant patient may be severely affected by the lack of denture comfort and stability. The cause and treatment of hypocalcemic serum levels should be addressed before implant reconstruction.

Elevated levels of serum calcium are associated with carcinoma in bones, dietary or absorptive disturbances, and hyperparathyroidism. The osteoporosis that accompanies this disorder has been observed in the mandible. Hyperparathyroidism also causes hypophosphatemia. Hypercalcemia associated with a significant elevation of alkaline phosphatase suggests Paget's disease of bone. All other biochemical values being normal, an elevated calcium value may be the result of laboratory error.[40] If phosphorus or alkaline phosphatase levels are also affected, medical evaluation and treatment are indicated before implant surgery.

Inorganic Phosphorus

Inorganic phosphorus levels maintain a ratio of 4:10 compared with calcium, and there is usually a reciprocal relationship. When the level of one increases, the other decreases. The common cause of elevated phosphorus is chronic glomerular disease, indicated by elevated blood urea nitrogen (BUN) and creatinine values.

In the absence of significant glomerular disease (normal BUN and creatinine values), phosphorus level abnormalities are usually associated with the endocrine system or bone metabolism. If an increase in phosphorus is associated with a decrease in calcium and normal renal function, hypoparathyroidism is suspected. Other endocrine disorders associated with an increased phosphorus level include hyperthyroidism, increased growth hormone secretion, and Cushing's syndrome.

Decreased levels of phosphorus may appear in hyperparathyroidism patients, especially when it is associated with hypercalcemia. The chronic use of antacids containing aluminum hydroxide also may induce hypophosphatemia and warrants investigation for a peptic ulcer.

Alkaline Phosphatase

Serum alkaline phosphatase determination helps evaluate hepatobiliary and bone diseases. The normal levels for this enzyme in adults are derived primarily from the liver; extremely high levels of alkaline phosphatase are often associated with hepatic disease.

In the absence of liver disease, elevations of alkaline phosphatase are often a sign of osteoblastic activity in the skeletal system. Therefore bone metastases, fractures, Paget's disease, and hyperparathyroidism increase the level of this serum enzyme. Serum alkaline phosphatase is normal in patients with adult osteoporosis. Low levels of alkaline phosphatase are usually not of clinical significance for the dentist.

Lactic Dehydrogenase

Lactic dehydrogenase (LDH) is an intracellular enzyme present in all tissues (normal 0 to 625 U/L). False elevated LDH levels occur as a result of hemolyzed blood specimens. Therefore if all other blood values are normal, LDH testing should be repeated before further investigation. The highest elevations of this enzyme are seen in patients with myocardial infarction, hemolytic disorders such as pernicious anemia, and liver disorders. Lactic dehydrogenase, aspartate aminotransferase (AST), bilirubin, uric acid, and total protein are indicators of hematologic and reticuloendothelial disorders. Blood disorders are particularly important to the implant surgeon. When LDH values are elevated, the complete blood count is evaluated for blood abnormalities.

Creatinine

Creatinine is an anhydride of creatine used in muscle metabolism (normal 0.7 to 1.5 mg/dL). It is freely filtered by the glomeruli and not reabsorbed. The constancy of formation and excretion permits creatinine levels to be an index of renal function. This important system should not be impaired during implant surgery. Kidney dysfunction may lead to osteoporosis and decreased bone healing because the kidney is required for complete formation of vitamin D. Medications can alter pharmacokinetics, and normal healing can also be affected by kidney disease.

Bilirubin

Within the CMP, seven tests are indicative of liver function (total protein, albumin, cholesterol, bilirubin, LDH, alkaline phosphatase, and AST). Primary hepatic disease is reflected in elevations of bilirubin and enzymes such as serum glutamic-oxaloacetic transaminase. Therefore for the evaluation of liver disease, bilirubin measurement is of primary importance. Approximately 80% of the bilirubin comes from the degradation of red blood cells. The liver is responsible for hundreds of chemicals and proper body functions. The function of this organ should be adequate for proper healing, drug pharmacokinetics, and long-term health.

Glycosylated Hemoglobin

Glycosylated hemoglobin, also termed hemoglobin A1c or HbA1c, is a test to monitor the control of the diabetic patient over the previous 2 to 3 months. This test measures the percentage of hemoglobin bound to glucose.

WWW.HIGHDENT.IR

Table 20-4 HbA1c Values versus Blood Glucose Levels

HBA1C (%)	AVERAGE BLOOD SUGAR (mg/dL)
6	120
7	150
8	180
9	210
10	240
11	270
12	300

Hemoglobin, a protein in red blood cells, binds to the HbA1c form of hemoglobin by a process termed *glycosylation*. The percent HbA1c reflects how much glucose is bound to the blood during the past 120-day life span of the RBCs. Glycosylated hemoglobin helps to minimize complications in patients exhibiting chronically elevated glucose levels. For nondiabetic patients, normal values are approximately 4.0% to 6.0%. Ideally, a diabetic patients will have their HbA1c at approximately 6%, which corresponds to an average glucose of 135 mg/dL (7.5 mmol/L). For every 1% change in the HbA1c, a resultant change of about 30 mg/dL (1.67 mmol/L) will be seen (Table 20-4).

SYSTEMIC DISEASE AND ORAL IMPLANTS

Systemic diseases play a vital role in treatment planning and implant therapy for patients. There are specific systemic diseases and conditions that undeniably affect bone metabolism, wound healing, and ultimately the success of implant therapy. The implant dentist should use this information on systemic conditions, which have a significant impact on patient selection and management. Some conservative surgery and prosthetic implant procedures are rarely contraindicated based on systemic conditions; other complex treatments must be excluded for the same condition. It is the responsibility of the implant dentist to understand the interrelationship of systemic diseases and implant dentistry. Common conditions that may affect the implant treatment are discussed in three steps. The first section describes the entity in general. The second section discusses dental implant treatment implications. The last section reviews dental implant management.

American Society of Anesthesiologists Physical Status Classification

Systemic diseases have a wide range of effects on a patient, depending on the severity of the disease. There are relatively few systemic diseases that always contraindicate implant surgery or prosthetics. However, several metabolic disorders have contraindications when the conditions are out of control or severe. In 1962 the American Society of Anesthesiologists (ASA) adopted a classification system for the severity of disease, and the system is widely used. It was designed to estimate the medical risk presented by a patient receiving general anesthesia for a surgical procedure. The classification system is valuable for determining any medical risk regardless of the method of anesthesia or type of surgery (Box 20-1).

Box 20-1 American Society of Anesthesiologists (ASA) Classifications

ASA I: A normal, healthy patient, without systemic disease.
ASA II: A patient with mild to moderate systemic disease.
ASA III: A patient with severe systemic disease, which limits or alters activity but is not incapacitating.
ASA IV: A patient with severe systemic disease, which is incapacitating and is a constant threat to life.
ASA V: A moribund patient not expected to live more than 24 hours without an operation.

Elective implant surgeries are not indicated for ASA IV or V patients.

The systemic conditions listed in this chapter are the most commonly observed in an implant practice; it is not intended that all conditions be included. The diseases discussed are classified as mild, moderate, or severe. A disease entity affects the host with varied intensity. Therefore mild diabetes may permit implant treatment, but the same disease in the severe form may contraindicate most implant therapy. As a result, a mild diabetic patient should be treated differently than the severe diabetic patient. A general sedation and treatment format may be established with most expressions of systemic diseases.

In addition to the range of disease expression, Misch has presented a variety of implant treatments delivered to a patient.[39,39a] In Table 20-5, four levels of surgical and prosthetic dental treatments are established. A systemic condition may contraindicate one class of treatment, yet a more simple implant procedure can still be performed. The four levels of treatment range from noninvasive procedures with little or no risk of gingival bleeding to those that are most complicated and invasive.

Type 1 procedures can be performed on most patients regardless of systemic condition. Type 2 procedures are more likely to cause gingival bleeding or bacterial invasion of the bony structures. Type 3 procedures are surgical procedures that require more time and technique. Type 4 procedures are advanced surgical procedures with more bleeding and greater risk of postoperative infection and complications.

WWW.HIGHDENT.IR

Table 20-5 Classifications of Dental Treatment

CLASSIFICATION	TREATMENT
Type 1	Examinations, radiographs, study model impressions, oral hygiene instruction, simple extractions, single-tooth implants, stage 2 uncovery with minimum tissue reflection, simple restorative dentistry
Type 2	Multiple simple extractions, multiple implants with minimum tissue reflection
Type 3	Difficult extractions, multiple root forms, ridge augmentation, unilateral sinus graft, unilateral subperiosteal implants with quadrant periosteal reflections
Type 4	Full-arch implant (complete subperiosteal implants, ramus frame implants, full-arch endosteal implants), orthognathic surgery, autogenous block bone augmentation, bilateral sinus graft

A relationship can be established between the severity of the disease (mild to severe) and the maximum involvement of the dental implant procedure (Table 20-6). For the more extensive procedure, the patient should be healthier; for a more severe form of the disease, the surgical procedure should be less invasive.

Cardiovascular Diseases

Hypertension

Hypertension is the most common primary diagnosis in the United States and accounts for more than 35 million health care visits per year. It affects 50 million Americans and approximately 1 billion people worldwide.[26,41] There are an estimated 7.1 million deaths per year attributable to hypertension, along with 62% of cerebrovascular disease and 49% of ischemic heart disease.[42] Approximately 30% of adults with hypertension are unaware that they have hypertension, and two thirds of patients treated are not controlled to blood pressure less than 140/90 mm Hg. Untreated, undiagnosed, and uncontrolled hypertension is a serious problem in society today.

Table 20-7 Blood Pressure Classification for Adults*

BLOOD PRESSURE CLASSIFICATION	SYSTOLIC BLOOD PRESSURE (mm Hg)	DIASTOLIC BLOOD PRESSURE (mm Hg)
Normal	< 120	and <80
Prehypertension	120-139	or 80-90
Stage 1 hypertension	140-159	or 90-99
Stage 2 hypertension	≥160	or ≥100

Adapted from Chobanian AV, Bakris GL, Black HR et al: Seventh report of the National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, *Hypertension* 42:1206, 2003.
*Treatment determined by highest blood pressure category.

With increasing age, the prevalence of hypertension increases. More than half of people aged 60 to 69 years and approximately three quarters of those age 70 and older are affected with hypertension.[2] A recent study showed that the lifetime risk of hypertension is 90% for men and women who were nonhypertensive at age 55 to 65 and live to age 80.[43] Because implant dentists treat a high percentage of elderly patients, coupled with the high prevalence in the general population, prevalence of patients that have uncontrolled or undiagnosed hypertension is very high. Failure to diagnose and detect hypertensive patients can result in life-threatening conditions such as stroke or myocardial infarction.

Recently, the Joint National Committee on Hypertension published an updated classification of blood pressure for adults.[44] This classification is based on the average of two or more seated blood pressure readings on each of two or more office visits (Table 20-7).

The Joint National Committee on Hypertension introduced a new category termed *prehypertension*. Patients in this category are at risk of developing

Table 20-6 Evaluation of Risk in Systemic Diseases

RISK	ASA CATEGORY	TYPE 1	TYPE 2	TYPE 3	TYPE 4
Normal	I	+	Sedation, SRP	Intravenous sedation	SRP
	II	+			
Mild	II	+	Sedation, SRP	Intravenous sedation	SRP
Moderate	II	+	Intravenous sedation, SRP, physician consultation	Hospitalization	Hospitalization
Severe	III	+	Postpone all elective procedures	Postpone all elective procedures	Postpone all elective procedures

ASA, American Society of Anesthesiologists; *SRP,* stress-reduction protocol; +, procedure may be performed with regular protocol after physician consultation.

WWW.HIGHDENT.IR

hypertension and are encouraged to make a lifestyle modification to reduce this risk. Additionally, the previous stage 2 and stage 3 have been combined to form Stage 2 hypertension. In patients younger than 50 years of age, diastolic hypertension predominates as a leading indicator. However, in patients older than age 50, the systolic is a better indicator of hypertension as systolic pressure rises with age. Contrary to past knowledge that diastolic pressure is more important than systolic pressure, studies have shown that with the aging population, uncontrolled systolic hypertension causes increased rates of cardiovascular and renal diseases.[44] Risk factors for hypertension include obesity, high sodium intake, poor diet, lack of exercise, alcohol consumption, and smoking. Special attention should be given to patients with a history of obstructive sleep apnea. Sleep apnea has been associated with a number of cardiovascular diseases including dysthymias, myocardial infarction, and stroke.[45] Greater than 50% of patients with sleep apnea also have hypertension.[46]

Hypertension is usually asymptomatic and is the major risk factor for coronary heart disease and cerebrovascular accidents leading to cardiovascular morbidity and mortality for people older than 50 years of age. Patients with stage 1 and stage 2 hypertension are susceptible to three times as much coronary disease, four times as much cardiac failure, and seven times as many strokes as normotensive patients. The medical history should focus on predisposing factors such as excessive alcohol intake, history of renal disease, stroke, other cardiovascular diseases, diabetes, obesity, and smoking.

Stage 1 and stage 2 hypertension is usually treated with antihypertensive medications, many of which have an impact on implant therapy because of their numerous side effects. These include orthostatic hypotension, dehydration, sedation, xerostomia, and depression. The side effects may alter treatment or require special precautions. For example, orthostatic hypotension affects a patient brought from a supine to an upright position, which can result in syncope and falling with possible injuries. The patient may feel lightheaded or even faint; these symptoms can be avoided by allowing patients to sit upright for several minutes after the completion of their dental procedure. Patients at high risk include the elderly, those with anxiety, patients taking multiple medications, and those who have undergone lengthy dental procedures.

Box 20-2 Supplements Associated with Increased Swelling and Increased Blood Pressure

- Celery
- Dandelion
- Elder
- Goldenseal
- Guaiacum
- Juniper

Calcium channel blockers used to treat hypertension or congestive heart failure can cause gingival hyperplasia around teeth or implants (similar to Dilantin) and have been linked to erythema multiforme and other types of oral ulceration.[47] Gingival overgrowth can result in pain, gingival bleeding, and difficulty in mastication. The incidence is approximately 1.7% to 3.8% of patients taking calcium channel blockers.[48] A newer agent blocking angiotensin II seems to exhibit no dental implications.[49]

The use of NSAIDs has been shown to lessen the effectiveness of various antihypertensive medications. Of the 50 million patients being treated with antihypertensive therapy, 12 million use NSAIDs concomitantly.[50,51] However, short-term use of NSAIDs has not been shown to have a clinically significant effect.[52]

In addition, severe hypertension can lead to angina pectoris, congestive heart failure, or even a cerebrovascular episode, which can be precipitated by a rapid increase in blood pressure during an injection or surgery. Myocardial infarction, retinal hemorrhage, and kidney failure are other serious complications.

When a patient indicates hypertension on the medical history form, the date of the diagnosis, the complications, and the medications should be reviewed. The former guidelines for hypertension therapy followed a stepped-care approach that permitted the patient to use the medications as a good indicator of the severity of the disease; single diuretic drugs are usually used to treat mild hypertension, whereas combination drugs indicate a more severe hypertension. Patients taking additional drugs such as clonidine exhibit more severe hypertension[53] and require a medical consultation before undergoing complicated implant surgery procedures.

Dental Implant Management

Because a high percentage of patients have hypertension, the dentist and staff members must be knowledgeable about the measurement, detection, and treatment of hypertension. The accurate measurement of blood pressure, along with a review of all medications including herbal and over-the-counter medications, should be an integral part of the implant consultation and examination (Table 20-8; Box 20-2).[54]

An increased blood pressure is not uncommon in the dental office setting, because stress associated with treatment (also termed *white coat syndrome*) leads to increased levels of catecholamine, which causes an increase in blood pressure and heart rate. Two important steps to decrease the stress in the dental office are a well-monitored stress reduction protocol and proper management of pain and discomfort. Stress reduction protocol may include premedication on the night before the appointment (flurazepam [Dalmane] 30 mg or diazepam [Valium] 5 to 10 mg), setting an early morning appointment, minimizing waiting room time, and

WWW.HIGHDENT.IR

Table 20-8 Recommendation for Blood Pressure Evaluation

INITIAL BLOOD PRESSURE	RECOMMENDATION	IMPLANT THERAPY
Normal	Recheck yearly	No contraindications
Prehypertension	Recheck every 6 months	No contraindications
Stage 1 hypertension	Recheck at next visit	If blood pressure remains high, patient is receiving antihypertensives, or there is previous organ damage, initiate medical consultation; stress reduction protocol
Stage 2 hypertension	Recheck at next visit; if unchanged, initiate medical consultation	Medical consultation Stress reduction protocol

Table 20-9 Dental Implant Management in Hypertensive Patients

RISK	SYSTOLIC (mm Hg)	DIASTOLIC (mm Hg)	TYPE 1	TYPE 2	TYPE 3	TYPE 4
Normal	<120	<80	+	+	Sedation	Sedation
Prehypertension	120-139	80-89	+	+	Sedation	Sedation
Hypertension						
Stage I	140-159	90-99	+	Sedation	Sedation	Sedation
Stage II	≥160	≥100	+	Sedation after physician consultation	Postpone all elective procedures	Postpone all elective procedures
	>180	>110	Refer and postpone all elective procedures			

+, May be performed with regular protocol after physician consultation.

ensuring the duration of treatment does not exceed the patient's limits. Adequate pain control is also important including preemptive analgesia, profound anesthesia during the procedure, and sufficient postoperative pain control including long-acting anesthetics (Table 20-9). A resting systolic pressure greater than 180 or a diastolic pressure greater than 110 should indicate that all elective procedures be delayed until blood pressure may be reduced to a safer level.

Angina Pectoris

Angina is defined as chest pain as a consequence of exertion and alleviated by rest. Angina pectoris is a form of coronary heart disease that is usually caused by arteriosclerotic heart disease; however, it may be caused by coronary artery spasm, severe aortic stenosis, aortic insufficiency, anemia, emboli, and hereditary connective tissue disease. The cause of angina is a discrepancy between the myocardial oxygen demand and the amount of oxygen being delivered through the coronary arteries.

The risk category for the typical angina patient is ASA III, and unstable angina or a recent onset may represent ASA IV risks. The classical symptom of retrosternal pain often develops during stress or physical exertion, radiates to the shoulders, left arm, or mandible or to the right arm, neck, palate, and tongue; these symptoms are relieved by rest.[55] Episodes of stable angina (exertional angina) last 3 to 5 minutes after the stress or exercise is halted. Intensity may vary from mild to severe.

Patients with a history of angina may be taking long-acting nitrates to prevent the occurrence of acute episodes. Sublingual or spray nitroglycerin is recommended for the treatment of acute episodes. When retrosternal pain occurs, myocardial infarction is part of the differential diagnosis. This pain is similar in region, but is more intense, does not cease within 3 to 5 minutes, and is not relieved by nitroglycerin. Risk factors for angina pectoris are smoking, hypertension, high cholesterol, obesity, and diabetes.

If a patient reports a history of angina, the severity of the disease is evaluated by the last attack, change in frequency, frequency and severity of attacks, and the medications prescribed. As with hypertension, the disease may be classified as mild, moderate, or severe (Table 20-10). The patient with mild angina may have one or fewer attacks each month; the patient with moderate angina may suffer infrequent but predictable attacks, even in the absence of excessive stress or exertion; patients with severe unstable angina have almost daily attacks, often with increasing severity. Recent-onset angina (less than 60 days) is classified as unstable (ASA IV).

The major concern for the implant dentist is the precipitation or management of the actual angina attack.

WWW.HIGHDENT.IR

Table 20-10 Dental Implant Management in Patients with Angina Pectoris

RISK		TYPE 1	TYPE 2	TYPE 3	TYPE 4
Mild	≤1/month; ASA II	+	+	Sedation supplemental oxygen	
Moderate	≤1/week; ASA III	+	Sedation, premedication, nitrates, supplemental oxygen	Sedation, premedication, nitrates, supplemental oxygen	Premedication, sedation, outpatient hospitalization
Severe	Daily/more; ASA IV; unstable	+	Physician consultation	Elective procedures contraindicated	Elective procedures contraindicated

ASA, American Society of Anesthesiologists, +, may be performed with regular protocol after physician consultation.

Precipitating factors are exertion, cold, heat, large meals, humidity, psychological stress, and dental-related stress. All of these factors cause catecholamine release, which in turn increases the heart rate, blood pressure, and myocardial oxygen demand.[56]

Dental Implant Management

The dental emergency kit should include nitroglycerin tablets (0.3 to 0.4 mg) or sublingual nitroglycerine spray, which are replaced every 6 months because of their short shelf life. During an angina attack, all dental treatment should be stopped immediately. Nitroglycerin is then administered sublingually and 100% oxygen is given at 6 L/min, with the patient in a semisupine or 45-degree position.

Vital signs should be monitored after nitroglycerin is administered because transient hypotension may occur. If the systolic blood pressure falls below 100 mm Hg, the patient's feet should be elevated. If the pain is not relieved in 8 to 10 minutes with the use of nitroglycerin at 5-minute intervals, emergency medical assistance should be initiated.

Patients with mild angina (up to one attack per month) may undergo most nonsurgical dental procedures performed with normal protocol (type 1). General cardiac precautions are advised, such as vital signs monitoring, and patients are instructed to bring their own nitroglycerin. Advanced restorative procedures and minor implant surgery (type 2) are performed with nitrous oxide or oral sedation. For more advanced implant procedures (types 3 and 4), appropriate sedation techniques should be used. Appointments should be as short as possible. This may require more than one surgical or restorative appointment. Use of vasoconstrictors should be limited to a maximum of 0.04 to 0.05 mg epinephrine, and concentrations greater than 1/100,000 should be avoided.

Patients with moderate angina (up to one attack per week) tolerate examination and most simple operative procedures (type 1). Prophylactic nitroglycerin (0.3 to 0.4 mg) or long-acting nitrates are given sublingually just before advanced operative or simple to moderate implant surgery (types 2 and 3). Antianxiety sedation with supplemental oxygen is required. The use of even small amounts of vasoconstrictor is debatable; many claim it is contraindicated. Advanced surgical procedures may require a hospital setting (type 4).

Patients with unstable angina (daily episodes) are limited to examination procedures performed under normal protocol. Medical consultation is recommended for any additional treatment. This form of angina has been represented as an absolute contraindication for elective dental surgery (ASA IV).

The side effects of nitroglycerin are important to recognize because prophylactic administration is in order for the patient with moderate to severe angina. There is a decrease in blood pressure, which causes a decrease of the blood flow to the brain. Fainting is possible. Therefore the patient should be sitting or lying down during nitroglycerin administration. The heart attempts to compensate for the decreased blood pressure; the pulse rate may increase to as much as 160 beats/min. Blushing of the face and shoulders is common after administration of nitroglycerin. If the patient has been taking long-acting nitrates, tolerance to the drug may occur; therefore two tablets may be needed at a time. A headache may occur after administration, which may be treated with analgesics.

Myocardial Infarction

Myocardial infarction (MI) is a prolonged ischemia or lack of oxygen resulting from a deficiency in the coronary arterial blood supply that causes injury to the myocardium. The end result is cellular death and necrosis of the heart muscle. Approximately 1.3% of patients older than 30 and 10% of patients 40 years or older undergoing noncardiac surgery in a hospital setting indicate a history of previous MI.[57,58] An acute MI may be precipitated when the patient undergoes unusual stress, either physical (painful stimuli) or emotional (anxiety). The patient usually has severe chest pain in the substernal or left precordial area during an MI episode. It may radiate to the left arm or mandible. The pain is similar to angina pectoris, but more severe. Cyanosis, cold sweat, weakness, nausea or vomiting, and irregular and increased pulse rate are all signs and symptoms of MI.

The complications of MI include arrhythmias and congestive heart failure (CHF). The larger the ischemic

WWW.HIGHDENT.IR

Table 20-11 Dental Implant Management in Patients with Myocardial Infarction

RISK	TYPE 1	TYPE 2	TYPE 3	TYPE 4
Mild (>12 mo)	+	+	Physician	Physician Hospitalization if general anesthesia required
Moderate (6-12 mo; ASA III)	+	Postpone all elective procedures	Postpone all elective procedures	Postpone all elective procedures
Severe (<6 mo; ASA IV)	+	Postpone all elective procedures	Postpone all elective procedures	Postpone all elective procedures

ASA, American Society of Anesthesiologists; +, may be performed with regular protocol after physician consultation.

area, the greater the risk of heart failure or life-threatening arrhythmias. Any history of MI indicates significant problems in the coronary vessels. Recent infarctions correspond to higher morbidity and death rates with even simple elective surgery. The risk of MI is less than 1% in the general population in a perioperative setting. Approximately 18% to 20% of patients with a recent history of MI will have an increase of complications, which have a high mortality rate of 40% to 70%. If general anesthesia and surgery are performed within 3 months of MI, the risk of another MI is 30%; if within 3 to 6 months, it is 15%. After 12 months, the incidence of recurrent MI stabilizes at about 5%.[59]

Dental Implant Management

The dental evaluation should include the dates of all episodes of MI, especially the latest, and any complications. Medical consultation should preclude any extensive restorative or surgical procedure. Patients with an MI in the preceding 6 months may have dental examinations without any special protocol. Any surgical treatment should be postponed if possible for 6 months. Patients who experienced an MI 6 to 12 months preceding consultation may have examinations, nonsurgical procedures, and simple emergency surgical procedures performed after medical consultation. Longer procedures should be segmented into several shorter appointments whenever possible. Stress reduction protocol is indicated. Elective implant procedures should be postponed for at least 12 months after MI[60] and only performed after physician consultation. Elective hospitalization is an accepted modality for all advanced surgical procedures, regardless of the time elapsed after an MI; it is mandatory if general anesthesia is performed (Table 20-11).

Congestive Heart Failure

Congestive heart failure is a pathophysiologic state in which an abnormality in cardiac function is responsible for failure of the heart to pump blood in adequate volume to meet the needs of the metabolizing tissues.[61] More than 3 million people in the United States suffer from CHF, with approximately 400,000 new patients being diagnosed each year. Every year, 30% to 40% of patients with CHF are hospitalized, which accounts for the leading diagnosis-related group of hospitalized patients older than age 65.[62]

The heart pumps about 2000 gallons of blood per day to the other organs and body tissues. It coordinates the function of two pumps simultaneously: the left side, the larger of the two sides, pushes the blood out into the body; the right side sends the blood to the lungs for oxygenation. When the heart has been damaged, the blood begins to back up in the lungs or body. The heart will attempt to compensate by increasing the rate of contraction and stretching the muscle to accommodate a larger volume of blood to contract with a greater force and eject more blood (Starling's law). Both of these compensation attempts of the heart maintain circulatory needs in the short term, but they exact a long-term price. Less blood is circulated because, in beating faster, the heart is left with less time to refill while the extra effort increases the heart muscle's demand for oxygen. When this need is not met, the heart rhythms can become dangerously abnormal (arrhythmic) and lead to death.

Compensatory measures are taken by other parts of the body to counteract the insufficient blood circulation. The kidneys retain water and salt. In the case of heart failure, this only further strains the heart by increasing the amount of blood it must pump. At the same time, the retained fluid may seep into the body tissue. The patient's medical history (e.g., questions about the patient's ability to climb stairs) will give clues along with observation of signs and symptoms to congestive heart failure.

Symptoms of CHF are listed in Box 20-3; they include abnormal tiredness or shortness of breath (dyspnea) brought on by slight activity or even occurring at rest (these symptoms are due to excess fluid in the lungs and partly to the excess work required of the heart); wheezing caused by fluid in the lungs (pulmonary edema); peripheral edema or swelling of the ankles (pedal edema) and lower legs; frequent urination at night; jugular venous distension; sounds at auscultation (S_3 gallop); and paroxysmal nocturnal dyspnea (the

WWW.HIGHDENT.IR

Box 20-3 Symptoms of Congestive Heart Failure

- Abnormal tiredness
- Shortness of breath
- Wheezing
- Edema of legs or ankles
- Frequent urination
- Paroxysmal nocturnal dyspnea
- Excessive weight gain
- Orthopnea
- Pulmonary edema
- Jugular venous distention
- S_3 gallop

Box 20-4 Classification of Congestive Heart Failure (New York Heart Association)

I. No symptoms and no limitation in ordinary physical activity.
II. Mild symptoms and slight limitation during ordinary activity. Comfortable at rest.
III. Marked limitation in activity due to symptoms, even during less-than-ordinary activity. Comfortable only at rest.
IV. Severe limitations. Experiences symptoms even while at rest.

sensation of being unable to breath), which may interrupt sleep. This symptom is due to the effect of gravity on fluid that has spent the day down at the feet. As the fluid flows back up, it may pool in the lungs, causing a feeling of suffocation. Excessive weight gain, as much as 20 to 30 lb, with no change in diet is also a symptom. This increase, purely from fluid retention, gives some indication of how poorly the heart is pumping.

Although heart failure affects both sides of the heart, one side tends to be more weakened, so the condition is often referred to as left-sided or right-sided failure. Left-sided failure is most common. The fluid backs up into the lungs and brings on the symptom of breathlessness. Right-sided heart failure causes fluid backup in the veins that return blood to the heart, leading to swelling of the extremities and the liver.

Patients with CHF may be classified as to the progression of their disease. The New York Heart Association (NYHA) provides a simple way to classify the extent of heart failure. This classification places patients in one of four categories based on limitation during physical activity (Box 20-4).[63]

Medications prescribed for CHF are loosely classified as the *three Ds:* digitalis, diuretics, and dilators. Digitalis (digoxin; Lanoxin) increases the heart's pumping action (positive inotropic); diuretics (furosemide; Lasix) eliminate excess salt and water; and vasodilators (e.g., angiotensin-converting enzyme) expand the blood vessels so that pressure decreases and blood can flow more readily. Because digitalis is such a common drug for treating CHF, the doctor should be familiar with its common side effects.

A lethal dose of digitalis is only twice the treatment dose. A dentist recognizing the more common side effects (i.e., nausea, vomiting, anorexia) should report them to the treating physician. The heart rate is decreased (negative chronotropic) because the drug decreases the effect of the atrioventricular node (negative dromotropic) and stimulates the vagus nerve. Premature ventricular contractions are more common because the heart rate is decreased. Less common side effects (e.g., chromatopsia, spots, a halo around objects) may be reported. Trigeminal neuralgia symptoms may occur with use of digitalis. Although very uncommon, this condition has been reported to the author; a decrease of the medication dose partially relieves the symptoms.

Calcium channel blockers are also used to treat congestive heart failure and hypertension. Calcium is needed for atrial contraction. By blocking calcium, sodium remains available for ventricle contraction. Gingival hyperplasia (similar to that caused by Dilantin) has been reported to occur around teeth, implants, or superstructure bars of overdentures, especially with nifedipine. Other side effects such as edema and constipation have also been reported.

Dental Implant Management

In patients classified as NYHA I and II, no medical consultation is indicated unless there exist additional systemic diseases. In NYHA III and IV, medical consultation is highly recommended for all implant type 3 and 4 procedures. A comprehensive stress reduction protocol is indicated for all patients with CHF. Intraoperatively and postoperatively, pain and anxiety control is important because increased stress can produce an increased myocardial workload with an increase in the degree of heart failure.

Positioning of patients may require modification from the standard supine position. Patients with CHF may exhibit orthopnea, which may prevent the supine position. Patients should be placed in the most recumbent position in which they may breathe comfortably and efficiently.[52] Oxygen supplementation during implant procedures is highly recommended so as to minimize the possibility of hypoxia.

Subacute Bacterial Endocarditis and Valvular Heart Disease

Bacterial endocarditis is an infection of the heart valves or the endothelial surfaces of the heart. It is the result of the growth of bacteria on damaged or altered cardiac surfaces. The microorganisms most often associated with endocarditis following dental treatment are alpha-hemolytic *Streptococcus viridans* and less frequently

WWW.HIGHDENT.IR

staphylococci and anaerobes. The disorder is serious, with a mortality rate of about 10%.[53] Dental procedures causing transient bacteremia are a major cause of bacterial endocarditis. As a result, the implant dentist should identify the patient at risk and implement prophylactic procedures (Box 20-5).

The American Heart Association recently revised its recommendations of antibiotic prophylaxis for dental procedures in the prevention of infectious endocarditis. The new guidelines are based on the scientific studies showing that the risks of taking antibiotics outweigh the benefits for most patients. Adverse risks of taking antibiotics range from mild to sever allergic reactions along with development of drug-resistant bacteria. In addition, published studies have verified that the majority of patients are more at risk of developing infectious endocarditis from everyday activities than from dental procedures.[63a]

The recommendation states that patients who have routinely taken prophylactic antibiotics in the past are no longer required to do so. The new guidelines, however, are aimed at patients who are considered to be at a high risk of developing infection endocarditis (see Box 20-5).[63b] Acquired valvular lesions, roughened areas of the heart from septal defects, and prosthetic heart valves also increase the risk of endocarditis. Patients with prosthetic heart valves develop endocarditis with an incidence rate of 4% per year. Patients with a history of infectious endocarditis have an increased chance of recurrence. The risk of a second infection of any patient with one previous episode of endocarditis is 10% per year. After the second infection, the risk of recurrence increases to 25%.

The risk of bacterial endocarditis increases with the amount of intraoral soft tissue trauma. There is a correlation between the incidence of endocarditis and the number of teeth extracted or the degree of a pre-existing inflammatory disease of the mouth.[64] A incidence of bacteremia is six times higher in patients with severe periodontal disease.[65] However, if scaling and root planing are performed before subsequent soft tissue surgery, the risk of endocarditis is greatly reduced. Bacteremia after traumatic tooth brushing, endodontic treatment, and paraffin chewing has also been reported.[66] Endocarditis may even occur in an edentulous patient with denture sores.[67] Chlorhexidine painted on isolated gingiva or irrigation of the sulcus 3 to 5 minutes before tooth extraction reduces postextraction bacteremia.

Box 20-5 American Heart Association Recommended Antibiotic Prophylaxis Guideline

Prophylactic Antibiotics No Longer Recommended for Patients with These Conditions

- Mitral valve prolapse
- Rheumatic heart disease
- Bicuspid valve disease
- Calcified aortic stenosis
- Congential heart conditions such as ventricular septal defect, atrial septal defect, and hypertrophic cardiomyopathy

Phrophylactic Antibiotics Indicated for Patients with These Conditions (High Risk)

- Artificial (prosthetic) heart valves
- History of infectious endocarditis
- Unrepaired or incompletely repaired cyanotic congential heart disease including shunts and conduits
- Congenital heart defects repaired with prosthetic material or device
- Repaired congential heart defects with residual defect at the site or adjacent to a prosthetic device
- Cardiac transplantation recipients who develop cardiac valvulopathy

Data from Wilson W, Taubert KA, Gewitz M et al: Prevention of infective endocarditis. guidelines from the American Heart Association: a guideline from the American Heart Association rheumatic fever, endocarditis, and Kawasaki disease committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group, *Circulation* April 19, 2007. Available at http://circ.ahajournals.org/cgi/content/abstract/CIRCULATIONAHA.106.183095v1. Accessed September 20, 2007.

Dental Implant Management

The implant dentist must be familiar with the antibiotic regimens for heart conditions requiring prophylaxis. A similar regimen is suggested for any person requiring antibiotic coverage. The standard regimen established by the American Heart Association in 1997 may be administered orally or parenterally (Table 20-12).[68] The oral regimen in adults is 2 g amoxicillin orally, 60 minutes before the procedure. A second dose is not necessary because of the prolonged serum levels above the minimal inhibitory concentration of most oral streptococci[69] and the prolonged serum inhibitory activity induced by amoxicillin against such strains (6 to 14 hours).[70] For patients unable to take oral medications, 2 g ampicillin is administered intramuscularly (IM) or intravenously (IV) 30 minutes before the procedure.

If the patient is allergic to penicillin, clindamycin 600 mg or cephalexin (or cefadroxil) 2 g are administered orally 1 hour before the procedure. For patients allergic to penicillin and not able to take oral medications, clindamycin 600 mg IV within 30 minutes of the procedure or cefazolin 1 g IM or IV 30 minutes before the procedure are the recommended regimens for oral procedures.[68] Erythromycin is no longer included because gastrointestinal upset and complicated pharmacokinetics of various formulations[68] make its use problematic.

In patients who are classified in the high-risk category for development of endocarditis, elective implant therapy may be contraindicated. Edentulous patients restored with implants must contend with transient bacteremia from chewing, brushing, or peri-implant disease. Endosteal implants, with an adequate width of attached gingiva, are the implants of choice for patients

WWW.HIGHDENT.IR

Table 20-12 Antibiotic Regimens for Heart Conditions Requiring Prophylaxis

SITUATION OF PATIENT	AGENT	REGIMEN
Standard general prophylaxis	Amoxicillin	Adults: 2.0 g, 1 hr before procedure
Unable to take oral medications	Ampicillin	Adults: 2.0 g IM or IV
Allergic to penicillin	Clindamycin	Adults: 600 mg, 1 hr before procedure
	Cephalexin* or cefadroxil*	Adults: 2.0 g, 1 hr before procedure
	Azithromycin or clarithromycin	Adults: 500 mg, 1 hr before procedure
Allergic to penicillin and unable to take oral medication	Clindamycin	Adults: 600 mg IV within 30 min before procedure
	Cefazolin*	Adults: 1.0 g IM or IV within 30 min before procedure

Adapted from *Prevention of infective endocarditis: guidelines from the American Heart Association*. A guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Copyright © 2007, American Heart Association, Inc.
IM, Intramuscularly; *IV*, intravenously.
* Cephalosporins should not be used in individuals with immediate-type hypersensitivity reaction (urticaria, angioedema, or anaphylaxis) to penicillins.

in this group who need implant-supported prostheses. Implants may be contraindicated for patients with a limited oral hygiene potential and for those with a history of multiple endocarditis events. In addition, intramucosal inserts may be contraindicated for many of these patients because a slight bleeding can occur on a routine basis for several weeks during the initial healing process.

Implant surgery in patients with aortic stenosis is contraindicated until after aortic valve replacement.[71] It has been recommended that patients with valve replacements postpone any elective implant surgery until 15 to 18 months after surgical completion. This is because these patients are at high risk for bacterial endocarditis and because of the use of high doses of anticoagulants.[72] Special precautions should always be adhered to in valve replacement patients, as their therapeutic bleeding times are usually high (INR 2.5 to 3.5).

Endocrine Disorders

Diabetes Mellitus

Diabetes mellitus is a major endocrine disorder that affects approximately 7% of the population with another 2% to 3% undiagnosed.[73] In patients age 60 years or older, 20.9% of all people in this age group suffer from diabetes (approximately 1 in 5).[74] Approximately 20.8 million children and adults suffer from diabetes and contribute to 225,000 deaths per year. Diabetes ranks as the sixth leading cause of death in the United States.

Over the years, the classification of diabetes has included "juvenile-onset" and "adult-onset" diabetes as well as "insulin-dependent" and "non–insulin-dependent" diabetes. The most current classification includes three general clinical categories; type 1 diabetes, type 2 diabetes, and gestational diabetes (pregnancy).[75,76]

In type 1 diabetes, insulin is not produced from the pancreas. This type of diabetes develops most frequently in children; however, the incidence in an older population is increasing. Type 2 diabetes is much more common and accounts for approximately 95% of the diabetic cases. This type of diabetes almost always occurs in adults and results from the body's inability to respond properly to the action of insulin, which is produced from the pancreas. The incidence of type 2 diabetes is estimated to double by the year 2025 because of aging, unhealthy diets, and obesity.[77]

The major symptoms of diabetes are polyuria, polydipsia, polyphagia, and weight loss. Therefore the patient's medical history should include questions concerning increased thirst, urination, appetite, or recent weight loss. Almost every cell membrane needs insulin to enable glucose penetration to occur, with the exception of those cells in the brain and spinal cord. With insulin deficiency, the glucose remains in the bloodstream and increases the blood glucose level. Diabetic patients are prone to develop infections and vascular complications. The healing process is affected by impaired vascular function, chemotaxis, impaired neutrophil function, and an anaerobic milieu. Protein metabolism is decreased, and healing of soft and hard tissue is delayed. Nerve regeneration is altered, and angiogenesis is impaired.

Many studies both in animals and humans have been conducted with respect to diabetes. Animal studies have shown a significant reduction in bone-implant contact[78] and a reduction of osseointegration in trabecular bone but not cortical bone.[79] Clinical human data include the recommendation that no contraindications exist for diet and orally controlled diabetes; however, for insulin-controlled patients, implants may be contraindicated.[80] Some studies have shown that insulin-controlled diabetic patients are implant candidates if they have antibiotic coverage, and other studies have seen no differences with success rates.[81,82] Additionally, researchers have concluded that

WWW.HIGHDENT.IR

Table 20-13 Dental Implant Management in Patients with Diabetes Mellitus

RISK	MEAN BLOOD GLUCOSE (mg/dL)	HBA1c (%)	TYPE 1	TYPE 2	TYPE 3	TYPE 4
Mild	≤150	≤7	+	+	Sedation; premedication; diet/insulin adjustment	Sedation; premedication; diet/insulin adjustment
Moderate	150-240	7-10	+	+	Sedation; premedication; diet/insulin adjustment; consultation	Diet/insulin adjustment; hospital consultation
Severe	Uncontrolled, >240	>10+	+	Postpone all elective procedures	Postpone all elective procedures	Postpone all elective procedures

+, May be performed with regular protocol after physician consultation.

implants are usually successful in patients with diabetes, provided the diabetes is controlled (patients should monitor HbA_{1C}). Uncontrolled diabetes should be treated and controlled during the implant surgery healing period.[83-86]

Dental Implant Management

The most serious complication for diabetic patients during dental procedures is hypoglycemia, which usually occurs as a result of excessive insulin level, hypoglycemic drugs, or inadequate food intake. Weakness, nervousness, tremor, palpitations, or sweating are all signs of hypoglycemia. Mild symptoms can be treated with sugar in the form of orange juice or candy. If the symptoms are not addressed, they may evolve from confusion and agitation to seizure, coma, and death.

The stress of surgery may provoke the release of counterregulatory hormones that will impair insulin regulation and may result in hyperglycemia and a catabolic state.

A stress reduction protocol is recommended on all patients including early morning appointments, adequate breakfast, pain and anxiety reduction, treatment breaks, and possible sedation.

Patients at low risk of complications related to diabetes are those who are asymptomatic and have good metabolic control. Their blood glucose levels are less than 150 mg/dL (average 100 mg/dL) with HbA1C 7.0 test scores. These patients may be treated with a normal protocol for all nonsurgical appointments (type 1). For surgical procedures, these patients need a little more care and attention. Need for a stress reduction protocol, diet evaluation before and after surgery, and control of the risk of infection are all addressed. Sedative procedures and antibiotics are often used for implant or advanced surgical procedures (types 3 or 4). Intravenous conscious sedation and infusion of glucose and saline solution (D_5W) can be used for lengthy procedures (Table 20-13).

Patients at moderate risk show periodic manifestations of the disease but are in metabolic balance because few complications of diabetes are present. Their blood glucose levels are around 200 mg/dL (HbA1C test scores at 8.0 to 9.0). Diet control, stress reduction protocol, aseptic technique, and antibiotics are more important for these individuals than for those in the low-risk group. Most nonsurgical procedures can follow a normal protocol (type 1). Oral or IV sedation should be considered for many surgical or restorative type 2 procedures. For all patients who have a moderate or high risk to uncontrolled diabetes, corticosteroids should not be prescribed as they may adversely affect blood sugar levels. Patients after surgery or prosthetic appointment who require long-lasting anesthetics may not eat until sensation returns to their oral structures, especially in the mandible. This may require a modification of their insulin doses, because normal food intake is not present. However, the dentist usually should not alter the patient's insulin without physician consultation and recommendation. Medical consultation should precede moderate or advanced surgical procedures (types 3 and 4). Sedative techniques and hospitalization should be considered for advanced surgical procedures (type 4).

Patients at high risk with uncontrolled diabetes report a history of frequent hypoglycemia and show multiple complications of diabetes. Their fasting blood sugar fluctuates widely, often exceeding 250 mg/dL (HbA1C test scores 10.0 or higher). These patients can follow type 1 procedures when a conscious effort is made to decrease stress. All other procedures, whether nonsurgical or surgical, require medical consultation. If possible, any treatment should be deferred until the medical condition is stabilized. Postoperatively, the patient's diet requires careful attention in relationship to insulin doses.

All diabetic patients are subject to a greater incidence and severity of periodontal disease, dental caries from xerostomia, candidiasis, burning mouth, and lichenoid reactions. Approximately 75% of these patients suffer from periodontal disease and exhibit increased alveolar bone loss and inflammatory gingival changes. Tissue abrasions are more likely in denture wearers because the depletion in oxygen tension decreases the rate of

WWW.HIGHDENT.IR

Table 20-14 Dental Implant Management in Patients with Thyroid Disorders

RISK		TYPE 1	TYPE 2	TYPE 3	TYPE 4
Mild	Medical examination <6 months Normal Fct last 6 months	+	+	+	+
Moderate	No symptoms No medical examination No Fct test	+	Decrease epinephrine, steroids, CNS depressants	Physician consultation	Physician consultation
Severe	Symptoms	+	Postpone all elective procedures	Postpone all elective procedures	Postpone all elective procedures

Fct, Function; +, may be performed with regular protocol after physician consultation; *CNS*, central nervous system.

epithelial growth and decreases tissue thickness. Burning mouth syndrome is associated with neuropathies, microangiopathy, and salivary gland dysfunction.

Thyroid Disorders

Thyroid disorders are the second most common endocrine problem, affecting approximately 1% of the general population, principally women. This is reflected in the rank held by Synthroid, the fourth most common drug prescribed in the United States.[52,87] Because the majority of patients in implant dentistry are women, a slightly higher prevalence of this disorder is seen in the dental implant practice.

The thyroid gland is one of the larger endocrine glands in the body and is situated at the level of C5 and T1 vertebral bodies, just below the laryngeal prominence. The main function of the thyroid gland is to produce hormones, the most common being thyroxine (T4) and triiodothyronine (T3). Thyroxine is responsible for the regulation of carbohydrate, protein, and lipid metabolism. In addition the hormone potentiates the action of other hormones such as catecholamines and growth hormones.

Abnormalities in the anterior pituitary gland or the thyroid can result in disorders of thyroxine production. Excessive production of thyroxine results in hyperthyroidism. Symptoms of this disorder include increased pulse rate, nervousness, intolerance to heat, excessive sweating, weakness of muscles, diarrhea, increased appetite, increased metabolism, and weight loss. Excessive thyroxine may also cause atrial fibrillation, angina, and CHF. Palpation of the patient's neck often reveals an enlarged thyroid gland (goiter) between the cricoid cartilage and the suprasternal notch.

The most common form of hypothyroidism is Hashimoto's thyroiditis, which is an autoimmune disease in which there is insufficient production of thyroxine. The related symptoms are a result of a decrease in metabolic rate. The patient complains of cold intolerance, fatigue, and weight gain. Eventually hoarseness and decreased mental activity occur, which may lead to coma. Thyroid function tests are used to confirm the diagnosis of hypothyroidism.

Dental Implant Management

Patients with hyperthyroidism are especially sensitive to catecholamines such as epinephrine in local anesthetics and gingival retraction cords. When exposure to catecholamines is coupled with stress (often related to dental procedures) and tissue damage (dental implant surgery), an exacerbation of the symptoms of hyperthyroidism may occur. The result is termed *thyrotoxicosis* or *thyroid storm*. This acute, life-threatening hypermetabolic state clinically presents with symptoms of fever, tachycardia, hypertension, and neurologic and gastrointestinal abnormalities. If left untreated, these symptoms may result in CHF and life-threatening arrhythmias.

The hypothyroid patient is particularly sensitive to CNS-depressant drugs, especially narcotics and sedative drugs such as diazepam or barbiturates. The risk of respiratory depression or cardiovascular depression or collapse must be considered.

Undiagnosed thyroid disease is rare, and the thyroid problems usually are related to previously treated thyroid disease. Therefore a patient with a history of thyroid disorders should be asked about temperature sensitivity, recent weight gain or loss, tremors, or changes in appetite. The pulse and respiration are carefully noted. The most common thyroid disorder patient seen by the implant dentist is the one with known and treated thyroid disease. Any patient with a thyroid disorder and a medical examination in the preceding 6 months who reports normal thyroid function and has no symptoms of the disease is at low risk. A euthyroid patient most often is an ASA II. A normal protocol can be followed for all dental implant surgery and restorative appointments (types 1 to 4) (Table 20-14). Studies have shown that medically controlled hypothyroid patients are not at a higher risk of implant failure and are not a contraindication for implant therapy.[88]

The patient with thyroid disorder who has no symptoms related to thyroid disorders, but has not had a physical or thyroid function test recently, is placed in the moderate-risk category. This patient may follow a normal protocol for type 1 procedures. Stress reduction protocol with or without sedation is suggested for

WWW.HIGHDENT.IR

simple to advanced operative appointments and simple surgical procedures (type 2). The use of epinephrine and of CNS depressants such as narcotic analgesics, barbiturates, and diazepam should be limited. For moderate to advanced implant procedures or surgery (types 3 and 4), medical reexaminations are often indicated. After thyroid control is established, these patients are placed in the low-risk category and normal protocol is adopted.

A symptomatic patient is at high risk and is usually classified as an ASA III risk, regardless of when the last medical evaluation was performed.[89] Such patients should have only examination procedures performed (type 1); all other treatment is deferred until a medical and laboratory evaluation confirms control of the disorder.

Adrenal Gland Disorders

The adrenal glands are endocrine organs located just above the kidneys. Epinephrine and norepinephrine are produced by chromaffin cells in the adrenal medulla, which forms the central portion of the gland. These hormones are largely responsible for the control of blood pressure, myocardial contractility and excitability, and general metabolism.[90] The outer portion of the gland or adrenal cortex produces three different types of hormones. Glucocorticoids regulate carbohydrate, fat, and protein metabolism and also help decrease inflammation. Synthetic glucocorticoid medications may be used by the implant dentist to decrease swelling and pain.[91] The mineralocorticoids maintain sodium and potassium balance, and the third category consists of the sex hormones. The hypothalamus, the anterior pituitary gland, and the adrenal glands interact to regulate glucocorticoid production. The mineralocorticoids are regulated by renin angiotensin hormone, the anterior pituitary gland, and serum potassium levels.

Cortisol is one of the most important glucocorticoids secreted by the adrenal cortex. Insufficient production and secretion of cortisol leads to primary adrenocortical insufficiency, also termed *Addison's disease*. A patient with Addison's disease shows symptoms of weakness, weight loss, orthostatic hypotension, nausea, and vomiting. The physical signs of primary insufficiency do not manifest until 90% of the gland is destroyed. Signs and symptoms develop insidiously over months. When these signs are noted, the implant dentist should require a medical consultation. These patients cannot increase their steroid production in response to stress and in the midst of surgery or long restorative procedures may have cardiovascular collapse. During the physical examination, the dentist can notice hyperpigmented areas on the face, lips, and gingiva.[92] An increase in serum potassium level (hyperkalemia) and decrease in serum glucose level are characteristic of Addison's disease.

When adrenal hypersecretion of cortisol is present, patients will show signs of Cushing's syndrome. The characteristic changes associated with this disease are moon facies, truncal obesity or "buffalo hump," muscle wasting, and hirsutism. Patients are hypertensive and long-term excess function of the cortex decreases collagen production. These patients bruise easily, have poor wound healing, experience osteoporosis, and are also at increased risk for infection. These elements are especially noteworthy to the implant dentist. Laboratory studies show an increase in blood glucose related to an interference with carbohydrate metabolism. The CBC often shows a slight decrease in eosinophil and lymphocyte counts.

Corticosteroids are potent anti-inflammatory drugs used to treat a number of systemic diseases and are one of the most prescribed drugs in medicine. Steroids are used for more than 80 conditions such as arthritis, collagen and vascular disorders, kidney diseases, asthma, and dermatologic disorders. However, the continued administration of exogenous steroids suppresses the natural function of the adrenal glands and cause a condition equivalent to Cushing's disease. As a result, patients under long-term steroid therapy are placed on the same protocol as patients with hypofunction of the adrenal glands.

Dental Implant Management

Patients with a history of adrenal gland disease, whether hyperfunctioning or hypofunctioning, face similar problems related to dentistry and stress. The body is unable to produce increased levels of steroids during stressful situations, and cardiovascular collapse may occur. As a result, additional steroids are prescribed for the patient just before the stressful situation. These doses are stopped within 3 days. The healthy patient will accelerate steroid production three to five times higher than regular levels to respond to the stress of surgery or dental procedures. Therefore for patients with known adrenal disorders, the physician should be contacted for consultation. The nature of the disorder and the recommended treatment should be evaluated.

The patient on regular maintenance doses of steroid in excess of prednisone 5 mg/day is at high risk of adrenal suppression. Adrenocortical suppression should be suspected if a patient has received a dose of 20 mg or more of cortisone or equivalent daily via the oral or parenteral route for a continuous period of 2 weeks or longer within 2 years of dental treatment (Table 20-15).[93] Consultation with the patient's physician is indicated. For simple to advanced operative procedures and simple extractions, and for periodontal or implant surgery (types 1 and 2), the steroid dose should be doubled up to 60 mg prednisone or equivalent (10 mg dexamethasone). The day after the procedure, the maintenance dose is returned to normal. Oral or intravenous conscious sedation is used to reduce stress. For moderate to advanced implant surgery or the very anxious patient, general anesthesia may be indicated. The day of the procedure, 60 mg prednisone is administered. This dose is reduced by 50% each day over a 2- to 3-day period to the maintenance dose. Antibiotics are also administered for 3 to 5 days.

WWW.HIGHDENT.IR

Table 20-15 Dental Implant Management in Patients with Adrenal Disorders

RISK		TYPE 1	TYPE 2	TYPES 3 AND 4
Mild	Equivalent of prednisone on alternate days for >1 year	+	Surgery on day of steroids	Sedation and antibiotics Steroids <60 mg prednisone on day 1 Dose ×/2 on day 2 Maintenance dose on day 3
Moderate	Equivalent of prednisone >20 mg or >7 days in past year	+	Sedation and antibiotics: 20-40 mg day 1 Dose × 2 on day 2 Dose × 4 on day 3	Sedation and antiobiotics: 60 mg day 1 Dose × 2 on day 2 Dose × 4 on day 3
Severe	Equivalent of prednisone 5 mg/day	+	Elective procedures contraindicated	Elective procedures contraindicated

+, May be performed with regular protocol.

Patients at significant or moderate risk for adrenal suppression are those formerly on steroid therapy of 20 mg prednisone or more for longer than 7 days within the preceding year. Simple to complex restorative procedures or simple surgery (types 1 and 2) suggest administration of 20 to 40 mg prednisone the day of the procedure. Sedation techniques and antibiotics for 3 to 5 days are suggested. The following day the steroid dose is reduced by 50%, and the third day the dosage is reduced by an additional 50% or returned to normal. For types 3 and 4, moderate to advanced surgical procedures, the protocol is further modified. Prednisone 60 mg or equivalent is administered the day of the surgery. This dose is reduced 50% the following day, and another 50% on the third day. General anesthesia may be used to reduce anxiety in the apprehensive patient.

Patients at low risk for adrenal suppression are those on alternate-day steroid therapy or those whose steroid therapy ended 1 year or more before the implant procedure. For these, dental procedures are scheduled the day steroids are taken or up to 60 mg prednisone is administered. On the second day the dose is reduced 50%; on the third day, the patient resumes the alternate-day schedule. Sedation and antibiotics are also used.

Steroids act in three different ways that affect implant surgery. They decrease inflammation and are useful in decreasing swelling and related pain. However, steroids also decrease protein synthesis and therefore delay healing. In addition, they decrease leukocytes and thereby reduce the patient's ability to fight infection. Therefore whenever steroids are given to patients for surgery, it is reasonable to prescribe antibiotics. After a loading dose, amoxicillin or clindamycin are given three times per day for 3 to 5 days.

Pregnancy

Implant surgery procedures are contraindicated for the pregnant patient. Not only is the mother the responsibility of the dentist, but so is the fetus. The radiographs or medications that may be needed for implant therapy and the increased stress are all reasons the elective implant surgical procedure should be postponed until after childbirth. However, after implant surgery has occurred, the patient may become pregnant while waiting for the restorative procedures, especially as modalities may require 3 months to 1 year of healing. Periodontal disease is often exacerbated during pregnancy. All elective dental care, with the exception of dental prophylaxis, should be deferred until after the birth. The only exceptions to this are caries control or emergency dental procedures. In these instances, medical clearance should be given for all drugs, including anesthetics, analgesics, and antibiotics. Usually lidocaine, penicillin, erythromycin, and acetaminophen (Tylenol) are approved. Aspirin, vasoconstrictors (epinephrine), and drugs that cause respiratory depression (e.g., narcotic analgesics) are usually contraindicated. Diazepam (Valium), nitrous oxide, and tetracycline are almost always contraindicated.

Dental Implant Management

Almost 15% of pregnancies are terminated by spontaneous abortion or miscarriage during the first trimester. Dental prophylactic appointments are suggested in the second or third trimester. The hygienist and dentist should realize that in the middle to late third trimester, hypotension can occur in a supine mother as a result of pressure of the fetus on the inferior vena cava.

Hematologic Diseases

Erythrocytic Disorders

In a healthy patient, 4 to 6 million RBCs per milliliter of blood are in circulation. Red blood cells make up the largest portion of the formed elements in blood. There are two main categories of erythrocyte disorders: polycythemia (increased erythrocyte count) and anemia (decrease in hemoglobin).

Polycythemia

Polycythemia is a rather rare chronic disorder characterized by splenic enlargement, hemorrhages,

WWW.HIGHDENT.IR

and thrombosis of peripheral veins. Death usually occurs within 6 to 10 years and complicated implant or reconstruction procedures are usually contraindicated.

Anemia

Anemia is the most common hematologic disorder. Almost all blood dyscrasias may at one time or another be associated with anemia. Anemia is not a disease entity; rather, it is a symptom complex that results from a decreased production of erythrocytes, an increased rate of their destruction, or a deficiency in iron. It is defined as a reduction in the oxygen-carrying capacity of the blood and results from a decrease in the number of erythrocytes or the abnormality of hemoglobin. There are a number of different types of anemia, the most common being iron deficiency anemia and relative bone marrow failure. Iron deficiency anemia may be caused by a decreased intake of iron, a decreased absorption of iron, or an increase in bleeding. Vitamin C increases the absorption of iron. The female patient may normally be anemic in menses or pregnancy. Mild anemia in a man, however, may indicate a serious underlying medical problem. The most common causes of anemia in men are peptic ulcers or carcinoma of the colon. These serious complications warrant medical evaluation of any male patient found to be anemic.

The general symptoms and signs are all a consequence of either a reduction of oxygen reaching the tissues or alterations of the red blood cell count. The symptoms of mild anemia include fatigue, anxiety, and sleeplessness. Chronic anemia is characterized by shortness of breath, abdominal pain, bone pain, tingling of extremities, muscular weakness, headaches, fainting, change in heart rhythm, and nausea. The general signs of anemia may include jaundice, pallor, spooning or cracking of the nails, hepatomegaly and splenomegaly, and lymphadenopathy. The oral signs of anemia affect the tongue; symptoms include a sore, painful, smooth tongue; loss of papillae; redness; loss of taste sensation; and paresthesia of the oral tissues.

Complications in the implant patient may affect both short-term and long-term prognoses. Bone maturation and development are often impaired in the long-term anemic patient. A faint, large trabecular pattern of bone may even appear radiographically, which indicates a 25% to 40% loss in trabecular pattern. Therefore the initial character of the bone needed to support the implant can be affected significantly. The decreased bone density affects the initial placement and may influence the initial amount of mature lamellar bone forming at the interface of an osteointegrated implant. The time needed for a proper interface formation is longer in poor-density bone.[94] However, after the implant is loaded successfully, the local strain environment will improve the bone density at the interface.

Abnormal bleeding is also a common complication of anemia; during extensive surgery, a decreased field vision from the hemorrhage may occur. Increased edema and subsequent increased discomfort postsurgically are common consequences. In addition, the excess edema increases the risk of postoperative infection and its consequences. Not only are anemic patients prone to more immediate infection from surgery, they are also more sensitive to chronic infection throughout their lives. This may affect the long-term maintenance of the proposed implant or abutment teeth.

Approximately 0.15% of the African American population has sickle cell anemia.[95] Patients with such a disorder usually show marked clinical manifestations and often die before the age of 40. Secondary infections are a common consequence with frequent history of osteomyelitis and bone infection. Because of these complications, implants are contraindicated in patients with sickle cell anemia.

The laboratory tests that diagnose anemia or polycythemia are in the CBC (see Table 20-1). An accurate test for anemia is the hematocrit, followed by the hemoglobin; the least accurate is the RBC count. The hematocrit indicates the percentage of a given volume of whole blood composed of erythrocytes. The normal values for men range from 40% to 50%; those for women from 35% to 45%. Hemoglobin composes almost 95% of the dry weight of red blood cells. Abnormal hemoglobin may result from its combination with substances other than oxygen (e.g., carbon monoxide) or genetic diseases (e.g., sickle cell diseases). Normal values for men are 13.5 to 18 g/dL; those for women are 12 to 16 g/dL. The minimum baseline recommended for surgery is 10 mg/dL, especially for elective implant surgery. For the majority of anemic patients, implant procedures are not contraindicated. However, preoperative and postoperative antibiotics should be administered and the risk of bleeding in anemic patients should not be potentiated by the prescription/use of aspirin. Hygiene appointments should be scheduled more frequently for these patients.

Leukocytic Disorders

Leukocyte disorders are an important consideration in hematologic diseases. The WBC count normally ranges from 4500 to 13,500/mm^3 in the adult. Leukocytosis is defined as an increase in circulating WBCs in excess of 13,500/mm^3. The most common cause of leukocytosis is infection. Leukemia, neoplasms, acute hemorrhage, and diseases associated with acute inflammation or necrosis (e.g., infarction, collagen diseases) are more serious causes of leukocytosis. Physiologic conditions such as exercise, pregnancy, and emotional stress can also lead to leukocytosis.

Leukopenia is a reduction in the number of circulating WBCs to less than 4500/mm^3. A decreased leukocyte count may accompany certain infections (e.g., infectious hepatitis), bone marrow damage (from radiation therapy), nutritional deficiency (e.g., vitamin B_{12}, folic acid), and blood diseases (e.g., anemia).

WWW.HIGHDENT.IR

Table 20-16 Dental Implant Management in Patients with Chronic Obstructive Pulmonary Disease

RISK	TYPE 1	TYPE 2	TYPE 3	TYPE 4
Mild (ASA II)	+	+	+	+
Moderate (ASA III)	+	Physician	Physician/moderate treatment	Physician/moderate treatment
Severe (ASA IV)	+	Postpone (hospitalization)	Elective procedures contraindicated	Elective procedures contraindicated

ASA, American Society of Anesthesiologists; +, may be performed with regular protocol.

In the potential implant candidate with leukocytosis or leukopenia, many complications can compromise the success of the implants and prosthesis. The most common is infection, not only during the initial healing phase but also long term. Delayed healing is also a consequence of WBC disorders. For most implant procedures, the first few months are critical for long-term success. Delayed healing may increase the risk of secondary infection.

Thrombocytopenia is caused by decreased production, increased destruction, or sequestration of platelets in the spleen, which results in potential bleeding complications during surgery. A platelet count should always be obtained, and a value lower than 50,000 U/L contraindicates elective dental surgery because of a significant risk of postoperative bleeding.[96]

Most oral implant procedures are contraindicated for the patient with acute or chronic leukemia. Acute leukemia is an inevitably fatal disease. These patients experience serious oral problems, either secondary to the disease process or as complications after chemotherapy. The patient with chronic leukemia will experience anemia and thrombocytopenia. Although the infection is less severe than in acute leukemia, radiolucent lesions of the jaws, oral ulcerations, hyperplastic gingiva, and bleeding complications develop in these patients.

Treatment planning modifications should shift toward a conservative approach when dealing with leukocyte disorders. Complications are more common than in erythrocyte disorders. If the condition is temporary, such as an acute infection, surgical procedures should be delayed until the infection has been controlled and the patient has returned to a normal condition.

Chronic Obstructive Pulmonary Diseases

Two common forms of chronic obstructive pulmonary disease (COPD) are emphysema and chronic bronchitis. COPD is the fourth most common cause of death after cardiovascular disease and accounts for more than 125,000 deaths per year in the United States.

The death rate of COPD is higher in women than men, and 80% to 90% of deaths occur in smokers. A smoker has a tenfold greater chance of dying from COPD than does a nonsmoker.[97] The usual course of COPD is progressive, resulting in a decline in pulmonary function over many years. Respiratory failure is usually precipitated by pulmonary infection, which eventually leads to death.[98]

Patients with COPD may have a combination of symptoms including chronic cough, sputum production, and shortness of breath. The examination evaluates tachycardia, tachypnea, wheezing, use of accessory respiratory muscles, cyanosis, an increase in the anteroposterior diameter of the chest (barrel chest), prolonged expiration, and failure of the right side of the heart.

Dental Implant Management

Patients with difficulty breathing only on significant exertion and normal laboratory blood gases are at minimal risk and may follow all restorative or surgical procedures with normal protocols (types 1 to 4) (Table 20-16). Dental management of COPD patients may require repositioning the patient from the normal supine position. Depending on the severity of the disease, orthopnea may result. The patient can be placed in the most recumbent position so that breathing is comfortable. Supplemental oxygen (2 to 3 L) should be administered throughout the dental procedures.

Patients with difficulty breathing on exertion in general are at moderate risk, as are patients on chronic bronchodilator therapy or who have recently used corticosteroids. These patients may follow examination procedures with normal protocol (type 1). A recent medical examination is recommended for all other procedures. Type 2 procedures should be performed in a hospital setting. If the patient is on bronchodilators, no epinephrine or vasoconstrictors should be added to the anesthetics or gingival retraction cord. Adrenal suppression should be evaluated for any patient on steroid therapy within the past year.

Patients at high risk are those with previously unrecognized COPD, acute exacerbation (e.g., respiratory infection), dyspnea at rest, or a history of carbon dioxide retention. Dental management of patients with COPD is staged according to the severity of the disease. If a patient has been hospitalized for respiratory difficulties, a medical consultation is warranted. The dentist should inquire regarding the carbon dioxide retention capability of these patients. Patients who retain carbon dioxide have a severe condition and are prone to respiratory

WWW.HIGHDENT.IR

failure when given sedatives, oxygen or nitrous oxide, and oxygen analgesia.

Examination procedures may be performed under normal protocol (type 1). Elective moderate procedures or advanced surgical or prosthetic procedures are usually contraindicated. However, if surgery or prosthetic procedures are needed to repair a previously inserted implant, they should be performed in the hospital. The use of epinephrine should be limited. Drugs that depress respiratory function, such as sedatives (including nitrous oxide), tranquilizers, and narcotics, should be discussed with the physician.

Liver Disease (Cirrhosis)

Cirrhosis is the third leading cause of death in young men between the ages of 35 and 54 years. It occurs as a result of injury to the liver with resultant loss of liver cells and progressive scarring. The major cause of cirrhosis is alcoholic liver disease.[99] In 1992 more than 7% of all adults met the diagnostic criteria for alcohol abuse and alcohol dependence, but as many as 15 to 20 million may be considered alcoholics. More than 25 million Americans have alcohol-related liver or gallbladder disease, and an estimated 900,000 Americans have cirrhosis.[100-102]

Two of the more important functions to the implant dentist are the synthesis of clotting factors and the ability to detoxify drugs. Hemostatic defects of liver disease are not only the reduced synthesis of clotting factors, but also an abnormal synthesis of fibrinogen and clotting proteins, Vitamin K deficiency, enhanced fibrinolytic activity, and quantitative and qualitative platelet defects. Of patients with liver disease, 50% have a prolonged PT and clinical bleeding. The inability to detoxify drugs may result in oversedation or respiratory depression. The laboratory evaluation of the implant candidate gives much insight to hepatic function. Many CMP tests are associated with the liver; however, the tests that relate most specifically to liver disorders are those for bilirubin and albumin. In addition, the CBC, PT, and even PTT tests may be affected. Therefore a history of abnormal bleeding may indicate liver dysfunction.

Dental Implant Management

Patients with no abnormal laboratory values for CMP, CBC, PTT, and PT are at low risk. A normal protocol is indicated for all procedures (types 1 to 4). Patients with an elevated PT of less than 1.5 times the control value, or bilirubin slightly affected, are at moderate risk. These patients should be referred to their physician. The use of sedatives and tranquilizers may need physician clearance. Nonsurgical and simple surgical procedures may follow normal protocols (types 1 and 2). However, strict attention to hemostasis is indicated. Bovine collagen such as CollaTape, topical thrombin, or additional sutures may be indicated. Moderate to advanced surgical procedures may require hospitalization (types 3 and 4). Postsurgical close surveillance is indicated. Elective implant therapy is a relative contraindication in the patient with symptoms of active alcoholism.

Patients with a PT greater than 1.5 times the control value, mild to severe thrombocytopenia (platelets lower than 140,000/mL), or several liver-related enzymes or chemicals affected (bilirubin, albumin, alkaline phosphatase, serum glutamic-oxaloacetic transaminase, and serum glutamic pyruvic transaminase) are at high risk. Elective dental procedures such as implants are usually contraindicated. If surgical procedures must be performed on a preexisting implant, hospitalization is recommended. Platelet transfusion may be required for even scaling procedures and administration of mandibular nerve blocks. Fresh frozen plasma may be used to correct PT to under half the control value.

Bone Diseases

Diseases of the skeletal system and specifically the jaws often influence decisions regarding treatment in the field of oral implants. Bone and calcium metabolism are directly related. Approximately 99% of the calcium in the body is held in the bones and teeth. Calcium equilibrium is decided by the calcium metabolism of the body, and this affects the bones. Alveolar bone responds to systemic bone-active agents.[103]

The parathyroid hormone is the most important regulator of extracellular calcium concentrations. Vitamin D, prostaglandins, lymphocytes (through osteoclast-activating factors), insulin, glucocorticoids, and estrogen also are involved in this complex balance. Prostaglandins act as a local factor and may cause osteolysis and hypercalcemia, leading to bone resorption.

Osteoporosis

The most common disease of bone metabolism the implant dentist will encounter is osteoporosis, an age-related disorder characterized by a decrease in bone mass, increased microarchitectural deterioration, and susceptibility to fractures. The World Health Organization defines *osteoporosis* as a bone mineral density level more than 2.5 standard deviations below the mean of normal young women.[104] Forty percent of postmenopausal women in the United States have bone mineral density levels denoting osteopenia, and 7% have scores correlated with osteoporosis.[105]

After age 60, almost one third of the population has this disorder; it occurs in twice as many women as men.[53] This condition is common in postmenopausal women or those with a history of ovariectomy. The lack of estrogen increases the likelihood of osteoporosis; the addition of estrogen is the single most effective treatment to increase calcium absorption in these women.

Half of all women present bone mineral density below the normal fracture threshold of a 20-year-old woman

WWW.HIGHDENT.IR

by the age of 65 years. It is estimated that 1.3 million fractures and 133,000 hip fractures occur every year as a result of osteoporosis. Most patients fail to recover normal activity; 24% die from complications related to the fracture within the first year.[106]

Risk factors for the development of osteoporosis are classified as nonmodifiable (sex, age, early menopause, small body frame, race, heredity) or modifiable (low bone mass, lack of exercise, smoking, alcohol, hyperparathyroidism).[107]

The osteoporotic changes in the jaws are similar to other bones in the body. The structure of the bone is normal; however, because of the uncoupling of the bone resorption and formation processes with emphasis on resorption, the cortical plates become thinner, the trabecular bone pattern becomes more discrete, and advanced demineralization occurs.[108,109] The bone loss related to osteoporosis may be expressed in both the dentate and edentulous patient. In one study of osteoporotic women who had their teeth at age 50, 44% had a complete denture by the age of 60, whereas only 15% of nonosteoporotic women had dentures.[110] A strong correlation was shown between periodontal disease and skeletal osteoporotic changes. In addition, women represent a greater percentage of patients with residual ridge resorption than men.[111] The loss of trabecular bone is accelerated in the edentulous patient because the factors involved in resorption are already established. Osteoporosis affects the trabecular bone mass loss to a greater extent than it does cortical bone.[112] Implants in the jaws can decrease the resorption process and should be encouraged.

Bone remodeling is a continuous process; however, bone mass increases during youth and diminishes with aging. The peak bone mass is usually reached by the age of 35 to 40 years and is usually 30% higher in men than in women. In the first 3 to 10 years after menopause, bone loss is rapid. Trabecular bone loss in women 80 years old reaches 40%, but is only 27% in men of the same age. Persons most at risk of osteoporosis are thin, postmenopausal, Caucasian women with a history of poor dietary calcium intake, cigarette smoking, and British or Northern European ancestry. Estrogen therapy can halt or retard severe bone demineralization caused by osteoporosis and can reduce fractures by about 50% compared with the fracture rate of untreated women. Premarin (estrogen replacement therapy) is the most common drug prescribed in the United States for the last several years.[13] However, long-term estrogen therapy has been linked to a slight increase in endometrial cancer, and recently drug regimens have been altered. Studies have evaluated the effect of estrogen replacement therapy on dental implant failures. Osteoporotic patients not taking estrogen have nearly twice the failure rate of maxillary implants in comparison to patients who were receiving estrogen therapy.[113]

Recent advances in radiology, such as dual-energy x-ray absorptiometry, can measure as little as 1 mg of bone mass change at such sites as the hip, spine, and wrist. Such measurements may accurately predict future fracture risk and identify the patients at risk.[114,115] The actual diagnosis and treatment of osteoporosis should be accomplished by the physician. The implant dentist can benefit the patient by noting the loss of trabecular bone and by early referral. Treatment of osteoporosis remains controversial. Its management concentrates on prevention. Regular exercise has shown to help maintain bone mass and increase bone strength. Adequate dietary calcium intake is essential. The advanced demineralization and consequent increase in bone loss of the completely edentulous may become a vicious circle. The denture is less secure, and the patient may not be able to follow the diet needed to maintain proper calcium absorption levels.

The recommended calcium intake is 800 mg/day. The average person in the United States ingests 450 to 550 mg. In postmenopausal women, 1500 mg may be required to maintain a positive calcium balance.[116] Calcium supplements of 1 to 2 g of elemental calcium per day have been shown in several studies to reduce the rate of bone loss. However, there is no evidence that these supplements lead to recovery of bone mass. Plain calcium carbonate tablets contain the greatest fraction of elemental calcium and are relatively inexpensive. It is insoluble and is absorbed after conversion into calcium chloride by gastric hydrochloric acid. Patients with achlorhydria (lack of hydrochloric acid) should be given salts other than calcium carbonate. If the patient has a lactate deficiency, lactate salts are contraindicated. Several food-drug interactions have been reported. Tetracycline and iron do not work effectively with calcium doses. Patients should also avoid phosphate (found in some dairy products or diet soda) or oxalic acid (in spinach and rhubarb) and the phytic acid in bran and whole grains, because these decrease calcium absorption. Patients with a history of renal calculi should avoid calcium supplements. Patients with renal dysfunction need periodic serum and urine calcium level checks, and their serum pH should be monitored to avoid hypercalcemia and metabolic alkalosis.

For patients with established osteoporosis, treatment options include bisphosphonates and calcitonin. Bisphosphonates are inhibitors of bone resorption. Calcitonin, which is normally secreted by the thyroid gland, inhibits bone resorption and alters calcium metabolism.

Dental Implant Implications

Patients with osteoporosis present the implant dentist with many challenges. Although not contraindicated, immediate stabilization of dental implants is a common concern because of decreased trabecular bone mass. Healing periods and implant surface characteristics

WWW.HIGHDENT.IR

should be selected for poorer-quality bone. Additionally, a large percentage of these patients are being treated with bisphosphonates, which have been attributed to various surgical complications.

Although osteoporosis is a significant factor for bone volume and density, it is not a contraindication for dental implants. Clinical reports in postmenopausal women older than 50 years had failure rates similar to other patients, and hormone replacement therapy did not influence the failure rate.[113,117,118] The bone density does affect the treatment plan, surgical approach, length of healing, and need for progressive loading. Implant designs should be greater in width, and surface conditions of implant bodies should be designed to increase bone contact and density. Bone stimulation to the healed interface will increase bone density, even in advanced osteoporotic changes.

Vitamin D Disorders (Osteomalacia)

Vitamin D is a hormone synthesized by the body in several steps involving the skin, liver, kidney, and intestine. It is activated by the kidney in conjunction with parathyroid hormone. The deficiency of vitamin D in the adult leads to osteomalacia. With this deficiency, the intestinal uptake and mobilization of calcium from the bone is altered, thus resulting in hypocalcemia. This will lead to an increased parathyroid hormone secretion, which increases the clearance of phosphorus from the kidneys. This resultant decrease in the concentration of phosphorus prevents a normal mineralization process. Anticonvulsant drugs, especially diphenylhydantoin and phenobarbital, may cause drug-induced osteomalacia. Many gastrointestinal disorders also may result in osteomalacia.

The oral findings of osteomalacia are usually not dramatic. A decrease in trabecular bone, indistinct lamina dura, and an increase in chronic periodontal disease have been reported. The treatment for osteomalacia is supplemental oral vitamin D (50,000 IU).

Dental Implant Implications

Treatment for osteomalacia is usually successful with radiographic changes seen months after treatment. There are no known reports of implant complications in osteomalacia patients; however, there is no contraindication as long as the disease is not active and well controlled.

Hyperparathyroidism

The clinical manifestations of this disease vary widely, depending on the severity. Mild forms may be asymptomatic. Renal colic disorders often occur with moderate disease. Severe hyperparathyroidism can cause bone, renal, and gastric disturbance. It has been noted that when skeletal depletion occurs as a result of stimulation by the parathyroid gland, alveolar bone may be affected before that of the rib, vertebrae, or long bones.

Oral changes related to this disorder occur only with advanced disease. The loss of lamina dura is the most significant finding. Clinically, patients with this disorder develop loose teeth. Altered trabecular bone pattern with the appearance of ground glass may also occur. In animals, secondary hyperparathyroidism affects alveolar bone loss greater than any other bone of the skeleton.[119] Central or peripheral giant cell tumors may also develop.

Dental Implant Implications

Dental implants are contraindicated in areas of active bony lesions. However, implant placement may be initiated after treatment and healing of the affected areas.

Fibrous Dysplasia

Fibrous dysplasia is a disorder in which fibrous connective tissue replaces areas of normal bone in an unorganized arrangement. It is found twice as often in women than it is in men and may affect a single bone or multiple bones. The single-bone disorder is more common in adults and is present in the jaws about 20% of the time. The condition is twice as common in the maxilla as in the mandible. The monostotic (single-bone) fibrous dysplasia begins as a painless, progressive lesion. The facial plate usually expands. The teeth may move as a consequence of the progression. Roots of the teeth may be displaced, but external resorption is rare. Radiographically the appearance of fibrous dysplasia is a noted increase in trabeculation, which presents a "mottled appearance." A ground-glass appearance may also be noted.[120] The polyostotic (multiple-lesion) fibrous dysplasia may affect one or virtually all bones. A predisposition to fracture may occur with this entity. There exists an increased incidence of malignancy (osteosarcoma, chondrosarcoma) in bones affected with fibrous dysplasia. Healing after trauma in patients with fibrous dysplasia is much different than normal bone. The tissue is hypocellular, leading to slow healing and an increased incidence of infection.

Dental Implant Implications

Implant dentistry is contraindicated in the regions of this disorder. The lack of bone and increased fibrous tissue decrease rigid fixation of the implant and are more susceptible to local infection processes. These local infections may spread through the bone and result in more advanced complications. Excision of fibrous dysplasia areas is usually the treatment choice. After the condition is corrected long-term, the area may receive an implant. Radiation therapy has been used, but may be responsible for the transformation of fibrous dysplasia into osteosarcoma or chondrosarcoma.

WWW.HIGHDENT.IR

Osteitis Deformans (Paget's Disease)

Osteitis deformans, or Paget's disease, is a common metabolic disease characterized by slow, progressive, uncontrolled resorption and deposition of bone. This disease is usually seen in Caucasian men older than 40 years. The etiology is unknown and usually affects the maxillary alveolar ridge twice as frequently as the mandibular ridge. Because of the enlargement of the middle one third of the face, the appearance of a "lion-like" deformity is often noted. Diastemas, tooth mobility, and bone pain are additional characteristics. Radiographically, a decreased radiodensity, large circumscribed radiolucencies, patchy areas of coalesced sclerotic bone (cotton-wool appearance), and marrow spaces that are replaced by fibrous tissue are observed. During the active phases of this disease, bone is highly vascular with the possibility of arteriovenous shunts, which may cause hemorrhagic complications.

Paget's disease is marked by high elevations of serum alkaline phosphatase, normal or elevated calcium, and normal phosphate levels. Edentulous patients are often unable to wear their prostheses without discomfort.[121] There is no specific treatment for Paget's disease, and these patients are predisposed to develop osteosarcoma and possibly osteomyelitis.

Dental Implant Implications

Oral implants are contraindicated in the regions affected by this disorder.

Multiple Myeloma

Multiple myeloma is a plasma cell neoplasm that originates in the bone marrow and is characterized by the abnormal proliferation of B cells. It causes severe hypercalcemia, immune suppression, anemia, and thrombocytopenia because it causes widespread bone destruction. The disease is usually found in patients between 40 and 70 years of age. Usually it affects several bones in the body with symptoms of skeletal pain. Pathologic fractures may occur. Punched-out lesions appear radiographically. Oral manifestations of the disease are common (80%) and may affect both the maxilla and the mandible. Paresthesia, swelling, tooth mobility, and tooth movement may occur. Gingival enlargements are also possible. Plasma cell malignancy, Bence Jones proteins in the urine, and anemia are possible laboratory findings associated with this disorder. There is no treatment for multiple myeloma, and the condition is usually fatal 2 to 3 years after onset.

Dental Implant Implications

Dental implants are a relative contraindication in patients with multiple myeloma because of the severity of this disease; however, a case report has described successful implant placement in a patient with this disease.[122]

Osteomyelitis

Osteomyelitis is an acute or chronic inflammatory bone disease that is bacterial in nature. The radiographic appearance is a poorly defined, radiolucent area with isolated fragments of bone (sequestra) that can exfoliate or become surrounded by bone (involucrum). This disorder can be caused by odontogenic and periodontal infections, trauma, dental implants, immunocompromised states, and hypovascularized bone. The treatment includes aggressive surgical drainage, with possible intravenous antibiotic intervention. Osteomyelitis usually occurs in the mandible and is rarely seen in the maxilla, possibly due to the increased vascularization.

Dental Implant Intervention

Osteomyelitis is usually a contraindication unless the etiologic factors are removed and adequate blood supply to the affected area is restored.

Osteogenesis Imperfecta

Osteogenesis imperfecta is the most common inherited bone disease and is characterized by poor bone quality and fragility. Bone fractures along with skeletal deformities are common with very poor healing. Histologically, defective osteoblasts lead to a reduction in bone matrix and abnormal collagen. The quality of the bone is poor with very thin cortical bone and thin, fine trabeculae.

Dental Implant Implications

Dental implants are not contraindicated; however, caution should be given to the very poor bone quality and questionable osseous healing.

Cement-Osseous Dysplasia

This disease is the most common fibro-osseous lesion usually occurring in the mandibular anterior region. There are three types of this disease (i.e., focal, periapical cementoma, and florid) that can vary radiographically as radiolucent, radiopaque, or a combination. The lesions are usually associated with the mandibular anterior teeth. Cement-osseous dysplasia usually occurs in middle-aged women and has a high incidence in African Americans.

Dental Implant Implications

Dental implants are not contraindicated unless in the sclerotic phase of the disease in which the bone is hypovascular. This bone has the ability to become infected easily with questionable healing. Special attention must be given so that the disease does not progress to osteomyelitis.

WWW.HIGHDENT.IR

Table 20-17 American Dental Association/American Academy of Orthopaedic Surgeons Suggested Antibiotic Prophylaxis Regimens

PATIENT TYPE	RECOMMENDED DRUG	SINGLE-DOSE REGIMEN
Patients not penicillin allergic	Amoxicillin, cephalexin, or cephradine	2 g PO 1 hr before dental procedure
Patients not penicillin allergic, unable to take oral medications	Ampicillin or cefazolin	Ampicillin 2 g IM or IV or cefazolin 1 g, 1 hr before dental procedure
Penicillin-allergic patients	Clindamycin	600 mg PO 1 hr before dental procedure
Penicillin-allergic patients, unable to take oral medications	Clindamycin	600 mg IV 1 hr before dental procedure

Data from American Dental Association, American Academy of Orthopedic Surgeons: Antibiotic prophylaxis for dental patients with total joint replacements, *J Am Dent Assoc* 134:895-899, 2003.
PO, By mouth; *IM*, intramuscularly; *IV*, intravenously.

Prosthetic Joints

Approximately 450,000 total joint arthroplasties are performed every year in the United States, and more than 60% are performed on patients older than age 65.[123,124] Dental implants may be used on patients with other prosthetic implants. Bacteremias can cause hematogenous seeding of total joint implants in the early and long term after implantation. The most critical period is up to 2 years after placement.[125,126] There appears to be no scientific evidence to support antibiotic prophylaxis in patients with total joint prosthesis to prevent hematogenous infections.[127] However, approximately 1% to 2% of these prosthetic joint prostheses will become infected each year. The literature reports an association between prosthetic joint infection and dental treatment. It is hypothesized that bacteria from the dental treatment may seed the prosthesis and produce infection. The evidence for this often seems circumstantial or coincidental because the anatomy, blood supply, microorganisms, and mechanisms of infection are all different.[128] Reported cases related to the mouth have involved regions of established suppuration such as a dental abscess or tonsillitis, rather than procedure-induced transient bacteremia.

The joint ADA/American Academy of Orthopaedic Surgeons advisory statement[123,124] recommends the aggressive treatment of acute orofacial infections in patients with total joint prosthesis because those bacteremias associated with acute infections can and do cause late implant infections. Antibiotic prophylaxis is not indicated for dental patients with pins, plates, and screws and is not routinely indicated for most dental patients with total joint replacement. The antibiotic prophylaxis regimen recommended is, however, indicated for patients with higher risk for hematogenous infections undergoing dental procedures with a higher bacteremic incidence (Table 20-17). In addition, it is recommended for dental procedures with higher bacteremic incidence performed on patients within 2 years of post-total joint implant surgery,[126] or those with a history of prosthetic joint infection or other conditions such as inflammatory arthropathies, immune suppression, type I diabetes, or hemophilia. Dental extractions, surgical placement of implants, periodontal surgery, and prophylactic cleaning of teeth and implants are examples of dental procedures with a higher risk of bacteremia.

Ectodermal Dysplasia

Ectodermal dysplasia (ED) is a genetically inherited disorder that occurs in 1 per 100,000 live births.[129] Clinically, ectodermal dysplasia has been divided into two broad categories: an X-linked hypohidrotic form (Christ-Siemens-Touraine syndrome) characterized by the classical triad of hypodontia, hypohidrosis, and hypotrichosis and by characteristic facial features such as prominent supraorbital ridges and a depressed nasal bridge,[130] and an autosomal inherited hydrotic form (Clouston's syndrome) that usually spares the sweat glands but affects teeth, hair, and nails.[131]

The most common intraoral feature off ED is hypodontia or anodontia. In these patients, conventional prosthodontic procedures often are not successful because of anatomical abnormalities that result in poor retention and stability. Because of this, dental implant therapy aimed at restoring function, esthetics, and psychological rehabilitation is an integral part in the management of adolescent patients with ED.[131] Numerous studies have been completed on dental implants in patients with ED. A 3-year study showed impressive success rates in preadolescents (ages 7 to 11, 87%), adolescents (ages 12 to 17, 90%), and in adults (older than 17, 97%).[132] Other positive case reports have shown dental implants as a successful adjunct to oral rehabilitation.[133,134]

Dental Implant Implications

Dental implants are not contraindicated in patients with ED. Although not ideal, implants may be placed in preadolescents with the advantage of function, esthetics, and psychological advantages. Alveolar bone has been shown to continue to grow after implants

WWW.HIGHDENT.IR

have been placed in edentulous ridges of children with ED. Transverse and sagittal growth is not restricted; however, vertical growth may result in submersion of the implants, necessitating prosthetic revision or possible use of longer abutments.[130]

SYSTEMIC AUTOIMMUNE DISEASES

Autoimmune diseases refers to a group of more than 80 serious, chronic illnesses that can affect almost any organ in the body. Approximately 75% of autoimmune diseases occur in women; these diseases are thought to have a genetic predisposition. However, autoimmune diseases are among the most poorly understood diseases, with symptoms extremely variable among individuals.

Sjögren's Syndrome

Sjögren's syndrome is an autoimmune disease in which immune cells attack and destroy exocrine glands that produce saliva and tears. This disorder affects an estimated 4 million people in the United States and 90% of females with an average age of onset in the late 40s. The classic symptoms of Sjögren's syndrome are xerostomia and xerophthalmia (dry eyes). Because of the xerostomia, patients are more susceptible to decay and the mucous membranes become atrophic and friable. Because of the lack of salivary secretions, complications may arise with the use of a tissue-borne prosthesis.

The healing response and integration of implants has been shown to be successful in patients with Sjögren's syndrome.[135] These implant-supported prostheses decrease soft tissue–borne prosthetic sore spots and discomfort.

Dental Implant Implications

There are no contraindications for dental implants in patients with a history of Sjögren's syndrome. However, it is advantageous for the prosthesis to be non–tissue-borne (FP-1, FP-2, FP-3) to minimize complications from xerostomia.

Systemic Lupus Erythematosus

Systemic lupus erythematosus is a chronic, potentially fatal autoimmune disease in which the immune system attacks cells and tissue in almost any part of the body. This disease usually affects women, and approximately 5 million people are afflicted with this disorder. The majority of lupus patients suffer from dermatologic manifestations such as malar rash (butterfly rash) and skin, mouth, and nasal lesions. Additional effects are hematologic, cardiac, pulmonary, and renal complications. There is no cure for lupus, and most patients are treated with corticosteroids and immunosuppressive drugs.

Dental Implant Implications

There is no direct contraindication to dental implant treatment in systemic lupus erythematosus patients. However, caution should be taken for possible associated organ damage and the use of high doses of corticosteroids and immunosuppressive drugs, which may contraindicate dental implants in those individuals.

Scleroderma

Scleroderma is a rare, chronic disease characterized by excessive deposits of collagen that causes musculoskeletal, pulmonary, and gastrointestinal involvement. The most common symptom is the hardening of the skin in which scarring can take place. Most patients have Raynaud's phenomenon, which affects the hands and feet. There is no cure for scleroderma, and various stages of the disease are treated with NSAIDs and immunosuppressant drugs.[136]

Dental Implant Implications

Numerous reports have discussed the successful treatment of scleroderma patients with dental implants. A fixed prosthesis is recommended because of the inability to retrieve a removable prosthesis because of possible dexterity problems. However, caution must given to the fact that a high percentage of these patients are being treated with immunosuppressive drugs, which may contraindicate the implant therapy.

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a chronic, inflammatory autoimmune disease that causes the patient's immune system to attack the muscles and joints of the body. This disorder is known for its painful and disabling inflammation that leads to substantial loss of mobility and dexterity. Rheumatoid arthritis is treated with a wide range of medications including disease-modifying antirheumatic drugs, antiinflammatory drugs, and analgesic medications.[137] Life expectancy of patients with RA is shortened by approximately 5 to 10 years.[138]

Dental Implant Implications

There is no direct contraindication for dental implants in patients who have RA. Because of the lack of mobility and dexterity, a fixed-implant restoration is indicated. Special attention should be given to the treatment medications, as immunosuppressive and glucocorticoid therapy may contraindicate implant treatment.

Human Immunodeficiency Virus

Human immunodeficiency virus (HIV) is a retrovirus that is responsible for acquired immunodeficiency syndrome (AIDS), which causes the immune system to be depressed, leading to life-threatening opportunistic

WWW.HIGHDENT.IR

infections. The World Health Organization recently estimated that 25 million have died since HIV was discovered on December 1, 1981.[139] There is no cure for either HIV or AIDS. A postexposure prophylaxis, an antiretroviral, has been shown to reduce the risk of infection after exposure. Highly active antiretroviral therapy, which is a protease inhibitor, has been highly beneficial since its introduction in 1996.

Dental Implant Implications

Numerous reports have shown successful dental implant therapy in HIV patients.[140,141] However, there are insufficient data to determine the association between HIV infection and the success of dental implants. Special care must be taken to evaluate the current status of the patient's immune system and the potentially toxic medications the patient is taking.

MEDICATIONS OF INTEREST TO IMPLANT DENTISTRY

Bisphosphonates

Bisphosphonates are a group of drugs that are widely used for several bone disorders and have been approved by the U.S. Food and Drug Administration for treatment of osteoporosis, metastatic bone cancer, and Paget's disease.[142] At this time, there are two main types of bisphosphonates: nitrogen containing and non-nitrogen containing, with subgroups of either oral or intravenous administration.

History

Bisphosphonates were first used for industrial purposes in the nineteenth century to prevent corrosion in the textile, fertilizer, and oil industries. In 1968 the first article describing use of bisphosphonates in medicine was published, discussing the inhibition of bone resorption qualities.[143] However, in 2002 reports of serious side effects from these medications after dental surgical procedures were documented. These complications from bisphosphonates are bisphosphonate osteonecrosis, bisphosphonate avascular necrosis, bisphosphonate osteomyelitis, osteochemonecrosis, and Bis-Phossy jaw.

The complications reported with respect to bisphosphonate use are very similar to conditions that were reported as early as the nineteenth century. In 1845 numerous cases of jaw necrosis were documented from workers in an industrial plant that manufactured matches. Symptoms included pain and tissue inflammation leading to progressive extension with greater areas of bone involvement and sequestration.[144] These lesions resulted in very high rates of morbidity and mortality until changes in environmental hygiene eradicated the problem. In the twentieth century, similar cases of jaw necrosis were seen in workers applying radium to watch instrument dials. These cases also were eradicated by changes in industrial hygiene.[145]

Initially, when the first cases of bisphosphonate necrosis were seen, they were treated and thought to be osteoradionecrosis. However, treatment such as surgical intervention and hyperbaric oxygen failed to produce conclusive resolution to the condition.[146,147] Marx and Ruggiero reported on bisphosphonate-induced osteonecrosis of the jaws, particularly from the more potent nitrogen-containing intravenous bisphosphonates (pamidronate and zoledronic acid). However, a small percentage of the cases involved the oral bisphosphonates: risedronate (Actonel) and alendronate (Fosamax).[148,149]

Chemistry

Bisphosphonates are synthetic compounds that have a chemical structure similar to inorganic pyrophosphate, which is an endogenous regulator of bone metabolism. Because bisphophonates comprise two phosphate groups linked together by phosphoether bonds (P-C-P structure), they are more resistant to breakdown by pyrophosphatases and hydrolysis.[150,151] The general structure of bisphosphonates is rather simple to modify; thus different generations vary dramatically according to their biological, therapeutic, and toxicologic characteristics (Box 20-6).[152]

Mechanisms of Action

Bisphosphonates work by suppressing and reducing bone resorption by osteoclasts. Directly, this is accomplished by preventing the recruitment and function of osteoclasts. Indirectly, they stimulate osteoblasts to produce inhibitors of osteoclast formation.[153] Because of this bone resorption suppression, diseases such as Paget's disease of bone, fibrous dysplasia, and metastatic bone cancer are treated very effectively and relieve pain symptoms.

The blood level half-life of bisphosphonates is very short, ranging from 30 minutes to 2 hours.[154] However, after these medications are absorbed into bone tissue, they can persist for up to 10 years in the skeletal tissues, depending on skeletal turnover time.[130,131] After jaw bone surgery, a radiolucent lesion or bone exposure may develop rather than a typical healing mechanism.

Box 20-6 Types of Bisphosphonates

Nitrogen Containing

Oral: alendronate (Fosamax); risendronate (Actonel)
Intravenous: pamidronate (Aredia); zoledronate (Zometa)

Non–Nitrogen Containing

Etidronate (Didronel)
Clodronate (Bonefas)
Tiludronate (Skelid)

WWW.HIGHDENT.IR

Osteonecrosis of the Jaws

Although there exist extensive data on the beneficial effects of bisphosphonates in the treatment of advanced bone diseases, numerous reports have documented the capability of these medications in causing local lesions of the bone osteonecrosis of the jaws.[148,149] Most cases initially reported were after a jaw bone surgery with the more potent nitrogen-containing bisphosphonates pamidronate and zoledronic acid. However, osteonecrosis lesions have been reported in the oral bisphosphonates risedronate and alendronate.

Osteonecrosis may remain asymptomatic for weeks and possibly months. Lesions usually develop around sharp bony areas and previous surgical sites, including extractions, retrograde apicoectomies, periodontal surgery, and dental implant surgery. Symptoms include pain, soft tissue swelling, infection, loosening of teeth, and drainage. Radiographically, osteolytic changes are seen and tissue biopsy has shown the presence of actinomyces, which is possibly caused by secondary infection.[154]

Management of Osteonecrosis

Initially, the first cases of biphosphonate osteonecrosis were treated with similar techniques as for osteoradionecrosis. These lesions failed to respond to antibiotics, surgical intervention, and hyperbaric oxygen. The most current recommendation of osteonecrosis is a nonsurgical approach to the management of these lesions because of impaired wound healing. Only minimal bony debridement of these lesions should be performed, such as reduction of sharp edges. Chlorhexidine mouthrinses (0.12%) and antibiotics should be used supplementally.[155] If lesions persist, referral to an oral and maxillofacial surgeon or dental specialist with experience in the treatment of this condition is recommended.

Laboratory Tests

It has been proposed that assays to monitor markers of bone turnover may help in the diagnosis and risk of developing bisphosphonate-associated osteonecrosis.[155a] C-telopeptides (CTx) are fragments of collagen that are released during bone remodeling and turnover. Because bisphosphonates reduce CTx levels, it is believed that evaluating serum CTx levels can be a reliable indicator of risk level. The CTx test (also called *C-terminal telopeptide* and *collagen type 1 C telopeptide*) is a serum blood test obtained by laboratories or hospitals (ICD9 diagnostic code 733.40)

Marx has suggested a preoperative protocol for administering bisphosphonates to patients who are undergoing oral surgical procedures.[155b] His protocol considers the type and duration of bisphosphonate use as well as radiographic and clinical risk factors. Depending upon the laboratory values obtained, a "drug holiday" may be indicated, which includes temporary interruption of bisphosphonate treatment. However, improvement of bisphosphonate levels may not be observed, because measurable levels have been to shown to persist in bone for up to 12 years after cessation of therapy[155c] (Box 20-7).

Dental Implant Implications

A comprehensive medical history is essential before any elective treatment is initiated. Potential risk factors should be documented; previous radiotherapy, chemotherapy, female gender, coagulopathies, exostosis, vascular disorders, alcohol abuse, and smoking. The most important history of bisphosphonates is the use of intravenous nitrogen-containing bisphosphonates such as pamidronate (Aredia) and zoledronic acid (Zometa). The American Academy of Periodontology stated invasive dental procedures should be avoided in patients taking IV bisphosphonate therapy unless absolutely necessary.[156] In September 2004, Novartis added bisphosphonate osteonecrosis warning to IV drug

Box 20-7 Protocol, Suggestions, and Assessment for Patients Taking Oral Bisphosphonates

Protocol and Suggestions*

Oral Bisphosophonate Use >3 Years

1. Physician approval to discontinue bisphosphonates 3 months before sugery and 3 months after sugery ("drug holiday").
2. Determine serum CTx levels during initial consultation and immediately before surgery; CTx levels must be >150 pg/mL before proceeding with surgery.
3. Detailed informed consent for bisphosphonate-associated osteonecrosis.

Oral Bisphosophonate Use <3 Years without Clinical or Radiographic Risk Factors†

1. Serum CTx level must be >150 pg/mL.
2. Proceed with surgery with detailed informed consent for bisphosphonate-associated osteonecrosis.
3. If serum CTx level <150 pg/mL, institute a physician-approved "drug holiday"; continue monitoring every 3 months until CTx levels >150 pg/mL.

Oral Bisphosophonate Use <3 Years with Clinical or Radiographic Risk Factors†

1. Physician-approved "drug holiday" for 3 months.
2. Serum CTx level must be >150 pg/mL to proceed with detailed informed consent for bisphosphonate-associated osteonecrosis.
3. If serum CTx level <150 pg/mL, continue monitoring every 3 months until CTx level >150 pg/mL.

Laboratory Risk Assessment

CTx Value (pg/mL)	Risk for Osteonecrosis
• 300-600 (normal)	None
• 150-299	None to minimal
• 101-149	Moderate
• < 100	High

*Marx RE: *Oral and intravenous bisphosphonate-induced osteonecrosis of the jaws: history, etiology, prevention, and treatment,* Chicago, 2007, Quintessence.
†Glucocorticoid use, widened lamina, or sclerotic bone.

WWW.HIGHDENT.IR

guidelines stating to obtain dental exam of the patient prior to drug administration and to "avoid" invasive dental procedures during dry use.

In the dental setting, the most common bisphosphonates that implant dentists are exposed to are oral nitrogen-containing bisphosphonates such as risedronate, ibandronate, and alendronate. The latest studies show that oral bisphosphonate has a very low probability of causing osteonecrosis.[157] For example, the risk of ulcerative colitis is possible with antibiotics, but the dentist informs the patient of the risk and prescribes the antibiotic when required. Likewise, the risk of oral bisphosphonates and bisphosphonate osteonecrosis is about the same (or less and therefore should be considered a low risk and yet prudent to inform the patient). However, because of the long half-life and the studies only being conducted for 3 years, future long-term complications may be less evident. With this in mind, the implant dentist should be cautioned on the possibility of developing osteonecrosis side effects. The risks versus benefits of dental treatment must be discussed with the patient in detail. A well-documented consent form is recommended with possible medical consultation if the patient has been on this medication for more than 3 years.

The use of glucocorticosteroids may be contraindicated in patients taking bisphosphonates, because these drugs have been associated with an increased occurrence of osteonecrosis.

Immunosuppressive Medications

Immunosuppressive drugs are medications that are used in immunosuppressive therapy to inhibit or prevent activity of the immune system. They are usually used to prevent rejection of transplanted organs and tissues and for the treatment of autoimmune diseases. These drugs have many side effects with the majority of them acting nonselectively; thus the body is unable to resist infections. There are four classes of immunosuppressive drugs: glucocorticoids (prednisone), cytostatics (chemotherapeutic agents), antibodies (polyclonal antibodies), and drugs acting on immunophilins (cyclosporine).

Cytostatics are common medications in the treatment of malignant disease. These drugs cannot discriminate between malignant and normal tissues and become cytoxic to normal tissue. Most chemotherapeutic agents are known to have cytoxic effects on bone, especially on grafted bone where the blood supply is compromised.[158] Because chemotherapeutic agents have a high toxicity for cells that have a high turnover rate, the oral mucosa is often affected. These mucosal ulcerations have been known to become secondarily infected. Very few studies have been completed on the relationship of chemotherapy and dental implants; however, the data show that there exist minimal deleterious effects on implant integration.[159,160]

Glucocorticoids have potent antiinflammatory and immunosuppressive properties. Because these drugs are widely used in the treatment of inflammatory and autoimmune diseases, special attention must be given to patients who are on long-term, high doses of glucocorticoids. These drugs impair many healthy anabolic processes in the body and suppress the immune system, which can lead to severe complications in dental implant patients.

Dental Implant Implications

In the past, chemotherapeutic agents were thought to be absolute contraindications for implant candidates with malignant disease.[161] However, with increased success of this therapy, patients now can be treated successfully, thus increasing their quality of life. Care must be taken to evaluate the timing of the chemotherapy.

Studies have shown high failure rates with dental implants and concurrent cytotoxic chemotherapy.[162] Therefore elective implant treatment is contraindicated in any patients who are actively receiving any type of chemotherapeutic medication. However, after chemotherapy treatment has been completed, implant treatment may be completed after careful consideration of the patient's immune system status and physician approval.

For patients receiving long-term, high-dose glucocorticoid therapy, medical consultation is necessary to ascertain the current status of the immune system. This medication can be a contraindication to dental implant therapy.

Anticoagulant Medications

Warfarin Sodium

Warfarin sodium (Coumadin) is used as an anticoagulant in a wide range of conditions such as ischemic heart disease, deep venous thrombosis, pulmonary emboli, and artificial heart valves. (Table 20-2 targets INR values for specific diseases.)

Warfarin sodium has a half-life of 40 hours, which has been known to vary among individuals from 20 to 60 hours. The mode of action of warfarin sodium is the interference of the synthesis of vitamin K, which is a cofactor in many reactions with the coagulation cascade.

Dental Implant Implications

Until recently, most medical practitioners have believed that the medication should be discontinued before dental surgery to prevent possible bleeding problems. However, there exist many documented cases of embolic complications in patients who discontinue the use of warfarin sodium[163,164] and thrombosis from rebound hypercaogulability.[165] In addition, studies have shown that dental surgery may be performed safely on patients receiving anticoagulant therapy as long as their INR values are within the therapeutic range.[166]

WWW.HIGHDENT.IR

Practitioners should consult with the patient's physician to determine the most recent INR before the surgery. If the INR values are within the therapeutic range (2 to 3.5), there is no need to discontinue use of the anticoagulant. If the INR value is above the therapeutic range (especially higher than 4.0), the physician should take appropriate steps to lower the INR to a safer level or possible discontinue the warfarin and supplement with heparin therapy or vitamin D. However, in all anticoagulant patients, special attention should be given to good surgical technique and use of appropriate local measures to control bleeding.

Aspirin

Aspirin or salicylic acid has been used as an antiinflammatory, analgesic, and antipyretic medication. However, in the 1980s, it was discovered that aspirin also had an antiplatelet effect at very low doses (0.5 to 1 mg/kg) versus higher doses needed for an antipyretic effect (5 to 10 mg/kg) and anti-inflammatory response (30 mg/kg).[167] Because of this research, low-dose aspirin has become a secondary preventive drug for patients who have cardiovascular and peripheral vascular disease.[168]

Aspirin works by inhibiting the formation of prostaglandin thromboxane A2 within the platelet, thus affecting thrombus formation by irreversibly decreasing platelet aggregation. There are numerous studies in the literature that advocate the discontinuation of aspirin therapy for 7 to 10 days before elective surgery,[169,170] whereas others have suggested that aspirin therapy should be continued regardless of the surgical procedure.[171,172] In a prospective dental extraction study with low-dose aspirin, it was shown that low-dose aspirin should not be discontinued before oral surgery and local hemostasis measures are sufficient.[173]

Dental Implant Implications

Because there exists no double-blind study supporting the recommendation of low-dose (<100 mg) aspirin discontinuation, especially with oral surgical procedures, there exists no reason to discontinue use for routine dental implant procedures. Interruption of aspirin therapy may expose the patient to the risk of developing thromboembolism, myocardial infarction, or cerebrovascular accident.[167]

However, for patients taking higher doses of aspirin (>100 mg), or in combination with other anticoagulants (clopidogrel, dipyridamole/aspirin [Aggrenox]), bleeding times should be obtained with possible physician consultation.

The platelet inhibitor clopidogrel (Plavix) is now number 36 in the top 50 drug list.[13] It is approved for the reduction of atherosclerotic events in patients of recent stroke, recent MI, or an established peripheral arterial disease. A recent study supports the use of clopidogrel and aspirin for at least 9 months in all cases involving coronary syndromes.[174] (The American Dental Association Council of Scientific Affairs does not support routine discontinuation of oral anticoagulant therapy and antiplatelet therapy for patients before dental treatment.[175]) However, if type 3 or 4 category treatment is scheduled, a consult with the physician before surgery is warranted to review the INR values.

Herbal Supplements

Over-the-counter herbal and dietary supplements are being consumed at a record pace for general health improvement and treatment of chronic conditions. Herbs have been known to have unwanted side effects and cause drug interactions, and they have been associated with surgical complications (Boxes 20-8 and 20-9). Many of these supplements contain active ingredients that have strong biological effects. The doses may be unregulated and variable. The *Journal of the American Medical Association* estimates that 15 million adults are at risk for adverse interactions between herbs and prescription medications.[175a] More than 2900 adverse events related to supplements have been reported to the Food and Drug Administration, including 104 deaths.[175b]

LIFESTYLE-RELATED FACTORS

Tobacco

In the United States, approximately 21% of the population smokes cigarettes. Although this has declined by 50% (42% of the population in 1965), 45 million

Box 20-8 Supplements Associated with Increased Bleeding

- Chamomile
- Dong quai
- Feverfew
- Fish oil
- Garlic
- Ginger
- Gingko
- Ginseng
- St. John's wort
- Vitamin E

Box 20-9 Supplements Associated with Drug Interactions with Nonsteroidal Antiinflammatory Drugs

- Feverfew
- Gingko
- Ginseng
- St. John's wort
- Uva-ursi

WWW.HIGHDENT.IR

Box 20-10 Detrimental Effects of Tobacco

1. Decrease blood flow (vasoconstriction)
2. Tissue hypoxia
3. Collagen deposition
4. Prostacyclin formation
5. Increases platelet aggregation
6. Polymorphonuclear neutrophil dysfunction
7. Increases fibrinogen
8. Blood viscosity
9. Systemic increase in epinephrine, norepinephrine

adults still smoke. Approximately 23% of men and 19% of women smoke cigarettes.[176] The use of tobacco has been implicated in many adverse systemic outcomes, including tooth loss and dental implant failure.[177,178]

A nationwide oral health survey established a definite association between smoking and poorer levels of periodontal health, even after adjustments had been made for age, sex, race, and oral hygiene habits.[179] Similar findings were documented in a study that established a relationship between periodontal attachment loss and smoking.[180] In addition, tobacco may be considered as a complicating factor in periodontal disease because it increases bone loss.[181,182]

In fact, the whole stomatognathic system suffers from the effect of tobacco byproducts (Box 20-10). Tobacco smoke decreases polymorphonuclear leukocyte activity, resulting in lower motility, a lower rate of chemotactic migration, and reduced phagocytic activity. These conditions contribute to a decreased resistance to inflammation, infection, and impaired wound healing potential.[183,184] Smoking is also associated with decreased calcium absorption.[185] Additional findings demonstrate a reduced mineral content in the bone of aging smokers and, to a greater degree, in postmenopausal female smokers.[185,186] The association of tobacco with intraoral carcinoma is well documented.

Reports in the literature demonstrate lower success rates for endosteal implants in smokers.[187-189] Failures seem to occur more in the maxilla than in the mandible and appear to occur in clusters.[187-190] However, similar maxillary implant survival rates have been observed in smokers, provided sufficient healing time, progressive bone loading, and prophylaxis procedures are implemented.[191,192]

With the possible detrimental effects of smoking on implants, it is recommended that patients be informed in detail about the risks of smoking and the possible consequences. Protocols have been recommended on smoking cessation before implant surgery. Smoking cessation after implant surgery has also been shown to improve implant survival.[193] Ideally, the patient is instructed to cease smoking for 2 weeks before surgery to allow for reversal of increased blood viscosity and platelet adhesion. Smoking cessation is continued for 8 weeks after implant surgery, which coincides with the osteoblastic phase of bone healing.[187] This has been shown to increase wound healing capabilities and reverse subgingival microflora.[194]

When incision line opening after surgery occurs, smoking will delay the secondary healing, contaminate a bone graft, and contribute to early bone loss during initial healing. Therefore the planning for implant surgeries with block bone grafts, especially C–h bone grafts, should emphasize the need for a smoking cessation protocol.

Dental Implant Implications

The implant team should advise potential implant patients of the detrimental effects that smoking has on their oral and systemic health. Complications must be discussed and highlighted in the informed consent. Patients should be encouraged to start a smoking cessation program before implant treatment. Smoking is not an absolute contraindication; however, the risks and possible morbidity on the respective procedures must be evaluated.

Psychological Diseases

Dental implant success may be compromised by psychological diseases such as anxiety and depression. One common type of psychological disease, depression, has been characterized as one of the leading causes of premature death and disability in the world. It is estimated that by the year 2020, depression will be second to ischemic heart disease as the leading cause of death.[195]

Depression is most often seen in patients who have a familial history of the disease, in association with chronic systemic disease, and in the elderly. Dental manifestations of this disease include the side effects from antidepressant medications, increase in caries and periodontal disease, increased smoking, xerostomia, chronic facial pain, and temporomandibular joint dysfunction.[196]

Dental Implant Management

Patients with active depression may have management problems, especially with the stress involved in undergoing implant procedures. Although this disease is episodic, special care must be taken in the treatment and monitoring of these patients. A thorough history should be taken with possible medical consultation in select cases.

Alcohol Use

Ethyl alcohol is one of the most widely used mood-altering drugs in the world. More than 95% of smokers also drink alcohol.[197] Alcoholism has been associated

WWW.HIGHDENT.IR

with diseases such as liver and metabolic dysfunction, bone marrow suppression resulting in bleeding complications, predisposition to infection, and delayed soft tissue healing.[198] The direct effect on bone includes decreased formation, increased resorption, decreased osteoblast function,[199] decreased wound healing, and increased parathyroid hormone secretion, which leads to a lower bone density.[200] However, it has been shown that withdrawal of alcohol can reverse the negative effects on osteoblast function in a matter of days.[201]

IRRADIATION

The treatment for cancer in the head and neck region is usually a combination of surgery, radiotherapy, and sometimes chemotherapy. Unfortunately, all of these treatment modalities have the potential to cause adverse effects on the soft and hard tissues of the oral cavity.[201] Surgical treatment of head and neck cancer usually results in anatomical impairment, leading to osseous defects. Head and neck radiotherapy results in progressive fibrosis of blood vessels and soft tissues, xerostomia, and decrease in the bone-healing quality of the jaws (Box 20-11).[202] Chemotherapeutic agents are associated with numerous wound healing and immunosuppressive states.

With radiotherapy, acute effects include mucositis, altered salivary function, and risk of mucosal infection. Long-term results include the tissue becoming hypovascular, hypoxic, and hypocellular. This is caused by changes in the vascularity and cellularity of hard and soft tissue, damage to the salivary glands, and increased collagen synthesis that results in fibrosis.[203] Because of these detrimental effects on the bone, wound repair and healing are significantly reduced after surgical procedures. When exposed to high levels of radiation, bone undergoes irreversible physiologic changes that include narrowing of the vascular channels (endarteritis), diminished blood flow, and loss of osteocytes. In time, the bone becomes nonvital, which leads to limited remodeling and healing potential.

Box 20-11 Irradiation Tissue Changes

Early

Salivary disorders
Skin/oral mucosa changes

Late

Demineralization
Fibrosis
Increased susceptibility to infection
Avascular necrosis

From Granstrom G, Jacobsson M, Tjellstrom A: Titanium implants in irradiated tissues: benefits from hyperbaric oxygen, *Int J Oral Maxillofac Implants* 7:15-25, 1992.

Radiotherapy to Previously Placed Implants

There exist very few studies on the effects of radiotherapy on preexisting dental implants. Short-term data show very minimal complications and failures.[204] However, in longer-term studies, failure rates seem to be higher.[205] At this time, more studies need to be conducted for conclusive results.

Implant Placement after Radiotherapy

In implant dentistry, there are many controversial issues in the dental implant rehabilitation of the irradiated patient. These issues include the amount of time after radiotherapy for the initiation of implant therapy and the use of hyperbaric oxygen therapy. With respect to the healing of dental implants, the ability of the bone to osseointegrate with implants has been shown to be dependent on the area of radiation, radiation dosage, and time elapsed since radiation exposure.[206] Implants into irradiated bone are associated with an increased risk of osteoradionecrosis, especially with irradiation above 50 Gy.[201,207] With respect to time, there are two antagonistic effects on the healing of irradiated tissues. A short-term positive effect has been shown to result in the improvement of the reduced bone-healing capacity.[208] A long-term negative effect has been shown to result in increasing vascular damage.[208]

The time between radiotherapy to implant placement does seem to have effects on the prognosis of implants. Most studies have shown that the longer the period for implant placement after radiotherapy, the higher the success rate and the lower the risk of osteoradionecrosis.[209] However, this subject is still controversial because various investigators have recommended different periods to initiate implant treatment ranging from 3 to 6 months,[210] 12 months,[211] and 24 months.[212]

Caution must be emphasized on past radiation therapy as earlier forms of radiation therapy (pre-1980s) were of lower energy, in contrast to current higher-energy levels that are less destructive. Because of this lower energy and associated higher destructive radiotherapy, progressive endarteritis has been shown to take place, which increases over time.[208]

The amount of radiation that is needed to cause the adverse effects has also been a topic of concern. Studies have shown that 50 Gy is the turning point with >50 Gy (71% survival) versus <50 Gy (84% survival). Therefore the presently available literature states that implant placement surgery may be completed on patients who have been irradiated at doses lower than 50 Gy.[213,214] Unfortunately, very few patients receiving doses above 50 Gy have been rehabilitated with implants. On the other hand, a study has shown that patients treated with implants who have received doses >120 Gy, have an implant survival rate that is very low with an increased risk of osteoradionecrosis (ORN).[215] Additionally, the dose given in Gy (formerly rad) can be

WWW.HIGHDENT.IR

misleading because the total number of fractions is not given. A more accurate evaluation of absorbed radiation dose has been termed the "cumulative radiation effect," which is calculated by[216]:

$$\frac{\text{Total time of TX}}{\text{(No. of TXs)}} \times \text{(Dose per TX)} \times \text{(No. of TXs)}$$

Studies have shown that implants placed in patients with a cumulative radiation effect of 18 to 20 (approximately 48 to 65 Gy standard fractionation) have a rather high success rate. Other reports have shown doses above a cumulative radiation effect of 40 (approximately 120 Gy standard fractionation) exhibit a high degree of failure.[205,217]

Osteoradionecrosis

The most significant risk in placing implants into irradiated bone is ORN, which is an irreversible, devitalization of irradiated bone that is characterized by necrotic, soft bone that fails to heal properly. The pathophysiologic mechanism is an imbalance in oxygen demand and oxygen availability, which is caused by endarteritis of the blood vessels. Clinical symptoms include pain, exposed necrotic bone, pathologic fractures, and suppuration.[218] The incidence of ORN is twice as high in dentate patients in comparison to edentulous patients, and the use of alcohol and tobacco leads to a higher onset rate.[219] Studies have shown the overall incidence of ORN after radiotherapy to be from 3% to 22%.[220] It is this risk that favors discretion in treating patients with radiation to the implant site before surgery. It is suggested the treatment is rendered by experienced clinicians, with a range of backgrounds and facilities including hyperbaric oxygen.

Hyperbaric Oxygen Therapy

One treatment to minimize the possibility of ORN is the use of hyperbaric oxygen. Prophylactic hyperbaric oxygen has been advocated to increase oxygen tension in irradiated bone, which will promote capillary angiogenesis and bone formation.[211] Recent data show oxygen under hyperbaric conditions acts synergistically with growth factors, which stimulates bone growth and turnover and also may act as a growth factor itself.[221] Hyperbaric oxygen has also been shown to act as a stimulator of osseointegration by increasing new bone formation, increasing bone turnover, and increasing the vascular supply to irradiated bone.[209] Studies have shown a 24% reduction of ORN in patients treated with hyperbaric oxygen versus antibiotic therapy.[222] The use of hyperbaric oxygen has been controversial. It was first introduced in the 1970s for the management of irradiated-damaged tissues. This was followed by a protocol using hyperbaric oxygen for irradiated patients undergoing dental implant surgery.[222] This protocol consisted of exposing the patient to short, intermittent, 100% oxygen inhalation at a pressure greater than 1 atmosphere. Usually, a patient receives approximately 20 treatments, for 90 minutes before placement followed by 10 minutes after placement.

Table 20-18 Protocol for the Treatment of Patients Receiving Radiation Therapy

RADIATION DOSE	TREATMENT
<20 Gy cumulative (approximately <50 Gy fractionation)	Informed consent, aseptic technique
>20 Gy cumulative (approximately >50 Gy fractionation)	Medical consultation, hyperbaric oxygen
Radiation therapy >20 years ago	Medical consultation, possible referral to cancer institution or hospital

Dental Implant Implications

Because of the higher incidence of dental implant failure in irradiated bone, special attention must be given to the irradiated patient. A thorough medical history including area of radiation exposure, type, and total cumulative dose must be determined and evaluated before implant therapy. With the use of hyperbaric oxygen, the rate of ORN and implant failure has been shown to be reduced. A generic protocol for the treatment of the prospective dental implant patient is shown in Table 20-18.[223]

Irradiation Patient Prosthetics

Because of the oral effects of radiotherapy, an implant-supported prosthesis (FP-1, FP-2, FP-3) is recommended over a soft tissue prosthesis (RP-4, RP-5). This will reduce the possible soft tissue irritation that is associated with postradiotherapy patients wearing removable prostheses.

References

1. Weijant RJ: Characteristics associated with the loss and perio implant tissue health of endosseous dental implants, *Int J Oral Maxillofac Implants* 7:367-372, 1992.
2. Redding SW, Montgomery ED: *Dentistry and systemic diseases*, Portland, Ore, 1990, JBK Publishing.
3. Misch CE: Medical evaluation of the implant candidate. Part I: vital signs and urinalysis, *J Oral Implant* 9:556-570, 1981.
4. Misch CE: Analysis of medical history pinpoints conditions that contraindicate implants, *Dentist* 67:23-24, 1989.
5. Dycht K: *Age wave: the challenges and opportunities of an aging America*, New York, 1988, St Martin's Press.
6. US Bureau of the Census: *Population reports, population estimates*, 25:1024, 1988.
7. Zamost B, Benumof JL: Anesthesia in geriatric patients. In Katz J, Benumof JL, Kadis LB, editors: *Anesthesia and uncommon diseases: pathophysiologic and clinical correlation*, ed 2, Philadelphia, 1976, WB Saunders.
8. Alder M, Kitchen S, Jrion A: *Data book on the elderly: a statistical portrait*, Washington, DC, 1987, US Department of Health and Human Services.

WWW.HIGHDENT.IR

9. American Association of Retired Persons: A profile of older Americans, *AARP Magazine,* 1988.
10. Levy SM, Baker KA, Semla TP et al: Use of medications with dental significance by a non-institutionalized elderly population, *Gerodontics* 4:119-125, 1988.
11. Heeling DK, Lemke JH, Semla TP et al: Medication use characteristics in the elderly. The Iowa 65+ rural health study, *J Am Geriatr Soc* 35:4-12, 1987.
12. Miller CS, Kaplan AL, Guest GF: Documenting medication use in adult dental patients, 1987-1991, *J Am Dent Assoc* 123:41-48, 1992.
13. Wynn RL: The top 50 prescription medications dispensed in pharmacies in 2004, *Gen Dent* 318-321, 2005.
14. Top 200 drugs of 1995, *Pharmacy Times,* pp 29-30, April 1996.
15. Moorthy AP, Coghlan K, O'Neil R: Drug therapy among dental outpatients, *Br Dent J* 156:261, 1984.
16. Becker DE, editor: *Drug therapy in dentistry,* Westland, Mich, 1995, Hayden-McNeil.
17. Pal S: Six drug classes cause most ADRs, *US Pharmacist* (hospital ed) 20:16, 1993.
18. *USP Drug information for the health care professional,* ed 15, vol 1, Rockville, Md, 1995, US Pharmacopeial Convention.
19. Marek CL: Avoiding prescribing errors, a systematic approach, *J Am Dent Assoc* 127:617-623, 1996.
20. Gage TW, Pickett FA: *Dental drug reference,* ed 2, St Louis, 1996, Mosby.
21. Malamed SF: Physical and psychological evaluation. In Malamed SF, editor: *Sedation: a guide to patient management,* ed 3, St Louis, 1995, Mosby.
22. Gilbert GH, Minaker KL: Principles of surgical risk assessment of the elderly patient, *J Oral Maxillofac Surg* 48:972-979, 1990.
23. Misch Implant Institute: *Patient evaluation booklet,* Tequesta, Fla, 1988, Nierman Practice Management.
24. McCarthy FM: Vital signs—the six-minute warning, *J Am Dent Assoc* 100:682-691, 1980.
25. Halstead C, editor: *Physical evaluation of the dental patient,* St Louis, 1982, Mosby.
26. Hajjar I, Kotchen TA: Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988-2000, *JAMA* 290:199-206, 2003.
27. Sabes WR, Green S, Craine C: Value of medical diagnostic screening tests for dental patients, *J Am Dent Assoc* 80:133-136, 1970.
28. Sones ST, Fazio R, Setkowicz A et al: Comparison of the nature and frequency of medical problems among patients in general specialty and hospital dental practices, *J Oral Med* 38:58, 1985.
29. *Implant criteria workshop,* Dearborn, Mich, 1978, American Academy of Implant Dentistry.
30. Misch CE: Medical evaluation of the implant candidate. Part II: Complete blood count and bleeding disorders, *Int J Oral Implant* 10:363-370, 1982.
31. Jolly DE: Interpreting the clinical laboratory, *Cal Dent Assoc J* 23:32-40, 1995.
32. American Association for Clinical Chemistry: *The coagulation cascade,* 2004. Available at http://www.labtestsonline.org/images/coag_cascade.pdf. Accessed September 27, 2007.
33. Corman L, Bolt RJ, editors: Medical evaluation of the pre-operative patient, *Med Clin North Am* 63:6, 1979.
34. Sherlock S: *Diseases of the liver and biliary system,* Oxford, 1975, Blackwell Scientific.
35. Hirsch J, Dalan JE, Deykin D et al: Oral anticoagulants: mechanism of action, clinical effectiveness, and optimal therapeutic range. *Chest* 102(suppl):312-326, 1992.
36. Lab Tests Online: *Comprehensive metabolic panel,* 2005. Available at http://www.labtestsonline.org/understanding/analytes/cmp/glance.html. Accessed September 27, 2007.
37. Johnson BS: Altered hemostasis: considerations for dental care, *Cal Dent Assoc J* 23:41-54, 1995.
38. Bennett B: Coagulation pathways: inter-relationships and control mechanisms, *Semin Hematol* 14:301, 1977.
39. Misch CE: Medical evaluation of the implant candidate. Part III. SMA 12/60, *J Oral Implant* 9:556-570, 1981.

39a. Misch CE: Medical evaluation. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.

40. Raslavicus PA, Mei Shen E: Laboratory diagnosis by chemical methods, *Dent Clin North Am* 18:155-170, 1974.
41. Burt VL, Whelton P, Roccella EJ et al: Prevalence of hypertension in the US adult population: results from the Third National Health and Nutrition Examination Survey, 1988-1991, *Hypertension* 25:305-313, 1995.
42. World Health Report 2002: *Reducing risks, promoting healthy life,* Geneva, Switzerland, 2002, World Health Organization.
43. Franklin SS, Gustin W, Wong ND et al: Hemodynamic patterns of age-related changes in blood pressure: the Framingham heart study, *Circulation* 96:308-315, 1997.
44. Chobanian AV, Bakris GL, Black HR et al: Seventh Report of the National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, *Hypertension* 42:1206, 2003.
45. Leung RS, Bradley TD: Sleep apnea and cardiovascular disease, *Am J Respir Crit Care Med* 164:2147-2167, 2001.
46. Peppard PE, Young T, Palta M et al: Prospective study of the association between sleep-disordered breathing and hypertension, *N Engl J Med* 342:1378-1384, 2000.
47. Muzyka BC, Glick M: The hypertensive dental patient, *J Am Dent Assoc* 128:1109-1120, 1997.
48. Ellis JS, Seymour RA, Steele JG et al: Prevalence of gingival overgrowth induced by calcium channel blockers: a community-based study, *J Periodontol* 70:63-67, 1999.
49. Velasquez MT: Angiotensin II receptor blockers: a new class of antihypertensive drugs, *Arch Fam Med* 5:351-356, 1996.
50. Houston MC, Welk M, Gray J et al: The effects of nonsteroidal antiinflammatory drugs on blood pressures of patients with hypertension controlled by verapamil, *Arch Intern Med* 155:1049-1054, 1995.
51. Brown J, Dollery C, Valdes G: Interaction of nonsteroidal anti-inflammatory drugs with antihypertensive and diuretic agents: control of vascular reactivity by endogenous prostanoids, *Am J Med* 81(Suppl 2B):43-57, 1986.
52. Herman WW, Konzelman JL, Prisan M: New national guidelines on hypertension: a summary for dentistry, *J Am Dent Assoc* 135; 576-584, 2004.
53. Sones S, Fazio R, Fang L, editors: *Principles and practice of oral medicine,* Philadelphia, 1984, WB Saunders.
54. Gordy FM, LeJeune RC, Copeland LB: The prevalence of hypertension in a dental school patient population, *Quintessence Int* 32:691-695, 2001.
55. Collins R, Peto R, MacMahon S et al: Blood pressure, stroke and coronary heart disease. Part 2. Short-term reductions in blood pressure: overview of randomised drug trials in their clinical epidemiological context, *Lancet* 335:827-838, 1990.
56. Herman WW, Konzelman JL: Angina, an update for dentistry, *J Am Dent Assoc* 127:98-104, 1996.
57. Goldman L, Caldera D: Risks of general anesthesia and elective operations in the hypertensive patient, *Anesthesiology* 50:285, 1979.

WWW.HIGHDENT.IR

58. Humphries JO: Survival after myocardial infarction: prognosis and management, *Mod Concepts Cardiovasc Dis* 46:51, 1977.
59. Pell S, D'Alonzo CA: Immediate mortality and five year survival of employed men with a first myocardial infarction, *N Engl J Med* 270:915, 1964.
60. Foster E, Davis KB, Carpenter JA et al: Risk of noncardiac operation in patients with defined coronary disease: the coronary artery surgery study (CASS) registry experience, *Ann Thorac Surg* 41:42, 1986.
61. Malamed SF: *Sedation: a guide to patient management*, ed 2, St Louis, 1989, Mosby.
62. *Heart disease health center*, 2007. Available at http://www.webmd.com/heart-disease/default.htm. Accessed September 27, 2007.
63. Heart failure (congestive heart failure). In *Merck onlinemedical library*, 2005. Available at http://www.merck.com/mmpe/sec07/ch074/ch074b.html#CIHFIAJG. Accessed September 27, 2007.

63a. American Dental Association: antibiotics and your heart: new guidelines from the American Heart Association, *J Am Dent Assoc* 138:920, 2007.

63b. Wilson W, Taubert KA, Gewitz M et al: Provention of infective codocarditis, guidelines from the American Heart Association: a guideline from the American Heart Association rheumatic fever, endocarditis, and Kawasaki disease committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisiciplinary Working Group, *Circulation* April 19, 2007. Available at http://circ.ahajournals.org/cgi/content/abstract/CIRCULATIONAHA.106.183095vI. Accessed September 20, 2007.

64. Burkett LW, Burn CG: Bacteremias following dental extraction: demonstration of source of bacteria by means of a non-pathogen (Serratia marcescens), *J Dent Res* 16:521, 1937.
65. Korn VA, Schaffer EM: A comparison of the postoperative bacteremias induced following different periodontal procedures, *J Periodontol* 33:226, 1962.
66. Bender IB, Seltzer S, Yermish M: The incidence of bacteremias in endodontic manipulation, *Oral Surg* 13:353, 1960.
67. Cameron IW: SBE in an edentulous patient: a case report, *Br Med J* 1:821, 1971.
68. Dajani AS, Taubert KA, Wilson W et al: Prevention of bacterial endocarditis. Recommendations by the American Heart Association, *J Am Med Assoc* 277:1794-1801, 1997.
69. Dajani AS, Bawdon RE, Berry MC: Oral amoxicillin as prophylaxis for endocarditis: what is the optimal dose? *Clin Inject Dis* 18:157-160, 1994.
70. Fluckiger U, Franciolo P, Blaser J et al: Role of amoxicillin serum levels for successful prophylaxis of experimental endocarditis due to tolerant streptococci, *J Inject Dis* 169:397-400, 1994.
71. von Wowern N: General and oral aspects of osteoporosis: a review, *Clin Oral Invest* 5:71-82, 2001.
72. Chanavaz M: Patient screening and medical evaluation for implant and preprosthetic surgery, *J Oral Implantol* 24:222-229, 1998.
73. Harris MI: Summary. In *Diabetes in America*, ed 2, Bethesda, Md, 1995, National Diabetes Data Group; National Institute of Diabetes and Digestive and Kidney Diseases; National Institutes of Health; National Institutes of Heath publication 95-1468.
74. Centers for Diseases Control and Prevention: *Diabetes data and trends.* Available at http://apps.nccd.cdc.gov/ddtstrs/. Accessed September 27, 2007.
75. Orchard TJ, Dorman JS, Maser RE et al: Prevalence of complications in IDDM by sex and duration. Pittsburgh Epidemiology of Diabetes Complications Study II, *Diabetes* 39:1116-1124, 1990.
76. Darnell JA, Saunders MJ: Oral manifestations of the diabetic patient, *Tex Dent J* 107:23-27, 1990.
77. World Health Organization: *Diabetes mellitus fact sheet, 2000.* Available at http://www.who.int/mediacentre/factsheets/fs312/en/index.html. Accessed July 5, 2007.
78. Nevins ML, Karimbux NY, Weber HP et al: Wound healing around endosseous implants in experimental diabetes, *Int J Oral Maxillofac Implants* 13:620-629, 1998.
79. McCracken M, Lemons JE, Rahemtulla F et al: Bone response to titanium alloy implants placed in diabetic rats, *Int J Oral Maxillofac Implants* 15:345-354, 2000.
80. Chanavaz M: Patient screening and medical evaluation for implant and preprosthetic surgery, *J Oral Implant* 24:222-229, 1998.
81. Peled M, Ardekian L, Tagger-Green N et al: Dental implants in patients with type 2 diabetes mellitus: a clinical study, *Implant Dent* 12:116-122, 2003.
82. Balshi TJ, Wolfinger GJ: Dental implants in the diabetic patient: a retrospective study, *Implant Dent* 8:355-359, 1999.
83. Sugerman PB, Barber MT: Patient selection for endosseous dental implants: oral and systemic considerations, *Int J Oral Maxillofac Implants* 17:191-201, 2002.
84. Raposo V: Simple diagnosis and treatment planning of the implant patient, *J Colo Dent Assoc* 80:16-17, 2001.
85. Tonetti MS: Risk factors for osseointegration, *Periodontology* 17:55-62, 2000.
86. Shernoff AF, Colwell JA, Bingham SF: Implants for Type 2 diabetic patients: VA implants in Diabetes Study Group, *Implant Dent* 3:183-185, 1994.
87. *Top 300 prescriptions for 2005.* Available at http://www.rxlist.com/top200.htm. Accessed July 5, 2007.
88. Attard NJ, Zarb GA: A study of dental implants in medically treated hypothyroid patients, *Clin Implant Dent Relat Res* 4:220-231, 2002.
89. Irvine WJ, Toft AD: The diagnosis and treatment of thyrotoxicosis, *Clin Endocrinol* 5:687, 1976.
90. Liddle GW, Melmon KC: The adrenals. In Williams RH, editor: *Textbook on endocrinology*, ed 5, Philadelphia, 1974, WB Saunders.
91. Misch CE, Moore P: Steroids and the reduction of pain, edema, and dysfunction in implant dentistry, *Int J Oral Implant* 6:27-31, 1989.
92. Dummett CO, Barens C: Oromucosal pigmentation: an updated literary review, *J Periodontol* 42:726, 1971.
93. McCarthy FM: Adrenal insufficiency. In McCarthy FM, editor: *Essentials of safe dentistry for the medically compromised patient*, Philadelphia, 1989, WB Saunders.
94. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing and progressive bone loading, *Int J Oral Implant* 6:23-31, 1990.
95. Konotey-Ahuke FI: The sickle cell diseases, *Arch Intern Med* 133:611, 1974.
96. Marder MZ: Medical conditions affecting the success of dental implants, *Compendium* 25:739-764, 2004.
97. American Lung Association: *Chronic obstructive pulmonary disorder (COPD) fact sheet*, Available at http://www.lungusa.org/site/pp.asp?c=dvLUK9O0E&b=3502. Accessed July 5, 2007.

WWW.HIGHDENT.IR

98. Burrows B: Physiologic variants of chronic obstructive lung disease, *Chest* 58(suppl 2):415, 1970.
99. Conn HO: Cirrhosis. In Schiff L, editor: *Diseases of the liver,* ed 45, Philadelphia, 1975, JB Lippincott.
100. Grant BF, Harforf TC, Dawson DA et al: Prevalence of DSM-IV alcohol abuse and dependence, United States, 1992, *Alcohol Health Res World* 18:243-248, 1994.
101. Lieber CS: Medical disorders of alcoholism, *N Engl J Med* 333:1058-1065, 1995.
102. Dufour C: Chronic liver disease and cirrhosis. In Everhart J, editor: *Digestive disease in the United States: epidemiology and impact,* Washington DC, Superintendent of Documents, 1993, Government Printing Office.
103. Baglink DJ, Wergedal JE, Yamamoto K et al: Systemic factors in alveolar bone loss, *J Prosthet Dent* 31:486-505, 1977.
104. Bone Health and Osteoporosis: *A Report of the Surgeon General,* Rockville, Md: Department of Health and Human Services, 2004.
105. Chestnut CH: Osteoporosis an undiagnosed disease, *JAMA* 286:2865-2866, 2001.
106. National Osteoporosis Foundation: *Boning up on osteoporosis, a guide to prevention and treatment,* Washington, DC, 1991, National Osteoporosis Foundation.
107. Jeffcoat M: The association between osteoporosis and oral bone loss, *J Periodontol* 76(11 suppl):2125-2132, 2005.
108. Dempster DW: Bone remodeling. In Coe FL, Favis MJ, editors: *Disorders of bone and mineral metabolisms,* New York, 1992, Raven Press.
109. Cann CE, Genant HK, Kolb FO et al: Quantitative computed tomography for prediction of vertebral fracture risk, *Bone* 6:1, 1985.
110. Heasman PA: The role of nonsteroidal antiinflammatory drugs in the management of periodontal disease, *J Dent Res* 16:247-257, 1988.
111. Ortman LF, Hausman E, Dunford RG: Skeletal osteopenia and residual ridge resorption, *J Prosthet Dent* 61:321-325, 1989.
112. Wakley GK, Baylink DJ: Systemic influences on the bone response to dental and orthopedic implants, *J Oral Implant* 14:285-311, 1988.
113. August M, Chung K, Chang Y et al: Influence of estrogen status on endosseous implant integration, *J Oral Maxillofac Surg* 59:1285-1289, 2001.
114. Lees B, Stevenson JC: Evaluation of dual energy x-ray absorptiometry and comparison with dual-photon absorptiometry, *Osteoporosis Int* 2:146-152, 1992.
115. Kellie SE: Diagnostic and therapeutic technology assessment (DATTA): measurement of bone density with dual energy x-ray absorptiometry (Dexa), *JAMA* 267:286-294, 1992.
116. Farley JR, Wergedal JE, Baylink DJ: Fluoride directly stimulates proliferation and alkaline phosphatase activity of bone forming cells, *Science* 222:330, 1983.
117. Minsk L, Polson AM: Dental implant outcomes in postmenopausal women undergoing hormone replacement, *Compend Contin Educ Dent* 19:859-866, 1998.
118. Friberg B, Ekestubbe A, Mellstrom E et al: Brånemark implants and osteoporosis: a clinical exploratory study, *Clin Impl Dent Relat Res* 3:50-56, 2001.
119. Henrikson P: Periodontal disease and calcium deficiency: an experimental study in the dog, *Acta Odontol Scand* 26(suppl 50):1-132, 1968.
120. Obisesan AA, Lagundoye SB, Daramola JO et al: The radiologic features of fibrous dysplasia of the craniofacial bones, *Oral Surg* 44:949, 1977.
121. McGousar DA: Clinical problems in Paget's disease affecting the jaws, *Br J Oral Surg* 11:230, 1974.
122. Sager RD, Thesis RM: Dental implants placed in a patient with multiple myeloma. Report of case, *J Am Dent Assoc* 121:699-701, 1990.
123. American Dental Association, American Academy of Orthopaedic Surgeons: Antibiotic prophylaxis for dental patients with total joint replacements—advisory statement, *J Am Dent Assoc* 128:1004-1008, 1997.
124. American Dental Association, American Academy of Orthopedic Surgeons: Antibiotic prophylaxis for dental patients with total joint replacements, *J Am Dent Assoc* 134:895-899, 2003.
125. Rubin R, Salvati EA, Lewis R: Infected total hip replacement after dental procedures, *Oral Surg Oral Med Oral Pathol* 41:13-23, 1976.
126. Hansen AD, Osmon RD, Neeson CL: Prevention of deep prosthetic joint infection, *J Bone Joint Surg Am* 78-A:458-471, 1996.
127. Pallasch TJ, Slots J: Antibiotic prophylaxis and the medically compromised patient, *Periodontology* 2000: 107-138, 1996.
128. McGowan DA: Dentistry and endocarditis, *Br Dent J* 169:69, 1990.
129. Clarke A: Hypohidrotic ectodermal dysplasia, *J Med Genet* 24:659-663, 1987.
130. Kearns G, Sharma A, Perrott D et al: Placement of endosseous implants in children and adolescents with hereditary ectodermal dysplasia, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 88:5-10, 1999.
131. Bergendal B, Bergendal T, Hallonsten AL et al: A multidisciplinary approach to oral rehabilitation with osseointegrated implants in children and adolescents with multiple aplasia, *Eur J Orthod* 18:119-129, 1996.
132. Guckes AD, McCarthy GR, Brahim J: Use of endosseous implants in a 3-year-old child with ectodermal dysplasia: case report and 5-year follow-up, *Pediatr Dent* 19:282-285, 1997.
133. Smith RA, Vargervik K, Kearns G, et al: Placement of an endosseous implant in a growing child with ectodermal dysplasia, *Oral Surg Oral Med Oral Pathol* 75:669-673, 1993.
134. Davarpanah M, Moon JW, Yang LR, et al: Dental implants in the oral rehabilitation of a teenager with hypohidrotic ectodermal dysplasia: report of a case, I*nt J Oral Maxillofac Implants* 12:252-258, 1997.
135. Binon PP, Fowler CN: Implant-supported fixed prosthesis treatment of a patient with Sjögren's syndrome: a clinical report, *Int J Oral Maxillofac Implants* 8:54-58, 1993.
136. Klippel J: *Systemic sclerosis and related syndromes: primer on the rheumatic diseases,* ed 11, 1997, The Arthritis Society.
137. Hasler P: Biological therapies directed against cells in autoimmune disease. *Springer Semin Immunopathol* 27:443-456, 2006.
138. Vital EM, Emery P: Advances in the treatment of early rheumatoid arthritis, *Am Fam Physician* 72:1002-1004, 2005.
139. Joint United Nations Programme on HIV/AIDS (2006): *Overview of the global AIDS epidemic, 2006 report on the global AIDS epidemic.* Available at http://www.unaids.org/en/HIV_data. Accessed June 8, 2006.
140. Shetty K, Achong R: Dental implants in the HIV-positive patient—case report and review of the literature, *Gen Dent* 53:434-437, 2005.
141. Achong RM, Shetty K, Arribas A et al: Implants in HIV-positive patients: 3 case reports, *J Oral Maxillofac Surg* 64:1199-1203, 2006.
142. Cruzan SM: *FDA advisory committee recommends approval of osteoporosis drug,* 1995. Available at http://www.fda.

WWW.HIGHDENT.IR

gov/bbs/topics/ANSWERS/ANS00673.html. Accessed September 26, 2007.
143. Fleisch H, Russell RG, Bisaz S et al: The influence of pyrophosphate analogues (diphosphonates) on the precipitation and dissolution, *Calcif Tissue Res* Suppl 10-10a, 1968.
144. Miles AE: Phosphorus necrosis of the jaw: "phossy jaw," *Br Dent J* 133:203-206, 1972.
145. Sherk HH, Harrison S, Martland MD: The radium watch-dial workers and the evolution of industrial medicine, *N J Med* 98;49-52, 2001.
146. Carter GD, Goss AN, Doecke C: Bisphosphonates and avascular necrosis of the jaw: a possible association, *Med J Aust* 182:413-415, 2005.
147. Vudiniabola S, Pirone C, Williamson J et al: Hyperbaric oxygen in the therapeutic management of osteoradionecrosis of the facial bones, *Int J Oral Maxillofac Surg* 29:435-438, 2000.
148. Marx RE: Pamidronate (Aredia) and zoledronate (Zometa) induced avascular necrosis of the jaws: a growing epidemic, *J Oral Maxillofac Surg* 61:1115-1117, 2003.
149. Ruggiero SL, Mehrotra B, Rosenberg TJ et al: Osteonecrosis of the jaws associated with the use of bisphosphonates: a review of 63 cases, *J Oral Maxillofac Surg* 62:527-534, 2004.
150. Sparidans RW, Twiss IM, Talbot S: Bisphosphonates in bone diseases, *Pharm World Sci* 20:206-213, 1998.
151. Rogers MJ, Gordon S, Benford HL et al: Cellular and molecular mechanisms of action of bisphosphonates, *Cancer* 88(suppl); 2961-2978, 2000.
152. Fleisch H: Development of bisphosphonates, *Breast Cancer Res* 4:30-34, 2002.
153. Martin TJ, Grin V: Bisphosphonates—mechanisms of action, *Aust Prescriber* 23:130-132, 2000.
154. Chun YH, Foster BL, Lukasavage PA: Bisphosphonate modulates cementoblast behavior in vitro, *J Periodontol* 76:1890-1900, 2005.
155. Landesberg RL, Wilson T, Grbic JT: Bisphosphonate-associated osteonecrosis of the jaw: conclusions based on an analysis of case series, *Dent Today* August, 52-56, 2006.
155a. Greenspan Sl, Rosen HN, Parker RA: Early changes in serum N-telopeptide and C-telopeptide cross-linked collagent type 1 predict long-term response to alendronate therapy in elderly women, *J Clin Endocrinol Metab* 85:3537-3540, 2000.
155b. Marx RE: *Oral and intravenous bisphosphonate-induced osteonecrosis of the jaws: history, etiology, prevention, and treatment*, Chicago, 2007, Quintessence.
155c. Lin JH, Russell G: Pharmacodynamics of alendronate: an overview, *Int J Clin Pract Suppl* 101:18-26, 1999.
156. American Academy of Periodontology: *Statement on bisphosphonates*. Available at http://www.perio.org/resources-products/bisphosphonates.htm. Accessed August 26, 2005.
157. Jeffcoat MK: Safety of oral bisphosphonates: controlled studies on alveolar bone, *Int J Oral Maxillofac Implants* 21:349-353, 2006.
158. Blanchaert RH: Implants in the medically challenged patient, *Dent Clin N Am* 42:1, 1998.
159. Steiner M, Windchy A, Gould AR: Effects of chemotherapy in patients with dental implants, *J Oral Implantol* 21: 142-147, 1995.
160. Sager RD, Theis RM: Dental implants placed in a patient with multiple myeloma: report of case, *J Am Dent Assoc* 21:699-701, 1990.
161. McDonald AR, Pogrel A, Sharma A: Effects of chemotherapy on osseointegration of implants: a case report, *J Oral Implant* 24:11-13, 1998.
162. Wolfaardt J, Granstrom G, Friberg B et al: A retrospective study on the effects of chemotherapy on osseous integration, *J Facial Somato Protho* 2:99-107, 1996.
163. Campbell JH, Alvarado F, Murray RA: Anticoagulation and minor oral surgery: should the anticoagulation regimen be altered? *J Oral Maxillofac Surg* 58:131-135, 2000.
164. Webster K, Wilde J: Management of anticoagulation in patients with prosthetic heart valves undergoing oral and maxillofacial operations, *Br J Oral Maxillofac Surg* 38:124-126, 2000.
165. Palareti G, Legnani C, Guazzaloca G et al: Activation of blood coagulation after abrupt or stepwise withdrawal of oral anticoagulants—a prospective study, *Thromb Haemost* 72:222-226, 1994.
166. Wahl MJ: Myths of dental surgery in patients receiving anticoagulant therapy, *J Am Dent Assoc* 131:77-81, 2000.
167. Patrono C: Aspirin and human platelets: from clinical trials to acetylation of cyclooxygenase and back, *Trends Pharmacol Sci* 10:453-458, 1989.
168. Ferraris VA, Ferraris SP, Lough FC et al: Preoperative aspirin ingestion increases operative blood loss after coronary artery bypass grafting, *Ann Thorac Surg* 45:71-74, 1988.
169. Conti CR: Aspirin and elective surgical procedures (editorial), *Clin Cardiol* 15:709-710, 1992.
170. Watson CJ, Deane AM, Doyle PT et al: Identifiable factors in postprostatectomy haemorrhage: the role of aspirin, *Br J Urol* 66:85-87, 1990.
171. Ferraris VA, Swanson E: Aspirin usage and intraoperative blood loss in patients undergoing unexpected operations, *Surg Gynecol Obstet* 156:439-442, 1983.
172. Lawrence C, Sakuntabhai A, Tiling-Grosse S: Effect of aspirin and nonsteroidal antiinflammatory drug therapy on bleeding complications in dermatologic surgical patients, *J Am Acad Dermatol* 31:988-992, 1994.
173. Ardekian L, Gaspar R, Peled M et al: Does low-dose aspirin therapy complicate oral surgical procedures? *J Am Dent Assoc* 131:331-335, 2000.
174. Jackson G: CURE—clopidogrel's major advance, *Int J Clin Pract* 55:155, 2001.
175. Jeske AH, Suchko GD: ADA Council on Scientific Affairs and Division of Science, JADA: lack of scientific basis for routine discontinuation of oral anticoagulant therapy before dental treatment, *J Am Dent Assoc* 134:1492-1497, 2003.
175a. Phillips KA, Veenstr DL: Potential role of pharmocogenics in reducing adverse drug reactions, *JAMA* 286:2270-2279, 2001.
175b. U.S. Food and Drug Administration: *Index to drug-specific information*. Available at http://www.fda.gov/cder/consumerinfo. Accessed September 20, 2007.
176. American Cancer Association: *Questions about smoking, tobacco, and health.* Available at http://www.cancer.org/docroot/PED/content/PED_10_2x_Questions_About_Smoking_Tobacco_and_Health.asp. Accessed July 5, 2007.
177. Krall EA, Garvey AJ, Garcia RI: Alveolar bone loss and tooth loss in male cigar and pipe smokers, *J Am Dent Assoc* 130:57-64, 1999.
178. Tomar SL, Asma S: Smoking—attributable periodontitis in the United States: findings from NHANES III, *J Periodontol* 71:743-751, 2000.
179. Ismail AI, Burt BA, Eklund SA: Epidemiologic patterns of smoking and periodontal disease in the United States, *J Am Dent Assoc* 106:617-621, 1983.

WWW.HIGHDENT.IR

180. Schenkein HA, Gunsolley JC, Koertge TE et al: Smoking and its effect on early-onset periodontitis, *J Am Dent Assoc* 126:1107-1113, 1995.
181. Arno A, Schei O, Loudal A et al: Alveolar bone loss as a function of tobacco consumption, *Acta Odont Scand* 17:3-10, 1959.
182. Feldman RS, Bravacos JS, Rose CL: Association between smoking different tobacco products and periodontal disease indexes, *J Periodontol* 54:481-487, 1983.
183. Noble RC, Penny BB: Comparison of leukocyte count and function in smoking and nonsmoking young men, *Immunology* 12:550-555, 1975.
184. Jones JK, Triplett RG: The relationship of cigarette smoking to impaired intra-oral wound healing, *J Oral Maxillofac Surg* 50:237-239, 1992.
185. Krall EA, Dawson Hughes B: Smoking and bone loss among postmenopausal women, *J Bone Miner Res* 6:331-338, 1991.
186. Rudgren A, Mellstrom D: The effect of tobacco smoke on the bone mineral content of the aging skeleton, *Mech Aging Dev* 28:273-277, 1984.
187. Bain CA, Moy PK: The association between the failure of dental implants and cigarette smoking, *Int J Oral Maxillofac Impl* 8:609-615, 1993.
188. Lambert PM, Morris HF, Ochi S: The influence of smoking on a 3-year clinical success of osseointegrated dental implants, *Ann Periodontol* 5:79-89, 2000.
189. DeBruyn H, Collaert B: The effect of smoking on early implant failure, *Clin Oral Impl Res* 5:260-264, 1994.
190. Quirynen M, Naert I, Van Steenberghe D et al: The cumulative failure rate of the Bränemark system in the overdenture, the fixed partial and the fixed full prosthesis design a prospective study on 1273 fixtures, *J Head Neck Pathol* 10:43-53, 1991.
191. Dietsh Misch F, Misch CE, Cianciola L: Sinus grafts in a smoking population—a retrospective study Abstract AO.
192. Karoussis IK, Bragger U, Salvi GE et al: Effect of implant design on survival and success rates of titanium oral implants: a 10-year prospective cohort study of the ITI Dental Implant System, *Clin Oral Implants Res* 15:8-17, 2004.
193. Bain CA: Smoking and implant failure—benefits of a smoking cessation protocol, *Int J Oral Maxillofac Implants* 11:1667-1674, 1996.
194. Grossi SG, Zambon J, Machtei EE: Effects of smoking and smoking cessation on healing after mechanical periodontal therapy, *J Am Dent Assoc* 128:599-607, 1997.
195. Marder MZ: Medical conditions affecting the success of dental implants, *Compend Contin Educ Dent* 25:739-764, 2004.
196. Friedlander AH, Mahler ME: Major depressive disorder: psychopathology, medical management and dental implications, *J Am Dent Assoc* 32:629-638, 2001.
197. Casiglia J, Zambon J, Machtei EE: Effects of smoking and smoking cessation on healing after mechanical periodontal therapy, *J Am Dent Assoc* 28:599-607, 1997.
198. Rees TD: Oral effects of drug abuse, *Crit Rev Oral Biol Med* 3:163-184, 1992.
199. Pepersack T, Fuss M, Otero J et al: Longitudinal study of bone metabolism after ethanol withdrawal in alcoholic patients, *J Bone Miner Res* 7:383-387, 1992.
200. Koch JP, Dunson B: Factors affecting bone healing following implant surgery, *J Oral Implantol* 22:7-11, 1996.
201. Visch LL, Van Wass MAJ, Schmitz PIM et al: A clinical evaluation of implants in irradiated oral cancer patients, *J Dent Res* 81:856-859, 2002.
202. Granstrom G, Jacobsson M, Tjellstrom A: Titanium implants in irradiated tissues: benefits from hyperbaric oxygen, *Int J Oral Maxillofac Implants* 7:15-25, 1992.
203. Hancock PJ, Epstein JB, Sadler GR: Oral and dental management related to radiation therapy for head and neck cancer, *J Can Dent Assoc* 69:585-590, 2003.
204. Granstrom G, Tjellstrom A, Albrekttson T: Postimplantation irradiation for head and neck cancer treatment, *Int J Oral Maxillofac Implants* 8:495, 1993.
205. Granstrom G: Osseointegration in irradiated tissues. Experience from our first 100 treated patients, *J Oral Maxillofac Surg* 63:579-585, 1996.
206. Esposito M, Hirsch JM, Lekholm U et al: Biological factors contributing to failures of osseointegrated oral implants, *Eur J Oral Sci* 106:721-764, 1998.
207. Esser F, Wagner W: Dental implants following radical oral cancer surgery and adjuvant radiotherapy, *Int J Oral Maxillofac Implants* 12:552-557, 1997.
208. Jacobsson M: *On behavior after irradiation* [master's thesis], Goteborg, Sweden, 1985, University of Goteborg.
209. Granstrom G: Hyperbaric oxygen as a stimulator of osseointegration. *Adv Otorhinolaryngol* 54:33, 1988.
210. King MA, Casarett GW, Weber DA: A study of irradiated bone: I. Histopathologic and physiologic changes, *J Nucl Med* 20:1142-1149, 1979.
211. Albrekttson T: A multicenter report on osseointegrated oral implants, *J Prosthet Dent* 60:75-84, 1988.
212. Taylor TD, Worthington P: Osseointegrated implant rehabilitation of the previously irradiated mandible: results of a limited trial at 3 to 7 years, *J Prosthet Dent* 69;60-69, 1993.
213. Keller EE: Placement of dental implants in irradiated mandible. A protocol without adjunctive hyperbaric oxygen, *J Oral Maxillofac Surg* 55:972, 1997.
214. Keller EE, Tolman DE, Zuck SL et al: Mandibular endosseous implants and autogenous bone grafting in irradiated tissue: a 10-year retrospective study, *Int J Maxillofac Implants* 12:800, 1997.
215. Granstrom G: Placement of dental implants in irradiated bone: the case for using hyperbaric oxygen, *J Oral Maxillofac Surg* 64:812-818, 2006.
216. Kirk I, Gray WH, Watson ER: Cumulative radiation effect, *Clin Radiol* 22:145, 1971.
217. Granstrom G, Tjellstrom A, Brånemark PI: Bone anchored reconstruction of the irradiated head and neck cancer patient. *Otolaryngol Head Neck Surg* 108:334, 1993.
218. Altasalo K, Grenman R, Virolaine E et al: A modified protocol to treat early osteoradionecrosis of the mandible, *Undersea Hyperb Med* 22:161-170, 1995.
219. Curi MM, Dib LL: Osteoradionecrosis of the jaws: a retrospective study of the background factors and treatment in 104 cases, *J Oral Maxillofac Surg* 55:540-544, 1997.
220. Altasalo K: Bone tissue response to irradiation and treatment model of mandibular irradiation injury, *Acta Otolaryngol* 428:1-54, 1986.
221. Granstrom G: Pathophysiological basis for HBO in the treatment of healing disorders in radio-injured normal tissues. In *Proceedings of the 5th ECHM Consensus Conference*, Lisbon; 85-93, 2001.
222. Marx R, Johnson RP, Kline SN: Prevention of osteoradionecrosis: a randomized prospective clinical trial of hyperbaric oxygen versus penicillin, *J Am Dent Assoc* 111:49-54, 1985.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 21

Pharmacology in Implant Dentistry

Randolph R. Resnik, Carl E. Misch

Because of the increase in demand and use of dental implants in dentistry today, a thorough understanding of the indications and protocol for the use of pharmacologic agents in implant dentistry is essential. The morbidity of implant-related complications may on occasion be significant; therefore indicated drug selection and sufficient dosage levels of medications are preoperatively and postoperatively indicated. The scope of implant treatment often encompasses an older population with more complex cases. As a result, a greater understanding of compromised wound healing and inadequate immune systems with respect to pharmacology is essential.

In implant dentistry, there is no consensus on the pharmacologic protocol based on both the patient's health status and procedure type. Many practitioners use medications empirically or generically with respect to all procedures with little basis on scientific facts and studies. This chapter will provide the implant dentist with an overview of the pharmacokinetics and pharmacodynamics of various classifications of medications along with an understanding of the proper prescribing protocol used in implant dentistry today with respect to different patient and procedure characteristics.

ANTIMICROBIALS

An important complication to prevent after implant surgery is infection. Infection can lead to a multitude of problems, including pain, swelling, loss of bone, and possible failure of the implant. Because of the risk of morbidity from infections, antimicrobial therapy is an essential component of the surgical protocol. Although adverse effects are associated with antibiotic therapy, these are usually mild and infrequent. The most common antimicrobials used in implant dentistry consist of antibiotics (local and systemic) and antimicrobial rinses (0.12% chlorhexidine gluconate).

Antibiotics

The use and understanding of the various antibiotic regimens available in implant dentistry are beneficial for the initial success and long-term maintenance of implant therapy. Antibiotic therapy in implant dentistry may be classified as either prophylactic (prevent infection) or therapeutic (treat infection).

Prophylactic Antibiotics

In general surgery, including its subspecialties, principles of antibiotic prophylaxis are well established. Guidelines are specific related to the procedure, the type of antibiotic, and the dosage regimen.[1,2] The use of prophylactic antibiotics in dentistry has also been documented in the prevention of complications for patients at risk of developing infectious endocarditis and immunocompromised patients. However, in oral implantology, there exists no consensus on the use and indications for prophylactic antibiotics. Disadvantages with the use of antibiotics include development of resistant bacteria, adverse reactions and possible resultant lax surgical technique.[3-5] As a result, the need for prophylactic antibiotics in healthy patients, type of antibiotic, dosage, and duration of coverage is controversial. On the other hand, postoperative surgical wound infections can have a significant impact on the well-being of the patient and the survival of the implant. Documented cases of potential consequences of infection range from increased pain and edema to even patient death. According to Esposito and Hirsch, one of the main causes of dental implant failure may be due to bacterial contamination at implant insertion.[6]

A local inoculum must be present for a surgical wound infection to occur, to overcome the host's defenses and allow growth of the bacteria. This process has many variables including various host, local tissue, and systemic and microbial virulence factors. Antibiotic prophylaxis is only one component of this complex cascade; however, the efficacy and impact

WWW.HIGHDENT.IR

of antimicrobial prophylaxis has been proven to be significant.[3]

Several studies have concluded there is benefit of preoperative antibiotics for dental implantology.[7-9] In the most comprehensive and controlled study to date, 33 hospitals formed the Dental Implant Clinical Research Group and concluded that the use of preoperative antibiotics significantly improved dental implant survival, both in early and later stages. In the evaluation of 2973 implants, a significant difference was found with the use of preoperative antibiotics (4.6% failure) compared with no antibiotics (10% failure).[7,8]

The main goal of the use of prophylactic antibiotics is to prevent infection during the initial healing period from the surgical wound site, thus decreasing the risk of infectious complications of the soft and hard tissues. Although there is no conclusive evidence on the mechanism of preoperative antibiotics, most likely a greater aseptic local environment is achieved. A landmark study by Burke defined the scientific basis for the perioperative use of antibiotics to prevent surgical wound infection.[10] From this work, several accepted principles have been established in the perioperative use of prophylactic antibiotics.[11]

Principle 1: The Procedure Should Have a Significant Risk for and Incidence of Postoperative Infection

To evaluate the risk for postoperative wound infection, a classification of operative wounds and risk of infection was developed by the American College of Surgeons (Committee on Control of Surgical Wound Infections). All surgical procedures were classified according to four levels of contamination and infection rates (Box 21-1). Within these classifications, it is generally accepted that all class 2, class 3, and class 4 procedures warrant the use of prophylactic antibiotics.[12]

By definition, elective dental implant surgery falls within the class 2 (clean-contaminated) category. Class 2 medical and dental surgical procedures have been shown to have an infection rate of 10% to 15%. However, with proper surgical technique and prophylactic antibiotics, the incidence of infection may be reduced to less than 1%.[13,14] In a healthy patient, risk of infection after dental implant surgery is influenced by numerous factors such as type and location of surgery, skill of the surgeon, methods of intraoperative management, patient factors, and aseptic technique.[13,15] Moreover, additional patient-related (systemic and local) risk factors that are not addressed in these classifications have also been correlated with increased susceptibility to infection. These factors must be addressed in reference to evaluation for the use and duration of antibiotic prophylaxis (Box 21-2).

One of the most significant surgical factors that may contribute to infection is poor aseptic technique. Various routes of transmission of virulent bacteria include (1) direct contact with the patient's blood or other body fluids; (2) indirect contact with contaminated objects; (3) contact of infected nasal, sinus, or oral mucosa; and (4) inhalation of airborne microorganisms. To prevent these conditions, a controlled, well-monitored aseptic setting should be achieved for the surgical procedure. The aseptic surgical site includes proper disinfection and draping procedures of the patient, hand scrubbing, sterile

Box 21-1 Surgical Wound Classifications with Associated Infection Rates

Class 1: Clean (<2%)
- Elective, nontraumatic surgery, no acute inflammation, respiratory, gastrointestinal, and biliary tracts not entered

Class 2: Clean-Contaminated (10% to 15%)
- Elective opening of the respiratory, gastrointestinal, and biliary tracts entered
- Elective dental implant and bone procedures

Class 3: Contaminated (20% to 30%)
- Inflammation, gross spillage from gastrointestinal and biliary tracts along with fresh traumatic injuries

Class 4: Dirty/Infected (50%)
- Established clinical infection, perforation of respiratory, gastrointestinal, and biliary tracts

Box 21-2 Factors Associated with Increased Risk of Infection for Dental Implant Procedures

Systemic Factors

Diabetes
Long-term corticosteroid use
Smoking
Immunocompromised systemic disorders
Malnutrition, obesity
Elderly population
ASA 3 or ASA 4

Local Factors

Use/type of grafting material (autogenous, allograft, alloplast)
Periodontal disease
Tissue inflammation
Odontogenic infections
Ill-fitting provisional prosthesis
Incision line opening
Inadequate hygiene

Surgical Factors

Poor aseptic technique
Skill/experience of the surgeon
Increased duration of surgery
Wound contamination during surgery
Foreign body (implant)

ASA, American Society of Anesthesiologists, physical status classification.

WWW.HIGHDENT.IR

Table 21-1 Probability of Wound Infection by Type of Wound, Risk Index, and ASA Status

OPERATION CLASSIFICATION	RISK INDEX 0	1	2
Clean	1.0%	2.3%	5.4%
Clean-contaminated	2.1%	4.0%	9.5%

Data from Cruse PJ, Foord R: A five-year prospective study of 23,649 surgical wounds, *Arch Surg* 107:206-210, 1973.
ASA, American Society of Anesthesiologists, physical status classification; *0*; ASA 1 or ASA 2, and no local or surgical factors; *1*, ASA ≥2, at least one of the local or surgical factors is present; *2*, ASA ≥2, both local and surgical factors are present.

Box 21-3 Microorganisms Most Commonly Associated with Peri-Implant Complications

- *Staphylococcus* spp.
- *Actinomyces* spp.
- Surface translocating bacteria
- *Wolinella* spp.
- *Capnocytophaga* spp.
- *Fusobacterium* spp.
- *Entamoeba gingivalis*
- Motile rods
- Fusiforms
- Spirochetes
- Enteric gram-negative bacteria
- *Candida albicans*

gowns worn by all surgical members, and maintenance of complete sterility of the instrumentation.

Another important surgical factor related to postoperative infection is the duration of the surgical procedure. This factor has been shown to be the second most critical risk factor (after wound contamination) affecting postoperative infection rates.[18] In general, surgical operations lasting less than 1 hour have an infection rate of 1.3%, whereas those lasting 3 hours increase the rate to more than 4%.[19-21] It is postulated that the rate of infection doubles with every hour of the procedure.[16]

The skill and the experience of the surgeon with the placement of implants have been shown to be significant in postoperative infections and implant failures. A recent study has shown that less experienced surgeons (<50 implants placed) have a 7.3% increase in failure rates in comparison to experienced surgeons.[8]

In the medical literature, it is well documented that the insertion of any prosthetic implant or device increases the chance of infection at the surgical site. A dental implant can act as a foreign body, and the host's defenses may therefore be compromised. The surface of the implant has been shown to facilitate bacterial adherence and the presence of an implant can compromise the host's defenses. This may result in normal flora with low virulence potential to cause infections at the implant-host interface, which has been shown to be very difficult to treat.[22-24]

The probability of risk for infection for a given procedure is related to local, systemic, and surgical factors. The patient's American Society of Anesthesiologists (ASA) score may be used as the systemic factor and then correlated with various local and surgical factors. A risk index may then be modified from the literature to correlate these factors to dental implant surgeries (Table 21-1). The probability of wound infection may then be correlated with the type of wound contamination (class 1 to 4) and the risk index. Therefore a class 2 wound and a risk index 2 has a greater risk of complications, and a class 1 wound and risk index 0 has the least risk of postoperative infection.[19,25]

Principle 2: The Appropriate Antibiotic for the Surgical Procedure Must Be Selected

The prophylactic antibiotic should be effective against the bacteria that are most likely to cause an infection. In the majority of cases, infections after surgery are from organisms that originate from the site of surgery.[11] Most postoperative infections are caused by endogenous bacteria including aerobic gram-positive cocci (streptococci), anaerobic gram-positive cocci (peptococci), and anaerobic gram-negative rods (bacteroides)[13] (Box 21-3).

Although oral infections are mixed infections in which anaerobes outnumber aerobes 2:1, it has been shown that anaerobes need the aerobes to provide an environment to proliferate.[26] Subsequent studies have shown that the early phase of intraoral infections involve streptococci that prepare the environment for subsequent anaerobic invasion.[27,28] With that in mind, the ideal antibiotic must be effective against these pathogens.

The second factor in selecting the correct antibiotic is to use the antibiotic with the least amount of adverse effects. These effects may vary from mild nausea to the extreme allergic reaction.

The final selection factor is that the antibiotic should ideally be bactericidal. The goal of antibiotic prophylaxis is to kill and destroy the bacteria. Bacteriostatic antibiotics work by inhibiting growth and reproduction of bacteria, thus allowing the host defenses to eliminate the resultant bacteria. However, if the host's defenses are compromised in any way, the bacteria and infection may flourish. Bactericidal antibiotics are advantageous over bacteriostatic antibiotics in that (1) there is less reliance on host resistance, (2) the bacteria may be destroyed by the antibiotic alone, (3) results are faster than with bacteriostatic medications, and (4) there is greater flexibility with dosage intervals.[13]

Principle 3: An Appropriate Tissue Concentration of the Antibiotic Must Be Present at the Time of Surgery

For an antibiotic to be effective, a sufficient tissue concentration must be present at the time of bacterial

WWW.HIGHDENT.IR

invasion. To accomplish this goal, the antibiotic should be given in a dose that will reach plasma levels that are three to four times the minimum inhibitory concentration of the expected bacteria.[29] The minimum inhibitory concentration is the lowest antibiotic concentration to destroy the specific bacteria. Usually, to achieve this cellular level, the antibiotic must be given at twice the therapeutic dose and at least 1 hour before surgery.[15] It has been shown that normal therapeutic blood levels are ineffective to counteract bacterial invasion. If antibiotic administration occurs after bacterial contamination, no preventive influence has been seen as compared with taking no preoperative antibiotic.

Principle 4: Use of the Shortest Effective Antibiotic

In a healthy patient, continuing antibiotics after surgery often does not decrease the incidence of surgical wound infections.[2,30,31] In a healthy patient, a single dose of antibiotics is usually sufficient. However, for patients or procedures with increased risk factors (see Box 21-2), a longer dose of antibiotics is warranted.[11] With the high degree of morbidity associated with dental implant infections, one must weigh the benefits versus risk involved for the extended use of antibiotics.

Complications of Antibiotic Prophylaxis

It is estimated that approximately 6% to 7% of patients taking antibiotics will have some type of adverse event.[32] Incidence of significant complications with the use of prophylactic antibiotics are minimal; however, a small percentage can be life-threatening. The risks associated with antibiotics include gastrointestinal (GI) tract complications, colonization of resistant or fungal strains, cross-reactions with other medications, and allergic reactions.

Allergic reactions have a wide range of complications, ranging from mild urticaria to an anaphylaxis and death. Studies have shown that 1% to 3% of the population receiving penicillin will exhibit urticaria type of reactions, with 0.04% to 0.011% having true anaphylactic episodes. Of this small percentage of anaphylactic reactions, 10% will be fatal.[33]

An unusual but increasing complication in the general population after antibiotic use is pseudomembranous colitis. This condition is caused by the intestinal flora being altered and colonized by *Clostridium difficile*. Penicillin and clindamycin use has been significantly associated with pseudomembranous colitis; however, all antibiotics have been shown as potential causative agents. The risk levels of colitis related to antibiotics are outlined in Table 21-2. The most common treatment for antibiotic-induced colitis is vancomycin or metronidazole.

The most recent concern of antibiotic use is the development of resistant bacteria. It has been observed that the overgrowth of resistant bacteria begins only after the host's susceptible bacteria are killed, which usually takes at least 3 days of antibiotic use. Therefore short-term (1 day) use of antibiotics has been shown to have little influence on the growth of resistant bacteria.[13]

Antibiotics Used in Implant Dentistry

Beta-Lactam Antibiotics

The most common beta-lactam antibiotics used in dentistry are the penicillins and cephalosporins. These antibiotics have similar chemical structures, and the mechanism of action is by inhibiting bacterial cell wall synthesis (bacteriocidal) via the interruption of the cross linking between peptidoglycan molecules.

Penicillin V

Penicillin V is one of the more common antibiotics used in dentistry today. It is well absorbed and will achieve peak serum levels within 30 minutes of administration with detectable blood levels for 4 hours. Penicillin V is effective against most *Streptococcus* species and oral anaerobes. The main disadvantages of penicillin are four times per day dosing and susceptibility to resistant bacteria.

Amoxicillin

Amoxicillin is a derivative of ampicillin, with the advantage of superior absorption and a bioavailability of 70% to 80% with a very low toxicity. It has excellent diffusion in infected tissues and adequate tissue concentrations are easily achieved. Amoxicillin is considered broad spectrum and is effective against gram-negative cocci and gram-negative bacilli. This antibiotic also has greater activity than penicillin V against streptococci and oral anaerobes.

Amoxicillin/Clavulanic Acid (Augmentin)

To counteract the activity of beta-lactamase destruction of penicillins by resistant bacteria such as *Streptococcus aureus*, a combination of two antibiotics was synthesized. Clavulanic acid, a beta-lactam antibiotic, was added to amoxicillin to form Augmentin. This combination antibiotic has an affinity for penicillinase-producing bacteria. It functions as a "suicide molecule" that inactivates the resistant bacteria. As a result of an increase in the prevalence of these specific bacteria (especially in the sinus), Augmentin is becoming more popular in oral implantology. This antibiotic is used mainly in cases in which penicillinase bacteria is suspected (or known by culture) and is very practical as a perioperative antibiotic for sinus augmentation (Figure 21-1).

Table 21-2 Risks for Antibiotic-Induced Pseudomembranous Colitis

HIGH	MEDIUM	LOW
Ampicillin	Penicillin	Tetracyclines
Amoxicillin	Erythromycin	Metronidazole
Cephalosporin	Quinolones	Vancomycin
Clindamycin		

WWW.HIGHDENT.IR

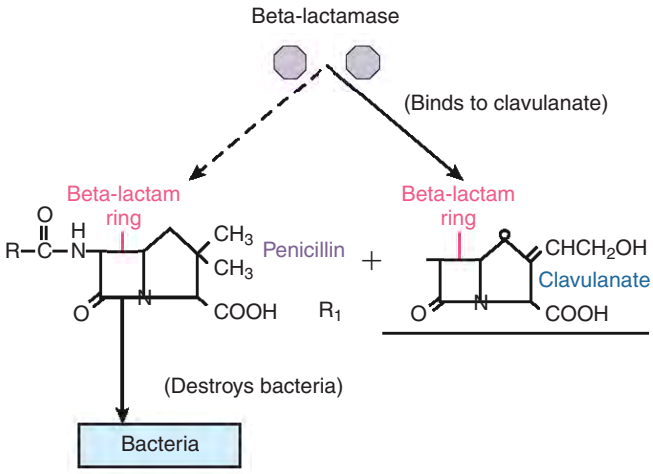


Figure 21-1 Beta-lactamase inactivation by the addition of clavulanic acid to amoxicillin (Augmentin). Because of the high binding affinity of clavulanic acid, beta-lactamase will be inactivated, allowing penicillin to destroy the bacteria.

Cephalexin/Cefadroxil

The first-generation cephalexin/cefadroxil antibiotics have an antibacterial spectrum similar to amoxicillin. However, they have the advantage of not being susceptible to beta-lactamase destruction by *S. aureus*. They are often used in dentistry as an alternative for the penicillin-allergic patient, although cross-reactivity between these two drugs may occur. Rates of cross-reactivity to first-generation cephalosporins with penicillin-allergic patients have been cited to be approximately 8% to 18%. The most recent studies have shown only patients who have had type I (immunoglobulin E: immediate hypersensitivity reactions) should not be administered a cephalosporin. If the patient has a previous history of a reaction that was not immunoglobulin E–mediated (types II, III, IV, or idiopathic reactions) a first-generation cephalosporin may be administered. Newer second- and third-generation cephalosporins exhibit a broader spectrum, less cross-reactivity, and a greater resistance to beta-lactamase destruction.[34]

Macrolides

The most common macrolide used in dentistry is erythromycin. It is active against most streptococci, staphylococci, and some anaerobes, and it is an alternative for patients who are allergic to penicillin. Erythromycin has the advantage of excellent absorption and, unlike many drugs, is affected by the presence of food. It is administered primarily by the oral route and has a relatively low toxicity. However, this antibiotic has a high incidence of nausea and is bacteriostatic rather than bacteriocidal, and it is therefore not an ideal first-line choice for infections in the oral cavity.

These characteristics are also unattractive when either high doses are required, severe infection exists, or the patient is immunocompromised and requires bacteriocidal activity. Even more disturbing is its implication in numerous drug interactions, including its proclivity for elevating serum levels of digoxin, theophylline, and carbamazepine. Erythromycin has also been found to retard conversion of terfenadine (Seldane), a nonsedating antihistamine, to its active metabolite. As a result, elevated serum concentrations of the predrug may result and lead to cardiotoxicity, presenting a particular form of ventricular tachycardia called *torsades de pointes*.

During the past several years, three novel macrolides have been introduced that offer advantages over erythromycin (i.e., clarithromycin [Biaxin], azithromycin [Zithromax]). Unlike other macrolides, they do not appear to inhibit hepatic cytochrome P450 isozymes, which account for most drug interactions of erythromycin. Biaxin produces less nausea and has better gram activity; Zithromax appears to be more effective against *Haemophilus influenzae*.

Clindamycin

The use of clindamycin has increased for the treatment of dental infections primarily because of its activity against anaerobic bacteria. It also is active against aerobic bacteria, such as streptococci and staphylococci, and it has superior effects against *Bacteroides fragilis*. Clindamycin (Cleocin phosphate) is also supplied in an aqueous 300-mg/2-mL solution that is sometimes used in the incorporation of graft material for sinus augmentation procedures. However, it is bacteriostatic in normal concentrations and has a rather high toxicity in larger concentrations. As a result, the main disadvantage of clindamycin is the occurrence of diarrhea in 20% to 30% of patients treated. This antibiotic also has a higher incidence of antibiotic-associated pseudomembranous colitis (PMC) caused by *C. difficile* when administrated for extended periods. PMC has been reported to occur with most long-term antibiotics.

The toxicity of antibiotics related to PMC is elevated with ampicillin, amoxicillin, cephalosporin, and clindamycin. Penicillin, erythromycin, and quinolones are moderate risk, and the lowest occurrence is with tetracycline, metronidazole, and vancomycin. The latter group is often used to even treat PMC conditions.

The patient should be informed that if either diarrhea or abdominal cramping occurs during or shortly after antibiotic therapy, the drug should be discontinued and the doctor should be notified.

Antidiarrheal medications should be avoided in these cases, because they hinder the fecal elimination of the pathogen. If it is necessary to continue management of the dental infection, imidazole or vancomycin is most logical (if not the original cause of the complications). Metronidazole is not only effective against anaerobes contributing to the dental infection, but is effective against *C. difficile*, the causative agent. If the condition persists after 3 days, the patient should be assessed by an internist for fluid and electrolyte imbalance.

WWW.HIGHDENT.IR

Tetracyclines

Tetracyclines have been available since the 1950s and have a wide spectrum of activity against streptococci, staphylococci, oral anaerobes, and gram-negative aerobic rods. Because this antibiotic has been so extensively used in the past, there exists a high degree of bacterial resistance. Tetracycline is an attractive adjunct for the treatment of gingival and periodontal disease with a high bioavailability in the gingival sulcus. For these reasons, tetracyclines are primary agents for treating implant disease and infections around implant posts. Their efficacy for managing infrabony infections is questionable, considering their inactivity when chelated with calcium complexes. The disadvantages of this antibiotic include a high incidence of promoting *Candida* spp. infections and may be associated with photosensitivity reactions.

Fluoroquinolones

A recent classification of antibiotics has had a definite impact on the treatment of infections in dentistry and medicine. Flouroquinolones are bactericidal antibiotics and have a broad antibacterial spectrum, which may be used either orally or parenterally. Ciprofloxacin was one of the first-generation quinolones and is the prototype antibiotic for this antibiotic classification. Newer third- and fourth-generation quinolones have been developed with great activity against resistant bacteria and anaerobic bacteria. In implant dentistry, fluoroquinolones are used mainly in the prophylactic and therapeutic treatment of sinus augmentation procedures.

Metronidazole

Metronidazole is a bacteriocidal antibiotic that is most often used for anaerobic infections. Because metronidazole has no activity against aerobic bacteria, it is seldom used for mixed infections unless it is combined with another antibiotic. However, it may be combined with penicillin when managing severe infections. Patients should be cautioned against drinking alcoholic beverages while taking this medication, because disulfiram-like reactions have been reported. These consist of severe nausea and abdominal cramping caused by the formation of a toxic compound resembling formaldehyde. Metronidazole should not be prescribed for patients taking the oral anticoagulant warfarin (Coumadin). The more common antibiotics and dosages used in oral implantology for prophylaxis, grafting and implant insertion, postoperative infection, and long-term complications are listed in Table 21-3.

Prophylactic Antibiotics in Oral Implantology

Postoperative wound infections can have a significant effect on the success of dental implants and bone grafting procedures. The occurrence of surgical host defenses allows an environment conducive to bacterial growth. This process is complex, with interactions of host, local tissues, and systemic and microbial virulence factors. Various measures attempt to minimize infection by modifying the host and local tissue factors. The use of antimicrobials has been shown to be significant in reducing postoperative infections.

The antibiotic chosen for prophylaxis should encompass the bacteria most known to be responsible for the type of infection found with the surgical procedure. Therefore the following antibiotics are suggested against pathogens known to cause postoperative surgical wound infections in bone grafting or implant surgery:

1. Amoxicillin: the usual drug of choice

If allergic,

2. Cephalexin (nonanaphylactic allergy to penicillin)
3. Clindamycin (anaphylactic allergy to penicillin)

For sinus involvement procedures (e.g., sinus grafts):

1. Augmentin
2. Levaquin (if history of recent use of antibiotics [within 4 weeks])

THERAPEUTIC USE OF ANTIBIOTICS: POSTOPERATIVE INFECTIONS

Acute postoperative infections have been shown to occur on the third to fourth day after surgery. The most common microorganisms associated with peri-implant, postoperative complications have been previously stated as listed in Box 21-3.

Local signs of infection are pain, inflammation, bleeding, and exudate at the site of surgery. Systemic signs include fever, headache, nausea, muscle aches, vomiting, and weakness. When surgical wound infections arise, a specific diagnosis is advantageous to treat the complication. When evaluating the various antibiotics possible that are effective against the bacteria in question, a broad-spectrum beta-lactam antibiotic is most often the first-line medication. The duration of treatment should include antibiotic administration for 3 days beyond the occurrence of significant clinical improvement, usually at the fourth day and therefore for a minimum of 7 days.[35]

Therapeutic Antibiotics in Implant Dentistry

The recommended treatment for intraoral infections associated with grafting or implant therapy include the following:

1. Surgical drainage

and

2. Systemic antibiotics

WWW.HIGHDENT.IR
انجمن دندانسازان و دندانپزشکان

Table 21-3 Commonly Used Antibiotics in Oral Implantology

GENERIC NAME	BRAND NAME	BACTERICIDAL/ BACTERIOSTATIC	THERAPEUTIC USUAL ADULT DOSE	MAXIMUM ADULT DOSE	PROPHYLACTIC DOSES
Amoxicillin	Amoxil Polymox Trimox	Bactericidal	250-500 mg TID	4 g/day`	SBE: 2 g 1 hr before Surgical: 1 g 1 hr before
Amoxicillin/ clavulanic acid	Augmentin	Bactericidal	250-500 mg TID or 825 mg BID	4 g/day	Surgical: 825 mg
Cephalexin	Biocef Cefanex Keftab Keflex	Bactericidal	250 mg QID or 500 mg BID	4 g/day	SBE: 2 g 1 hr before Surgical: 1 g 1 hr before
Cefadroxil	Duricef Ultracef	Bactericidal	500 mg BID	4 g/day	SBE: 2 g 1 hr before Surgical: 1 g 1 hr before
Azithromycin	Zithromax	Bacteriostatic	500 mg immediately, 1000 mg/day	—	SBE: 500 mg 1 hr before
Clarithromycin	Biaxin	Bacteriostatic	250 mg	—	SBE: 500 mg 1 hr before
Erythromycin	E-mycin E-tab	Bacteriostatic	250 mg QID	4 g/day	—
Tetracycline	Achromycin Sumycin	Bacteriostatic	250 mg QID	4 g/day	—
Clindamycin hydrochloride	Cleocin HCl	Bacteriostatic	150-300 mg TID or QID	1.8 mg/day	SBE: 600 mg 1 hr before Surgical: 600 mg 1 hr before
Metronidazole	Flagyl	Bactericidal	250 mg TID or QID	4 g/day	
Levofloxacin	Levaquin	Bactericidal	500 mg/day	500 mg/day	Surgical: 500 mg
Moxifloxacin	Avelox	Bactericidal	400 mg/day	400 mg/day	—
Trimethoprim/ sulfametho-xazole	Bactrim Septra	Bacteriostatic	160 mg (DS) BID 80 mg BID	—	—

SBE, Subacute bacterial endocarditis; *DS,* double strength.

Amoxicillin (500 mg): Two immediately, then one tablet three times daily for 1 week

or if penicillin allergy exists

Clindamycin (300 mg): Two immediately, then one tablet three times daily for 1 week

NOTE: If no improvement is seen after 4 days, a culture and sensitivity test should be administered to select the antibiotic most effective against the responsible organisms.

Until culture and sensitivity test results are obtained, change antibiotic to:

Levaquin (50 mg): one tablet daily for 1 week

and

3. 0.12% chlorhexidine gluconate rinse (1/2 oz twice daily for 2 weeks)

Chlorhexidine

Another modality for antimicrobial prophylaxis for implant surgery is the use of an oral rinse, 0.12% chlorhexidine digluconate (Peridex; Procter & Gamble, Cincinnati, Ohio). Chlorhexidine gluconate is a potent antibacterial that causes lysis by binding to bacterial cell membranes. It has high substantivity that allows it, at high concentrations, to exhibit bacteriocidal qualities by causing bacterial cytoplasm precipitation and cell

WWW.HIGHDENT.IR

death.[36,37] In the oral cavity, chlorhexidine has been shown to have a slow release from tissue surfaces over a 12-hour period.[38,39]

In vitro studies have shown an inhibitory effect of chlorhexidine on cultured epithelium and cell growth; however, clinical studies have not shown this effect.[40,41,44] To the contrary, the use of chlorhexidine has been shown to be an effective adjuvant in reducing plaque accumulation, enhance mucosal health,[42-44] improve soft tissue healing,[45,46] treat periodontal disease, prevent alveolar osteitis,[47,48] improve tissue healing after extractions,[49] reverse peri-implantitis,[50] and been shown to have no adverse affect on implant surfaces.[51]

When evaluating the effect of preoperative chlorhexidine before dental implant surgery, a significant reduction in the number of infectious complications (2 to 1), and a sixfold difference in implant failures in comparison to no use of chlorhexidine has been shown.[52]

Use of Chlorhexidine in Oral Implantology

As a consequence of many reported benefits of chlorhexidine, the use of this antiseptic is suggested in many ways in oral implantology as follows:

1. Patient presurgical rinse: used in the aseptic protocol before surgery for reduction of bacterial load
2. Surface antiseptic: intra- and extraoral scrub of patient, scrubbing of hands before gowns and gloves
3. Postsurgical rinse: rinse twice a day until incision line closure
4. Peri-implant maintenance on daily basis
5. Treatment of postoperative infections

MANAGEMENT OF POSTOPERATIVE INFLAMMATION

The management of postsurgical swelling is crucial to the pain management, control of edema, and incidence of postoperative infection. In most dental implant surgeries, tissue is traumatized, which results in some degree of an inflammatory reaction. By controlling the extent of inflammation associated with surgical procedures, edema, trismus, pain, and infection may be reduced.

The mediators of the inflammatory process include cyclooxygenase and prostaglandins, which play a significant role in the development of postoperative inflammation and pain (Figure 21-2). When tissue manipulation or damage occurs, phospholipids are converted into arachidonic acid by way of phospholipase A_2. Arachidonic acid, which is an amino acid, is released into the tissue, which produces prostaglandins by enzymatic breakdown by cyclooxygenases. The end result is the formation of leukotrienes, prostacyclins, prostaglandins, and thromboxane A_2, which are the mediators for inflammation and pain. For postoperative treatment, medications such as ibuprofen (nonsteroidal antiinflammatory drugs [NSAIDs]) and glucocorticosteroids (steroids) are used, which play an integral part in counteracting the negative effects of this cascade.

Nonsteroidal Antiinflammatory Drugs

Nonsteroidal antiinflammatory drugs have an analgesic effect as well as an antiinflammatory effect. This drug class reduces inflammation by inhibiting the synthesis of prostaglandins from arachidonic acid. Therefore the use of the popular analgesic drug ibuprofen has a secondary beneficial antiinflammatory effect. NSAIDs do not have a ceiling effect for inflammation; however, higher doses to achieve antiinflammatory qualities are accompanied by serious side effects. In implant dentistry, the use of ibuprofen is suggested as a preemptive analgesic agent, because it has antiinflammatory properties in type 1 to 4 procedures.

Glucocorticosteroids

The adrenal cortex, which uses cholesterol as a substrate, synthesizes and secretes two types of steroid hormones—the androgens and corticosteroids. The corticosteroids are classified additionally by there major actions: (1) glucocorticoids, which have effects on carbohydrate metabolism and have potent antiinflammatory actions, and (2) mineralocorticoids, which have sodium-retaining qualities. The use of synthetic glucocorticosteroids has become very popular in the postoperative management of inflammation after oral surgical procedures. These synthetic glucocorticoids have greater antiinflammatory potency in comparison to natural steroids with very little sodium and water retention. Most of these steroids have similar chemical structures; however, they differ in their milligram potency.[53] Their antiinflammatory effects are achieved by altering the connective tissue response to injury, thus causing a decrease in hyperemia, which results in less exudation and cellular migration along with infiltration at the site of injury.[54,55]

There is a wide range of glucocorticoid preparations available for local, oral, and parenteral administration.

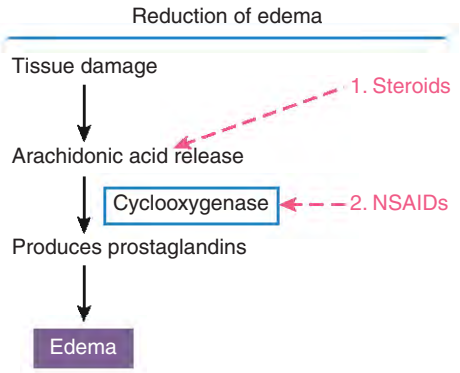


Figure 21-2 Mechanism of action for nonsteroidal antiinflammatory drugs (NSAIDs) and steroids in the reduction of inflammation.

WWW.HIGHDENT.IR
انجمن دندانسازان و دندانپزشکان

Table 21-4 Synthetic Glucocorticoids

GLUCOCORTICOIDS	ANTIINFLAMMATORY POTENCY	EQUIVALENT DOSE (mg)	DURATION (hr)
Short-acting			
Hydrocortisone	1.0	20	<12
Cortisone	0.8	25	<12
Intermediate-acting			
Prednisone	4.0	5	24 to 36
Prednisolone	4.0	5	24 to 36
Long-acting			
Dexamethasone	25	0.75	>48

In relation to the naturally occurring cortisol (hydrocortisone), synthetic glucocorticoids are longer acting and more potent. The main differences are based on the classification as short-acting (<12 hours), intermediate-acting (12 to 36 hours), and long-acting (>36 hours). A summary of the most common glucocorticosteroids is shown in Table 21-4.[53]

Mechanism of Action

Glucocorticoids bind to glucocorticoid receptors within cells and form a glucocorticoid-GR complex. This complex alters the synthesis of mRNA from the DNA molecule, thus affecting the production of different proteins. By suppressing the production of proteins that are involved in inflammation, glucocorticoids also activate lipocortins, which have been shown to inhibit the action of phospholipase A2. Phospholipase A2 is a key enzyme involved in the release of arachidonic acid from cell membranes.

Arachidonic acid is an omega-6 fatty acid that is incorporated into cell membranes. When a cell is damaged, arachidonic acid is released from cell membranes and is converted into inflammatory and pain prostaglandins by cyclooxygenase (COX)-2 enzymes. The release of arachidonic acid requires the activation of enzyme PLA2. However, lipocortins, which cause the inhibition of PLA2, prevent the release of arachidonic acid, thereby reducing the amounts of inflammatory prostaglandins.

Adrenal Suppression

Glucocorticoids are essential for the body to adapt to stressful situations. Adrenal insufficiency may predispose a person to an inability to respond to stress. Adrenal suppression has been shown to occur after 7 to 10 days of steroid administration. In stressful situations, cardiovascular collapse may occur and, if not treated appropriately, may be life-threatening. Because most dental implant procedures maintain a high level of stress, the implant dentist must be able to assess the level of adrenal suppression on patients taking glucocorticoid replacement therapy.

Prolonged, long-term steroid therapy causing adrenal suppression is a well-known phenomenon. The amount of suppression is a function of both the duration of treatment and the dose administered. Studies have shown that short-term use of corticosteroids does not significantly affect the hypothalamus-pituitary adrenal (HPA) axis, and normal levels of cortisol, which are initially suppressed, recover to normal levels after 7 days.[57] The conclusion is that the HPA axis, although altered by the initial dexamethasone therapy, is restored completely. Additionally, the amount of surgical stress involved with oral surgical procedures appears to be of insufficient magnitude to overcome the HPA suppression of the negative feedback mechanism caused by the steroid administration. Therapeutic levels of steroid are present at a cellular level to prevent any manifestations of adrenal insufficiency.[58]

Timing

The use of synthetic steroids should be based on the production of the natural steroid cortisol (hydrocortisone) in the body. Normally, cortisol is produced from plasma cholesterol at a rate of 15 to 30 mg per day.[59] Under stressful situations (e.g., infection, illness, trauma), as much as 300 mg of cortisol can be secreted. Plasma concentrations of cortisol are several-fold higher in the morning as compared with the afternoon. Studies have shown that a dose of dexamethasone given in the morning (8:00 AM) does not significantly alter the level of endogenous circulating cortisol. However, the same dose in the late afternoon (4:00 PM) can cause complete suppression of the HPA cycle.[60] This secretion rate is dictated by the pituitary-adrenal axis with a feedback-inhibition cycle.[61] Therefore administration of glucocorticoids should ideally be given in the morning so that simulation of normal diurnal rhythm is achieved, thus minimizing the possibility of HPA suppression.[62]

Glucocorticoids in Medicine and Dentistry

Since the advent of glucocorticoids in 1942, these medications have been used clinically in two ways: (1) therapeutic treatments in various inflammatory diseases

WWW.HIGHDENT.IR

and autoimmune diseases and (2) prophylactic treatment of inflammation and associated pain. Today, they are still used for an array of autoimmune diseases. Glucocorticoids have been well documented in the dental literature as being advantageous in the prevention of postoperative complications after traumatic oral surgery,[63] intraoral sagittal osteotomy,[64] vestibuloplasty with palatal mucosal grafts, and reduction of edema and pain after oral surgical procedures.[54-56,65] Additionally, they have been shown to be associated with less need of pain medication after oral surgical procedures.[66,67] These drugs have been shown to have the ability to be long-lasting in duration and cause minimal effects on wound healing, infection, and adrenal suppression, with minimal central nervous system alteration.[61]

Glucocorticoids in Implant Dentistry

The use of glucocorticoids is an integral part in the treatment of postsurgical edema after dental implant procedures. The selection of the ideal synthetic glucocorticoid for dental implant surgery should maintain high antiinflammatory potency with minimal mineralocorticoid effects. The glucocorticoid that best suits the requirements is the long-acting glucocorticoid dexamethasone (Decadron). It is imperative that this drug be administered before surgery so that adequate blood levels are obtained. Also, it should be given in the morning in conjunction with the natural release of cortisol. This timing will interfere the least with the adrenocortical system. Because inflammation usually peaks between 48 and 72 hours, the postoperative regimen of dexamethasone should not exceed 3 days after surgery. The dose should not exceed the equivalence of 300 mg of cortisol with a decreasing dose the second and third day to reduce possible side effects. This high-dose, short-term glucocorticoid therapy has been shown not to significantly effect the HPA axis.[58,68,69]

A significant additional benefit of the administration of dexamethasone is the potent antiemetic effects for the prophylactic treatment of postoperative nausea and vomiting. This is now an accepted medication for hospital-based outpatient surgery usually given in doses of 8 to 10 mg intravenously.[70,71,106]

Contraindications to the use of corticosteroids include active infections (viral, bacterial, fungal), tuberculosis, ocular herpes simplex, primary glaucoma, acute psychosis, and diabetes mellitus. Special attention must be given to diabetic patients, as glucocorticoids have an anti-insulin action that results in increased serum glucose and glycosuria.[72]

Cryotherapy

An additional therapeutic regimen to help reduce the amount and duration of postoperative inflammation is the application of cold dressings. It is reported that cold dressings in the form of ice bags or premanufactured ice packs applied extraorally to the surgical site will minimize edema.[73] The application of cold dressings is believed to cause vasoconstriction of the capillary vessels, thus reducing the flow of blood and lymph in this region, resulting in less inflammation.[74] Also, with the lower temperature at the surgical site, cell metabolism is reduced. As a result, the cells in the trauma region consume less oxygen, which allows them to survive a longer time period of ischemia.

Cryotherapy as an Antiinflammatory Agent

The use of cryotherapy is highly advised in any implant procedure in which excessive inflammation is expected. Cold dressings (ice packs) should be applied extraorally over the surgical site for 20 minutes on/20 minutes off for the first 24 to 36 hours. Caution must be taken to limit the application of ice for no longer than 2 days, because prolonged use may cause rebound swelling and cell destruction.

POSTSURGICAL PAIN MANAGEMENT

Pain has been documented to be inadequately treated in 50% of all surgical procedures.[75] These painful experiences predispose the patient to amplification of noxious stimuli (hyperalgesia) and cause typically painless sensations to be experienced as pain (allodynia).[76,77] Therefore patients who have had painful experiences may have increased pain and the need for additional analgesic use in future surgeries. The goal for pain control in oral implantology is to have adequate analgesic levels before the cessation of local anesthesia and a well-administrated postoperative analgesic regimen for patient comfort.

Preemptive Analgesia

Preemptive analgesia is defined as the introduction of an analgesic regimen before the onset of noxious stimuli. In relation to dental implant surgery, it is advantageous to have adequate analgesic blood levels present before the initiation of surgery. The goal is to prevent sensitization of the nervous system to subsequent stimuli that could possibly amplify pain. Dental implant surgery is ideal for this type of treatment because it is usually elective and the timing of noxious stimuli is known.

Manipulation of hard and soft tissues during implant and bone grafting procedures predispose the patient to postoperative pain. The extent of tissue reflection, amount of bone preparation, inherent patient factors, and duration of the surgical procedure have an effect on the intensity and duration of postoperative pain. Hyperalgesia is characterized by enhanced sensations of pain, a pain threshold reduction, and an increase in the suprathreshold noxious stimuli. With administration of analgesics before tissue damage, the sensitivity of these receptors is dramatically reduced and may be eliminated.[80]

WWW.HIGHDENT.IR
انجمن دندانسازان و دندانپزشکان

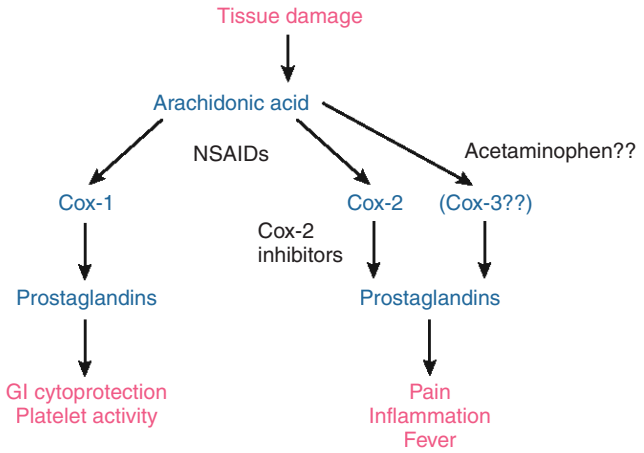


Figure 21-3 Mechanism of action of the various cyclooxygenase enzymes. *NSAIDs,* Nonsteroidal antiinflammatory drugs; *GI,* gastrointestinal.

Box 21-4 Analgesic Classifications in Dentistry

Nonopioids

1. Nonsteroidal antiinflammatory drugs
2. Acetaminophen
3. Tramadol
4. Cyclooxygenase-2 inhibitors

Opioids

1. Codeine
2. Hydrocodone
3. Oxycodone
4. Meperidine

Adjuvants

1. Glucocorticoids
2. Long-acting anesthetics
3. Tricyclic antidepressants

Mechanism of Pain

The mechanism of painful stimuli is modulated by the peripheral and central nervous systems. Noxious stimuli (tissue damage) cause peripheral nociceptors to transmit signals along nerve fibers lying in the dorsal root ganglion. Their axons synapse in the dorsal horn of the spinal cord and then travel along the spinothalamic tract of the spinal cord to the thalamus and the cortex. Within the cortex and thalamus, the signals originating from tissue damage form the subjective interpretation of pain.

With repeated noxious stimuli, peripheral nociceptors become more responsive. The sensitivity to these receptors are further enhanced by tissue factors and inflammatory mediators released in the course of tissue damage. Numerous inflammatory mediators are present which include prostaglandins, kinins, leukotrienes, substance P, and histamine. These mediators initiate and magnify the nociceptive impulses that are transmitted to the central nervous system for the perception of pain.

The most important mediators, prostaglandins, are extremely important in sensitizing peripheral neurons to the local stimuli. Prostaglandins are also synthesized in the spinal cord and brain and enhance pain sensitivity by recruiting secondary neurons to respond to the primary stimulus.[81]

One of most commonly used analgesics, NSAIDs, work at the site of tissue damage and the spinal cord and brain to prevent prostaglandin formation by inhibiting cyclooxygenase (COX). COX is an enzyme that breaks down arachidonic acid to prostaglandin synthesis. In the tissue, there exist two well-identified cyclooxygenases, COX-1 and COX-2. COX-1 enzymes support hemostasis (platelet degranulation and adhesion), stomach mucosal integrity, and regulation of kidney function. COX-2 enzymes are an inducible form whose synthesis is activated in damaged tissue, which leads to the formation of proinflammatory prostaglandins that play a major role in inflammation, pain, and fever. A relatively new COX has been described (COX-3) that is found in the brain and is thought to be the site of action of acetaminophen.[82]

In contrast to NSAIDs, opioids have a different mechanism of action to reduce pain. Opioids act on the central nervous system by binding to specific receptors (μ opioid), thus preventing transmission of nociceptive pathways, while also activating inhibitory pathways that descend to the spinal cord. By binding to these μ-opioid receptors, substance P is prevented from being released, thus preventing painful stimuli.[83]

MANAGEMENT OF POSTOPERATIVE PAIN

In implant dentistry, different classifications and mechanisms of pain suppression may be used. A pain control protocol has been established, which simplifies and standardizes the various aspects of pain relief (Figure 21-3; Box 21-4; Table 21-5).

1. Nonopioid analgesics (nonnarcotics)
2. Opioid analgesics (narcotics)
3. Adjuvants

Nonopioid Medications

The nonopioid analgesics used in implant dentistry include acetaminophen, NSAIDs, COX-2 inhibitors, and tramadol.

Acetaminophen

The mode of action of acetaminophen is not known; however, it is believed to involve the prostaglandin pathways within the central nervous system with little influence on peripheral prostaglandin synthesis. COX-3 enzyme has been described that is fully expressed in the brain, spinal cord, and heart. The primary function

WWW.HIGHDENT.IR

Table 21-5 Analgesic Agents Used to Control Postoperative Surgical Pain

ANALGESIC	BRAND NAME	ONSET (hr)	PEAK (hr)	DURATION (hr)	RECOMMENDED DOSE	DOSING INTERVAL (hr)	MAXIMUM DOSE/DAY
Nonopioid							
Acetaminophen	Tylenol	0.5	0.5-2	4-6	650-1000 mg	4-6	4000 mg
Ibuprofen	Motrin Advil	0.5	1-2	4-6	400 mg	4-6	2400 mg
Naproxen	Anaprox	1	2-4	5-7	275-550 mg	6-8	1375 mg
Tramadol	Ultram	0.5	1-2	4-6	50-100 mg	4-6	400 mg
Opioid							
Codeine	Tylenol with codeine	0.1-0.3	0.5-1	4-6	60 mg	3-4	
Hydrocodone	Dicodid	0.25-0.5	0.5	4-8	5-10 mg	4-6	
Oxycodone	Percocet	0.25-0.5	1	4-6	5-10 mg	4-6	
Meperidine	Demerol	0.1-0.45	0.5-1	2-4	50-100 mg	6	
Propoxyphene	Darvon	0.5-1	2-2.5	4-6	65-130 mg	4-6	

is to regulate pain responses and fever and has been postulated to be the site of action of acetaminophen.[84]

Acetaminophen is indicated for mild to moderate pain and a safe alternative to NSAIDs. It has excellent analgesic and antipyretic properties and is void of side effects that are associated with NSAIDs. Like NSAIDs, acetaminophen also has a ceiling dose (4 g/day) for analgesic effects. However, unlike NSAIDs, acetaminophen is limited in that it has minimal anti-inflammatory qualities. The main side effect is liver damage, which is associated with long-term use of this drug.

Nonsteriodal Antiinflammatory Drugs

The NSAIDs are one of the most commonly used analgesics in implant dentistry today. Clinical trials have shown that NSAIDs are effective in all levels of pain (mild, moderate, severe).[85,86] The mechanism of action of NSAIDs is thought to arise from the inhibition of the synthesis of prostaglandins from arachidonic acid. With the inhibition of COX, conversion of arachidonic acid to the immediate precursors of prostaglandins is prevented. Thus with the lack of prostaglandins in the tissue, the hyperanalgesia and edema associated with the acute inflammation is minimized.[87]

The main reasons that NSAIDs are so widely used is the fact that they work very well as analgesics and have variable effects on inflammation (drug and dose dependent). Inflammation and pain are two separate entities with analgesic doses having a ceiling effect[88] and antiinflammatory doses not having a ceiling effect. In regards to the analgesic effect, there exists no reason to exceed the analgesic ceiling for the treatment of acute pain, as higher doses give no additional pain relief while increasing the likelihood of side effects.

The side effects of NSAIDs are numerous, including GI disturbances (dyspepsia, erosions, ulcerations) and liver, renal, and cardiac effects.[89] This group of medications is responsible for the largest number of serious drug-related complications, surpassing all other drugs by a wide margin.[90] In 2005, GI-related deaths from NSAIDs were the fourteenth leading cause of death in the United States, ranked after homicides (thirteenth) and before atherosclerosis (fifteenth).[91] The various types of NSAIDs and their associated risks are shown in Table 21-6.[103]

Table 21-6 Relative Risks of NSAIDs for Gastrointestinal Complications

NSAID	RELATIVE RISK
None	1
Ibuprofen	2.1
Ketoprofen	3.2
Naproxen	4.3
Indomethacin	5.5
Aspirin	8 to 11
Ketorolac	24.7

NSAID, Nonsteroidal antiinflammatory drug.

NSAIDs have very little effect on platelet aggregation because bleeding times are not prolonged. With prolonged use of NSAIDs, interference with most classes of antihypertensives has been noted. Therefore if patients take NSAIDs for more than 5 days postoperatively, blood pressure should be monitored.

Ibuprofen

Ibuprofen was first introduced in 1969 as a new NSAID and has since been the most popular prescribed NSAID.[93] Ibuprofen is used to treat mild to moderate pain and has been proven to significantly reduce postoperative

WWW.HIGHDENT.IR

dental pain in clinical studies.[94,95] The analgesic ceiling dose is 400 mg/dose and 1200 mg/day[96]; at these doses it has been shown to be as safe as acetaminophen, while achieving better analgesia with less nausea and cramping.[98]

Aspirin

Acetylsalicylic acid (ASA) was the first prototypical NSAID. It has analgesic, antiinflammatory, and antipyretic properties. However, at analgesic doses its relative risk for GI complications is high. Acetylsalicylic acid is not a drug of choice in the management of dental implant surgical patients because of its very significant antiplatelet effects.

Tramadol

Tramadol represents a unique classification of analgesic because it is a centrally acting analgesic with two complementary characteristics: opioid and antidepressant. It works by inhibition of norepinephrine and serotonin reuptake within pain pathways of the central nervous system and also by its relative weak affinity for the μ-opioid receptor.[98] Tramadol is a nonscheduled drug and is associated with fewer opioid-like side effects, such as dependence, sedation, respiratory depression, and constipation.[99,100] Tramadol's analgesic efficacy is similar to codeine (60 mg) and is indicated for moderate to moderately severe pain management. This drug is an appropriate analgesic alternative for the treatment of postoperative pain in patients who have NSAID-related GI and opioid intolerance. Tramadol has been shown to be effective in the reduction of pain when used in combination with acetaminophen. Ultracet (tramadol/acetaminophen) has demonstrated excellent efficacy in pain studies and is supplied as a combination analgesic containing 37.5 mg tramadol and 325 mg acetaminophen.[101,102]

COX-2 Inhibitors

The latest classification of analgesic drugs to fight pain and inflammation is the COX-2 inhibitors (i.e., celecoxib, rofecoxib, valdecoxib). The main advantage of this classification of medications is the lack of GI side effects. Recently, several of these drugs have been recalled by the U.S. Food and Drug Administration because of serious cardiovascular complications. Additionally, this classification of medication has not been shown to achieve better pain control or antiinflammatory properties than ibuprofen.

Narcotics (Opioids)

Narcotics (opioids) are the primary medications for analgesia of moderate to severe pain from dental origin. They are centrally acting analgesics that act as agonists at μ and κ opioid receptors. Morphine, which is a naturally occurring opioid, is generally accepted as the prototypical narcotic. All other narcotics are compared in potency to morphine.

Unlike nonopioids, opioids do not have a ceiling effect for analgesia. As the dose increases, the analgesic effect increases. However, in addition to relieving pain by μ-receptor binding, euphoria, nausea, vomiting, and constipation may occur. With high doses, sedation and respiratory depression are possible. With chronic use, physical and psychological dependence are common.

The following section discusses the most commonly used narcotics in oral implantology. Structurally, these narcotics are similar to morphine and provide the same degree of pain relief and unlimited efficacy at equipotent doses.

Codeine

Codeine is a naturally occurring alkaloid that is classified as a mild analgesic. Codeine has excellent antitussive properties; however, it is associated with high degrees of nausea and constipation. Orally administered codeine is only 60% bioavailable, which results in only 10% being demethylated to morphine. This 10% is the only part responsible for analgesic properties, thus allowing 90% to have no analgesic efficacy. Because of the side effects and low potency compared with other opioids, codeine is usually not the first choice of narcotics used in oral implantology.

Hydrocodone

Hydrocodone bitartrate is a semisynthetic narcotic analgesic and antitussive with multiple actions qualitatively similar to codeine. It is usually used as a combination analgesic, being combined with either acetaminophen or ibuprofen. For several years, this narcotic has been the most frequently dispensed prescription medication in the United States. Hydrocodone is habit forming, and the most frequent adverse reactions are dizziness, sedation, nausea, and vomiting.

Oxycodone

Oxycodone is a semisynthetic opioid with analgesic action similar to morphine. It is recommended for moderate to severe pain with its principal actions being analgesia and sedation. It has excellent oral bioavailability because it retains half of its analgesic activity when administered orally. Oxycodone has the same adverse effects as most other opioids, with an increased potential for abuse and drug dependence. Oxycodone is marketed as a combination narcotic, combined with either acetaminophen (Percocet) or aspirin (Percodan). A slow-release oxycodone (Oxycontin) has recently been released, which has a high abuse potential.

Meperidine

Meperidine is mostly used in hospital settings via intramuscular administration. A majority of meperidine

WWW.HIGHDENT.IR

Table 21-7 Combination Analgesics

GENERIC NAME	BRAND NAME	AVERAGE ADULT DOSE	SCHEDULE
5 mg codeine/300 mg acetaminophen	Tylenol # 1	1-2 tablets every 4 hr	III
15 mg codeine/300 mg acetaminophen	Tylenol # 2	1-2 tablets every 4 hr	III
30 mg codeine/300 mg acetaminophen	Tylenol # 3	1-2 tablets every 4 hr	III
60 mg codeine/300 mg acetaminophen	Tylenol # 4	1 tablet every 4 hr	III
5 mg hydrocodone/500 mg acetaminophen	Vicodin/Lortab 5/500	1-2 tablets every 4-6 hr (maximum 8 tablets/24 hr)	III
7.5 mg hydrocodone/750 mg acetaminophen	Vicodin ES	1 tablet every 4-6 hr	III
7.5 mg hydrocodone/650 mg acetaminophen	Lorcet	1 tablet every 4-6 hr	III
10 mg hydrocodone/660 mg acetaminophen	Vicodin	1 tablet every 4-6 hr	III
10 mg hydrocodone/650 mg acetaminophen	Lorcet 10/650	1 tablet every 4-6 hr	III
7.5 mg hydrocodone/200 mg ibuprofen	Vicoprofen	1-2 tablets every 6 hr	III
5 mg oxycodone/325 mg acetaminophen	Percocet 5/325	1-2 tablets every 4-6 hr	II
7.5 mg oxycodone/500 mg acetaminophen	Percocet 7.5/500	1-2 tablets every 4-6 hr/ maximum 8/day	II
10 mg oxycodone/650 mg acetaminophen	Percocet 10/650	1 tablet 4-6 hr	II
5 mg oxycodone/400 mg ibuprofen	Combunox	1 tablet every 6 hr/maximum 4/day	II

is converted to normeperidine, which is a metabolite that has no analgesic property; however, is a very strong central nervous system (CNS) stimulant. Because meperidine in oral form has a poor oral bioavailability (25%), a greater risk evolves with the accumulation of normeperidine. As a result, meperidine is a poor choice for an orally administered opioid.

COMBINATION ANALGESIC THERAPY FOR POSTOPERATIVE PAIN

A pain management strategy using multiple analgesics with different mechanisms of action is termed *combination analgesic therapy*. The goal of combining different types of analgesics is to increase the analgesic effect while decreasing possible side effects. When multiple drugs are used in combination, synergistic and additive effects allow for the use of lower doses of each individual drug.

With combination therapy, acetaminophen or NSAIDs are used with an opioid. Because of the ceiling effects of acetaminophen and NSAIDs, further increases in dosage will not provide any additional analgesia; however, they will increase side effects (Table 21-7).

Analgesic Agents in Oral Implantology

The selection of an analgesic or analgesic regimen for management of postsurgical pain is ideally based on the expected pain intensity. This may be based on the patient's medical history, past pain threshold, type of procedure, extent of tissue reflection, and duration of procedure. Because of the various agents and numerous options for the treatment of postsurgical pain after dental implant surgery, a pain control protocol was formulated to aid in the proper administration of these agents. According to the World Health Organization guidelines, the procedure and patient must be evaluated and classified as mild, moderate, or severe.

Mild Pain

Mild pain is self-limited and usually will be resolved with normal recommended doses of NSAIDs.

Moderate Pain

Moderate pain is more intense pain than mild and usually will not be resolved totally by NSAIDs. It will interfere with function and disrupt the activities of daily living.

Severe Pain

Severe pain is defined as pain that interferes with some or all of the activities of daily living. The patient may be confined to bed, and strong opioid treatment will need to be continued for days. Adjuvant drug therapies may be needed for supplementation.

Control of Postoperative Surgical Pain

The goal of postsurgical pain management is to optimize patient comfort through pharmacologic and behavioral strategies. The World Health Organization formulated an analgesic "ladder" for the treatment of pain management. The following protocol describes three steps in the treatment of acute pain (Box 21-5)[103]:

1. The first step is to maximize the use of NSAIDs (acetaminophen, ibuprofen) for mild to moderate pain. Adjuvant medications such as glucocorticoids and cryotherapy are often suggested.
2. When moderate pain is expected or persists, an opioid (hydrocodone, codeine) should be added

WWW.HIGHDENT.IR

Box 21-5 World Health Organization Analgesic Ladder

Three-Step Conceptual Model

1. Nonopioid + adjuvant
2. Nonopioid + adjuvant + opioid (moderate)
3. Nonopioid + adjuvant + opioid (severe)

to the NSAID. The fixed dose of opioids with the NSAIDs provides additive analgesia. Glucocorticoids and cryotherapy are encouraged.

3. Moderate to severe pain that is expected or persists should be treated by increasing the dosage of the opioid. Glucocorticoids and cryotherapy are of particular benefit when not contraindicated.

With the guidelines from the World Health Organization, a pain control protocol was formulated for treatment of procedures based on the expected postoperative pain (Table 21-8).

Table 21-8 Recommended Pain Control Protocol

DRUG	DOSE
PCP1: Mild Pain Expected	
Ibuprofen	400 mg 1 hr before
PCP 2: Mild to Moderate Pain Expected	
Ibuprofen + Hydrocodone (Vicodin)	400 mg 1 hr before surgery + continue 4 times daily for 2 days 5 mg/500 mg as needed
PCP 3: Moderate Pain Expected	
Ibuprofen + Hydrocodone (Vicodin ES)	400 mg 1 hr before surgery + continue 4 times daily for 2 days, then as needed 7.5 mg/750 mg 4 times daily for 2 days, then as needed
PCP 4: Severe Pain Expected	
Ibuprofen + Hydrocodone (Vicodin HP)	400 mg 1 hr before surgery + continue 4 times daily for 4 days, then as needed 10 mg/660 mg 4 times daily for 2 days, then as needed

Local Anesthetics

Local anesthetics are an integral component of all dental implant surgical procedures. They are necessary to perform surgery without pain and are effective for decreasing onset and duration of pain. The dental surgeon must have significant knowledge of the pharmacokinetics of the different local anesthetics used in implant dentistry. The most commonly used dental anesthetics are amides, which are known for their low toxicity and relative lack of allergenicity.

Local anesthetics prevent postoperative pain by blocking the generation and conduction of action potentials in sensory neurons. This will prevent surgically induced nociceptive impulses from reaching the central nervous system and causing centrally mediated postoperative hyperalgesia. Table 21-9 provides local anesthetic dosage information.

Lidocaine

The compound with which most other local anesthetics are compared is 2% lidocaine-1/100,000 epinephrine.

Table 21-9 Local Anesthetic Dosage Information

ANESTHETIC SOLUTION	MAXIMUM DOSE	pKa	ONSET (min)	DURATION (min)		ELIMINATION HALF-LIFE (min)
				MAXILLA	MANDIBLE	
2% Lidocaine (1:100 K epinephrine)	7 mg/kg	7.9	2-4	170	190	90
2% Mepivacaine (1:20 K neocobefrin)	6.6 mg/kg	7.6	2-4	130	185	115
4% Articaine (1:100 K epinephrine)	7 mg/kg	7.8	2-4	140	270	20
0.5% Bupivacaine (1:200 K epinephrine)	1.3 mg/kg	8.1	5-8	340	440	210
3% Mepivacaine No epinephrine	6.6 mg/kg	7.6	2-4	90	165	115

Data from Haas DA: An update on local anesthetics in dentistry, *J Can Dent Assoc* 68:546-551, 2002.

WWW.HIGHDENT.IR

This solution is most commonly used in infiltration or block anesthesia and is considered a medium-duration anesthetic. Lidocaine is supplied in two other forms: a higher-concentration vasoconstrictor (1/50,000 epinephrine) and with no vasoconstrictor (plain).

Mepivacaine

Mepivacaine is an anesthetic that is very similar to lidocaine in onset of action, duration, and toxicity. The usual dosage used in dentistry is a 2% solution with the addition of 1/20,000 levonordefrin (Neo-Cobefrin) as the vasoconstrictor. This local anesthetic is also made in a 3% (plain) solution, which is used for short procedures or when a vasoconstrictor is contraindicated.

Articaine

Articaine is a newer amide-type of anesthetic that was approved in 2000 by the U.S. Food and Drug Association for use in the United States. Articaine differs structurally from other amide anesthetics, allowing it to have a better lipid solubility, which improves permeability of the lipid barriers in nerve membranes. Second, articaine has a very short half-life (20 minutes) in comparison to the other amide anesthetics. This shorter half-life results because it is hydrolyzed over 90% by plasma esterases and not by the liver as with the other amides. As a result, articaine is of less concern in liver-impaired individuals and a safer drug for reinjections in longer-duration procedures.

Long-Acting Anesthetics

Postoperative dental pain has been shown to reach its maximum intensity during the first 12 hours postoperatively.[104] When comparing analgesia (reduction in the sensation of pain) with anesthesia (complete elimination of feeling and sensation of pain), complete elimination of pain can be beneficial throughout the immediate postoperative period. Local anesthetics play a key role in the postoperative pain experience for the patients. If the implant surgeon can keep the patient comfortable during the initial period, pain and discomfort in the short term will also be minimized. The greater duration of anesthesia and decreased postoperative pain is effective in reducing the amount of analgesics required after surgery.[105]

The most common long-acting amide anesthetic is bupivacaine (Marcaine). This local anesthetic can play a vital role in pain management. Because of its unique pharmacokinetics, bupivacaine has been studied extensively and has been proven to be safe and far superior to other long-acting local anesthetics. Bupivacaine is an amide local anesthetic that is structurally similar to lidocaine and mepivacaine. It is more potent and less toxic than other types of amide anesthetics. Because of its high pKa (8.1), bupivacaine lasts two to three times longer than lidocaine or mepivacaine. The epinephrine concentration of bupivacaine is much lower (1/200,000 epinephrine) than standard anesthetics, thus limiting its ability to affect hemostasis.

Local Anesthetic Overdosage

A serious complication, local anesthetic overdosage, is of great concern in implant dentistry. Because many implant-related surgeries are of longer duration, a greater amount of anesthetic is often administered. Special attention must be taken during implant surgery as to the number of cartridges and type of anesthetic used during a procedure. Table 21-10 lists anesthetics and the manufacturers' maximum recommended dose by weight of patient to carpules. However, the maximum number of cartridges is time-dependent. The elimination half-life is not indicative of anesthetic duration; however, it may be used as a guide for repeated anesthetic administration during a lengthy procedure. After one half-life, as much as 50% of the permissible dose can be administered with reasonable safety if liver function is normal.

Special care must be given to the use of combination local anesthetics. In implant dentistry, it is common to use two amide anesthetics together—lidocaine and bupivacaine. Although acceptable, total doses should not exceed combined maximum recommended doses. Calculations should factor in the total dose of the combination and whether sufficient time has elapsed for elimination of the initial dose.[106] If local anesthetic toxicity reactions occur,[107] CNS excitation, convulsions,

Table 21-10 Maximum Manufacturer-Recommended Number of Anesthetic Capsules

WEIGHT OF PATIENT (lb)	2% LIDOCAINE 1/100 K EPINEPHRINE	2% MEPIVACAINE 1/20 K NEOCOBEFRIN	4% ARTICAINE 1/100 K EPINEPHRINE	5% BUPIVACAINE 1/200 K EPINEPHRINE
80	6.5	6.5	3.5	5
100	8	8	4.5	6.5
120	10	10	5.5	8
140	11.5	11	6	9
160	13	11	7	10
180	13.5	11	7	10
200	13.5	11	7	10

Data from Malamed SF: *Handbook of local anesthesia,* ed 4, St Louis, 1997, Mosby.

WWW.HIGHDENT.IR

Box 21-6 Signs and Symptoms of Local Anesthetic Toxicity

Mild Symptoms

Talkativeness
Slurred speech
Apprehension
Localized muscle twitching
Lightheadedness/dizziness
Tinnitus
Disorientation

Progressive Symptoms

Lethargy
Unresponsiveness
Drowsiness/sedation
Lack of muscle tone
Mild drop in blood pressure, heart, and respiratory rate

Adapted from Bennett CR: *Monheinis local anesthesia and pain control in dental practice,* ed 7, St Louis, 1984, Mosby.

respiratory depression, and cardiac arrest may occur (Box 21-6).

Most amide anesthetics (except for articaine) are metabolized by the liver by a microsomal enzyme system. Therefore special attention should be given to patients with decreased liver function, especially in elderly patients (e.g., chronic alcoholism, hepatitis). The half-life of lidocaine has been shown to be greater than 2.5 times the normal values in patients with hepatic disease.[108] Special attention must be given to the amount of anesthetic used and concern for reinjection must be strictly evaluated in these patients. In addition to liver dysfunction, the kidneys are the primary organs responsible for excretion of the local anesthetics and its metabolites. Patients with significant renal impairment will also have difficulty in removing the anesthetics from the blood, resulting in an increased chance of toxicity.

Patients with cardiovascular disease should be well evaluated before the use of epinephrine-containing anesthetics, and care should be taken as to the amount of epinephrine administered. Recommendations on the maximum safe dose for a healthy patient are 0.2 mg epinephrine versus 0.04 mg epinephrine for the cardiac-impaired patient. It should be noted that when epinephrine is not included in the anesthetic, the systemic uptake of the drug is more rapid and the maximum number of carpules given is significantly less in comparison to anesthetics with vasoconstrictors.

Long-Acting Anesthetics in Implant Dentistry

To keep the patient as comfortable as possible, the use of long-acting anesthetics is highly recommended both in the beginning and at the end of the procedure. By administering a long-acting anesthetic at the end, the patient will remain "pain free" longer and will have a decrease in the initiation of noxious stimuli. However, care must be given to the number and amount of local anesthetic to avoid overdosage.

SEDATIVE AGENTS

The use of conscious sedation is a valuable adjunct to dental implant procedures. The American Dental Association defines *conscious sedation* as a minimally depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway and respond appropriately to physical stimulation or verbal command and that is produced by a pharmacologic or nonpharmacologic method or combination thereof.[109] Several sedative agents are currently available for oral and intravenous sedation. Table 21-11 provides the most commonly used oral and intravenous sedative agents.

Benzodiazepines

The benzodiazepines are the most effective drugs available for dental-related anxiety. These drugs have depressant effects on the subcortical levels of the CNS. Benzodiazepines produce anxiolysis and anterograde amnesia, which are extremely useful for patients undergoing conscious sedation for dental procedures. The exact mechanism is not known, but benzodiazepines are thought to have an effect on the limbic system and the thalamus, which are involved with emotions and behavior.[110]

Diazepam (Valium)

Diazepam is usually not an effective agent for highly apprehensive patients unless administered intravenously. However, it is extremely effective if given orally the night before the procedure with a dose of 5 to 10 mg. Advantages of diazepam for dental procedures is that it reduces salivary flow and relaxes skeletal muscles. The main disadvantage of diazepam is the 24-hour half-life for adults and an 85-hour half-life for elderly patients. The active metabolites (desmethyldiazepam and oxazepam) are responsible for the prolonged sedation and recovery along with impaired psychomotor impairment.[110,111]

Midazolam (Versed)

Midazolam is a fast-acting benzodiazepine that is twice as potent as diazepam. It is available as a syrup and also as a formulated injectable solution. Midazolam possesses anticonvulsant properties and also is an excellent muscle relaxant, sedative, and amnesic. The inhibitory effects in the CNS are intensified; therefore midazolam should not be combined with other CNS depressant drugs.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Table 21-11 Most Commonly Used Oral and Intravenous Sedative Agents

SEDATIVE AGENT	CLASS	ADMINISTRATION	ONSET (min)	DURATION (hr)	HALF-LIFE (hr)	ACTIVE METABOLITES	ORAL DOSE	IV DOSE	AMNESIA	ANALGESIA
Triazolam	Benzodiazepine	PO	60	1-2	2-3	No	0.125-0.25 mg	—	Yes	No
Diazepam	Benzodiazepine	PO/IV	PO: 60 IV: 1-2	0.25-0.5	21-37	Yes	0.2-0.5 mg/kg; maximum 15 mg	0.1 mg/kg	Yes	No
Lorazepam	Benzodiazepine	PO/IV	Oral: 120-240	IV: 1-2	10-20	No	0.053 mg/kg; maximum 4 mg	0.03-0.04 mg/kg	Yes	No
Brevital	Barbiturate	IV	0.5	0.3	4	No	—	0.2-0.4 mg/kg	Yes	No
Fentanyl	Narcotic	IV	0.5	0.75-1	3-4	No	—	1-2 µg/kg	No	Yes
Propofol	Sedative hypnotic	IV	0.2-0.5	3-8 min	0.5-1.5	No	—	25-100 µg/kg/min	Yes	No
Midazolam	Benzodiazepine	PO/IV	0.5-1	0.25-1.25	1-4	No	0.5 mg/kg	0.01-0.1 mg/kg	Yes	No

PO, By mouth; *IV,* intravenous.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Table 21-12 Procedure-Specific Pharmacologic Protocol for Oral Implantology

TYPE	PATIENT SELECTION	PROCEDURES	ANTIBIOTIC	NSAID	GLUCOCORTICOID	ANTIMICROBIAL	ANALGESIC
Type 1	ASA1/ASA2	Simple extractions Single-tooth implants/healed sites Second-stage Uncovery Limited soft tissue reflection surgery	Amoxicillin: 1 g 1 hr before surgery	Ibuprofen: 400 mg 1 hr before surgery		Chlorhexidine (intraoral/extraoral) 1/2 oz two times daily for 2 wk	Pain control protocol*
Type 2	ASA1/ASA2	Multiple simple extractions Traumatic extractions Multiple implants/limited reflection Socket grafting Immediate implants without pathology	Amoxicillin: 1 g 1 hr before surgery	Ibuprofen: 400 mg 1 hr before surgery	Decadron 4 mg: 1 tablet morning of surgery	Chlorhexidine (intraoral/extraoral) 1/2 oz 2 times daily for 2 wk	Pain control protocol
Type 3	ASA1/ASA2	Membrane bone grafting (allograft/xenograft/alloplast) Multiple implants/extensive reflection Multiple immediate implants	Amoxicillin: 1 g 1 hr before surgery, 500 mg 3 times daily for 3 days	Ibuprofen: 400 mg 1 hr before surgery 400 mg 4 times daily for 2 days	Decadron 4 mg: 2 tablets morning of surgery 2 tablets in the morning day after surgery 1 tablet in the morning 2 days after surgery	Chlorhexidine (intraoral/extraoral) 1/2 oz 2 times daily for 2 wk	Pain control protocol
Type 4	Any of the following: ≥ASA 2 Long-duration surgery Less experienced surgeon Immunocompromised Active periodontal disease	Full-arch implant/extensive reflection Sinus lift (SA-2) Autogenous block bone grafts	Amoxicillin: 1 g 1 hr before surgery, then 500 mg 3 times daily for 5 days	Ibuprofen: 400 mg 1 hr before surgery, 400 mg 3 times daily for 2 days, then for evening pain	Decadron 4 mg: 2 tablets morning of surgery 2 tablets morning day after surgery 1 tablet morning 2 days after surgery	Chlorhexidine (intraoral/extraoral) 1/2 oz 2 times daily for 2 wk	Pain control protocol
Type 4 sinus	SA-3/SA-4 sinus patients	SA-3/SA-4 sinus patients	Augmentin (875 mg/125 mg): 2 tablets starting 1 day before surgery, then 1 tablet 2 times daily for 5 days	Ibuprofen: 400 mg 1 hr before surgery 400 mg 3 times daily for 2 days, then for evening pain	Decadron 4 mg: 3 tablets in the morning day before surgery 2 tablets morning of surgery 2 tablets in the morning day after surgery 1 tablets in the morning 2 days after surgery	Chlorhexidine (intraoral/extraoral) 1/2 oz 2 times daily for 2 wk	Pain control protocol

ASA, American Society of Anesthesiologists; NSAID, nonsteroidal antiinflammatory drug; *SA,* subantral.
* See Table 21-8 for pain control protocols.

WWW.HIGHDENT.IR

Triazolam (Halcion)

Triazolam is an orally administered benzodiazepine and short-term hypnotic drug. When given orally, this drug is fast acting and has been shown to be safe and effective for dental procedures. Studies have shown that triazolam given in doses of 0.25 to 0.5 mg does not produce adverse effects in respiration, heart rate, or arterial pressure. This drug is also ideal for patients with hypertension, as the blood pressure has been shown to decrease by five points.[110, 111]

Additional Sedative Anxiolytics

Fentanyl

Fentanyl is a synthetic opioid agonist narcotic that produces analgesia, drowsiness, sedation, and euphoria, but no amnesia. All opioid agonists produce dose-dependent depression of ventilation. Respiratory depression is a result of a decreased response of the ventilatory centers to carbon dioxide. For this reason, care should be taken when administering opioid agonists, especially in combination with other sedatives. Nausea and vomiting are another undesirable effect of opioid agonists. Opioid-induced nausea and vomiting are caused by direct stimulation of dopamine receptors in the chemoreceptor trigger zone in the fourth floor of the fourth ventricle.[110]

Propofol (Diprivan)

Propofol is an intravenous sedative-hypnotic agent commercially introduced in the United States in 1989 by Zeneca Pharmaceuticals. It was the first of a new class of intravenous anesthetic agents: the alkylphenols. Propofol is an ideal sedative anesthetic for dentistry because it is fast-acting and possesses a short half-life. The elimination half-life of propofol has been estimated to be between 2 and 24 hours. However, its duration of clinical effect is much shorter because propofol is rapidly distributed into peripheral tissues. Because of its pronounced respiratory depressant effect and its narrow therapeutic range, propofol should be administered only by individuals trained in airway management.[110,111]

Reversal Agents

Flumazenil (Anexate, Lanexat, Mazicon, Romazicon) is a benzodiazepine antagonist used as a reversal agent for the treatment of benzodiazepine overdose. It reverses the effects of benzodiazepines by competitive inhibition at the benzodiazepine binding site on the $GABA_A$ receptor. It was introduced in 1987 by Hoffman-LaRoche under the name Anexate.

The onset of action is rapid and usually effects are seen within 1 to 2 minutes. The peak effect is seen at 6 to 10 minutes. The recommended dose for adults is 200 µg every 1 to 2 minutes until the effect is seen, to a maximum of 3 mg per hour. It is available as a clear, colorless solution for intravenous injection, containing 500 µg in 5 mL. It is hepatically metabolized to inactive compounds, which are excreted in the urine.[110,111]

NOTE: Many benzodiazepines have longer half-lives than flumazenil. Therefore repeated doses of flumazenil may be required to prevent recurrent symptoms of overdosage after the initial dose of flumazenil wears off. It is hepatically metabolized to inactive compounds that are excreted in the urine.

Naloxone (Narcan) is a drug used as a reversal for narcotic toxicity. Naloxone is injected intravenously for fastest action. The drug acts after about 2 minutes, and its effects may last about 45 minutes.

Many opioids have a longer half-life than naloxone. Therefore patients receiving naloxone should be monitored for resedation and may require repeated doses of naloxone if resedation or respiratory depress occurs.[110,111]

COMPREHENSIVE PHARMACOLOGIC PROTOCOL

Because of the many variables (e.g., local, systemic, surgical) that need to be considered with the use of pharmacologic agents in implant dentistry, a protocol has been developed to standardize the prophylactic use of these agents. A four-category classification is proposed based on the patient's ASA status and procedure type (Table 21-12).

References

1. Munckoff W: Antibiotics for surgical prophylaxis, *Aust Prescriber* 28:38-40, 2005.
2. Stone HH, Haney BB, Kolb LD et al: Prophylactic and preventative antibiotic therapy, *Ann Surg* 189:691-699, 1979.
3. Peterson LJ, Booth DF: Efficacy of antibiotic prophylaxis in intraoral orthognathic surgery, *J Oral Surg* 34:1088, 1976.
4. Peterson JA, Cardo VA, Stratigos GT: An examination of antibiotic prophylaxis in oral and maxillofacial surgery, *J Oral Surg* 28:753, 1970.
5. Gynther GW, Kondell PA, Moberg LE et al: Dental implant installation without antibiotic prophylaxis, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 85:509-511, 1998.
6. Esposito M, Hirsch JM: Biological factors contributing to failure of osseointegrated oral implants, *Eur J Oral Sci* 106:721-764, 1998.
7. Laskin D, Dent C, Morris H: The influence of preoperative antibiotics on success of endosseous implants at 36 months, *Annu Periodontol* 5:166-174, 2000.
8. Dent CD, Olson JW, Farish SE et al: Influence of preoperative antibiotics on success of endosseous implants up to and including stage 2 surgery, *J Oral Maxillofac Surg* 55:19-24, 1997.
9. Larsen P, McGlumphy E: Antibiotic prophylaxis for placement of dental implants, *J Oral Maxillofac Surg* 51: 194, 1993.
10. Burke JF: The effective period of preventive antibiotic action in experimental incisions and dermal lesions, *Surgery* 50:161, 1961.

WWW.HIGHDENT.IR

11. Peterson LJ: Antibiotics: their use in therapy and prophylaxis. In Kruger GO, editor: *Oral and maxillofacial surgery*, St Louis, 1984, Mosby.
12. Woods RK, Dellinger MD: Current guidelines for antibiotic prophylaxis surgical wounds, *Am Fam Physician* 57:2731-2740, 1998.
13. Peterson LJ: Antibiotic prophylaxis against wound infections in oral and maxillofacial surgery, *J Oral Maxillofac Surg* 48:617, 1990.
14. Olson M, O'Connor M, Schwartz ML: Surgical wound infection: a 5-year prospective study of 10,193 wounds at the Minneapolis VA Medical Center, *Ann Surg* 199:253, 1984.
15. Page CP, Bohnen JMA: Antimicrobial prophylaxis for surgical wounds: guidelines for clinical care, *Arch Surg* 128:79, 1993.
16. Rider CA: Infection control within the oral surgeon's office, *Compend Contin Educ Dent* 25:529-534, 2004.
17. Friberg B: Sterile operating conditions for the placement of intraoral implants, *J Oral Maxillofac Surg* 54:1334-1336, 1996.
18. Haley RW, Culver DH, Morgan WM et al: Identifying patients at risk of surgical wound infection: a simple multivariate index of patient susceptibility and wound contamination, *Am J Epidemiol* 121:206-215, 1985.
19. Garibaldi RA, Cushing D: Risk factors for post-operative infection, *Am J Med* 91(suppl 3B):158S-157S, 1991.
20. Cruse PJ, Foord R: A five-year prospective study of 23,649 surgical wounds, *Arch Surg* 107:206-210, 1973.
21. Cruse PJ, Foord R: The epidemiology of wound infection: a 10-year prospective study of 62,939 wounds, *Surg Clin North Am* 60:27-40, 1980.
22. Gristina AG, Costerton JW: Bacterial adherence and the glycocalyx and their role in musculoskeletal infections, *Orthop Clin North Am* 15:517-535, 1984.
23. Lee KH, Maiden MF: Microbiata of successful osseointegrated dental implants, *J Periodontol* 70:131, 1999.
24. Drake DR, Paul J: Primary bacterial colonization of implant surfaces, *Int J Oral Maxillofac Implants* 14:226, 1999.
25. Culver DH, Horan TC, Gaynes RP et al: Surgical wound infection rates by wound class, operative procedure and patient risk index: National Nosocomial Infections Surveillance System, *Am J Med* 91:152-157, 1991.
26. Greenberg RN, James RB, Marier RL et al: Microbiologic and antibiotic aspects of infections in the oral and maxillofacial region, *J Oral Surg* 37:873-884, 1979.
27. Aderhold L, Knothe H, Frenkel G: The bacteriology of dentogenous pyogenic infections, *Oral Surg Oral Med Oral Pathol* 52:583-587, 1981.
28. Lewis MA, MacFarlane TW, McGowan DA: Quantitative bacteriology of acute dento-alveolar abscesses, *J Med Microbiol* 21:101-104, 1986.
29. Norris LH, Doku HC: Antimicrobial prophylaxis in oral surgery, *Oral Maxillofacial Surg Infect* 2:85-92, 1992.
30. Hossein K, Dahlin C: Influence of different prophylactic antibiotic regimens on implant survival rate: a retrospective clinical study, *Clin Dent Res Relat Res* 7:32-35, 2005.
31. Binhamed A, Stowkeych A: Single preoperative dose versus long-term prophylactic regimens in dental implant surgery, *Int J Oral Max Implants* 20:115-117, 2005.
32. Alanis A, Weinstein AJ: Adverse reactions associated with the use of oral penicillins and cephalosporins, *Med Clin North Am* 67:113, 1983.
33. Parker CW: Allergic reactions in man, *Pharmacol Rev* 34:85-104, 1982.
34. Pichichero ME: Prescribing cephalosporins to penicillin-allergic patients, *North Am Pharmacother* 54:1-4, 2006.
35. Newman MG, Van Winkehoff AJ: *Antibiotic and antimicrobial use in dental practice*, ed 2, Chicago, 2001, Quintessence.
36. Hugo WB, Longworth AR: The effects of chlorhexidine on the electrophoretic mobility, cytoplasmic constituents, dehydrogenase activity and cell walls of *E. coli* and *S. aureus*, *J Pharmacy Pharmacol* 18:569-578, 2001.
37. Ciancio SG, Bourgault PC: *Clinical pharmacology for dental professionals*, ed 3, Chicago, 1989, Year Book Medical Publishers.
38. Schiott C, Loe H: The effect of chlorhexidine mouthrinses on the human oral flora, *J Periodont Res* 5:84-89, 1970.
39. Bonesvoll P, Lokken P: Influence of concentration, time, temperature and pH on the retention of chlorhexidine in the oral cavity after mouth rinses, *Arch Oral Biol* 19:1025-1029, 1974.
40. Helgeland K, Heyden G: Effect of chlorhexidine on animal cells in vitro, *Scand J Dent Res* 79:209-215, 1971.
41. Goldschmidt P, Cogen R: Cytopathologic effects of chlorhexidine on human cells, *J Periodontol* 48:212-215, 1977.
42. Langeback J, Bay L: The effect of chlorhexidine mouthrinse on healing after gingivectomy, Scand J Dent Res 84:224-228, 1976.
43. Newman MG, Sanz M: Effect of 0.12% chlorhexidine on bacterial recolonization following periodontal surgery, *J Periodontol* 60:577-581, 1989.
44. Sanz M, Newman MG: Clinical enhancement of post-periodontal surgical therapy by a 0.12% chlorhexidine gluconate mouthrinse, *J Periodontol* 60:570-576, 1989.
45. Brownstein CN, Briggs SD: Irrigation with chlorhexidine to resolve naturally occurring gingivitis: a methodologic study, *J Clin Periodontol* 17:558, 1990.
46. Beiswanger DD, Mallat ME: Clinical effects of a 0.12% chlorhexidine rinse as an adjunct to scaling and root planning, *J Clin Dent* 3:33, 1992.
47. Larson PE: The effect of a chlorhexidine rinse on the incidence of alveolar osteitis following the surgical removal of impacted third molars, *J Oral Maxillofac Surg* 49:932, 1991.
48. Ragano JR, Szkutnik AJ: Evaluation of 0.12% chlorhexidine rinse on the prevention of alveolar osteitis, *Oral Surg Oral Med Oral Pathol* 72:524, 1991.
49. Lang NP, Schild U: Effect of chlorhexidine (0.12%) rinses on periodontal tissue healing after tooth extraction. I. Clinical parameters, *J Clin Periodontol* 21:422, 1994.
50. Hammerle CHF, Fourmousis I: Successful bone fill in late peri-implant defects using guided tissue regeneration: a short communication, *J Periodontol* 66:303, 1995.
51. Thomson-Neal D, Evans GH: Effects of various prophylactic treatments on titanium, sapphire and hydroxyapatite-coated implants: an SEM study, *Int J Perodont Restorative Dent* 9:300, 1989.
52. Lambert PM, Morris HF: The influence of 0.12% chlorhexidine digluconate rinses on the incidence of infectious complications and implant success, *J Oral Maxillofac Surg* 55:25-30, 1997.
53. *Accepted dental therapeutics*, ed 40, Chicago, 1984, American Dental Association.
54. Esen E, Tasar F: Determination of the anti-inflammatory effects of methylprednisolone on the sequelae of third molar surgery, *J Oral Maxillofac Surg* 57:1201-1206, 1999.

WWW.HIGHDENT.IR

55. Messer EJ, Keller JJ: The use of intraoral dexamethasone after extraction of mandibular third molars, *Oral Surg* 40:594-597, 1975.
56. Neuper EA, Lee JW, Philput CB et al: Evaluation of dexamethasone for reduction of postsurgical sequelae of third molar removal, *J Oral Maxillofac Surg* 50: 1177-1182, 1992.
57. Hooley JR, Hohl TH: Use of steroids in the prevention of some complications after traumatic surgery, *J Oral Surg* 32:8634-866, 1974.
58. Williamson LW, Lorson EL, Osborn DB: Hypothalamic-pituitary-adrenal suppression after short-term dexamethasone therapy for oral surgical procedures, *J Oral Surg* 38:20-28, 1980.
59. Misch CE, Moore P: Steroids and the reduction of pain, edema and dysfunction in implant dentistry, *Int J Oral Implant* 6:27-31, 1989.
60. Nichols T, Nugent CA, Tyle, FH: Diurnal variation in suppression of adrenal function by glucocorticoids, *J Clin Endocrinol Metab* 25:343, 1965.
61. Bahn SL: Glucocorticosteroids in dentistry, *J Am Dent Assoc* 105:476-481, 1982.
62. Messer EJ, Keller JJ: The use of intraoral dexamethasone after extraction of mandibular third molars, *Oral Surg* 40:594-597, 1975.
63. Ross R, White CP: Evaluation of hydrocortisone in prevention of postoperative complications after oral surgery: a preliminary report, *J Oral Surg* 16:220, 1958.
64. Guernsey LH, DeChamplain RW: Sequelae and complications of the intraoral sagittal osteotomy in the mandibular, *Oral Surg Oral Med Oral Pathol* 32:176-192, 1971.
65. Moore PA, Barr P, Smiga ER et al: Preemptive rofecoxib and dexamethasone for prevention of pain and trismus following third molar surgery, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 99:E1-E7, 2005.
66. Hooley JR, Francis FH: Betamethasone in traumatic oral surgery, *J Oral Surg* 27:398-403, 1969.
67. Baxendale BR, Vater M, Lavery KM: Dexamethasone reduces pain and swelling following extraction of third molar teeth, *Anaesthesia* 48:961-964, 1993.
68. Sisk A, Bonnington GJ: Evaluation of methylprednisolone and flurbiprofen for inhibition of postoperative inflammatory response, *Oral Surg Oral Med Oral Pathol* 60; 137-145, 1985.
69. Montgomery MT, Hogg JP, Roberts DL et al: The use of glucocorticosteroids to lessen the inflammatory sequelae following third molar surgery, *J Oral Maxillofac Surg* 48;179-187, 1990.
70. Wang JJ, Ho ST, Lee SC et al: The prophylactic effect of dexamethasone on postoperative nausea and vomiting in women undergoing thyroidectomy: a comparison of droperidol with saline, *Anesth Analg* 89:200-203, 1999.
71. Liu K, Hsu CC, Chia YY: Effect of dexamethasone on postoperative emesis and pain, *Br J Anaesth* 80:85-86, 1998.
72. Beaver WT: Combination analgesics, *Am J Med* 77:38-53, 1984.
73. Filho JRL, Silva EDO: The influence of cryotherapy of reduction of swelling, pain, trismus after third molar extraction, *J Am Dent Assoc* 136:774-778, 2005.
74. Forsgren H, Heimdahl A: Effect of application of cold dressings on postoperative course in oral surgery, *Int J Oral Surg* 14:223-228, 1985.
75. Carr DB, Jacox AK: *Clinical practice guidelines for acute pain management: operative or medical procedures and trauma*, Washington, DC, 1992, Agency for Health Care Policy and Research, DHHS publication no. 95-0034.
76. Bachiocco V, Scesi M: Individual pain history and familial pain tolerance models: relationships to post-surgical pain, *Clin J Pain* 9:266-271, 1993.
77. Taenzer P, Melzack R: Influence of psychological factors on postoperative pain, mood and analgesic requirements, *Pain* 24:331-342, 1986.
78. Curtis, JW, McClain, JB: The incidence and severity of complication and pain following periodontal surgery, *J Periodontol* 56:597-601, 1985.
79. Jackson DL, Roszkowski MT, Moore PA: Management of acute postoperative pain. In Fonseca RJ, editor: *Oral and maxillofacial surgery*, London, 2000, Saunders.
80. Gottschalk A, Smith DS: New concepts in acute pain therapy: preemptive analgesia, *Am Fam Physician* 63:1979-1984, 2001.
81. Huynh MP, Yagiela JA: Current concepts in acute pain management, *J Calif Dent Assoc* May 31:1-13, 2003.
82. Chandraasekharan NV, Dai H: COX-3, a cyclooxygenase-1 variant inhibited by acetaminophen and other analgesic/antipyretic drugs: cloning, structure, and expression, *Proc Natl Acad Sci U S A* 99:13926-13931, 2002.
83. Basbaum AL, Leveine JD: Opiate analgesia. How central is a peripheral target? *N Engl J Med* 325:1168-1169, 1991.
84. Schwab JM, Schluesener HJ: COX-3: just another COX or the solitary elusive target of paracetamol? *Lancet* 361:981-982, 2003.
85. Ahmad N, Grad HA: The efficacy of non-opioid analgesics for post-operative dental pain: a meta-analysis, *Anesth Prog* 44:119-126, 1997.
86. Dionne RA, Gordon SM: Nonsteroidal anti-inflammatory drugs for acute pain control, *Dent Clin North Am* 38: 645-667, 1994.
87. Jackson, DL, Moore PA: Preoperative nonsteroidal anti-inflammatory medication for the prevention of postoperative dental pain, *J Am Dent Assoc* 119:641-647, 1989.
88. Ruffalo RL, Jackson RL, Ofman JJ et al: The impact of NSAID selection on gastrointestinal injury risk for cardiovascular events: identifying and treating patients at risk, *Therapy* 20:570-576, 2002.
89. Hernández-Diaz S, García-Rodríguez LA: Epidemiologic assessment of the safety of conventional nonsteroidal anti-inflammatory drugs, *Am J Med* 110(suppl 3A): 20S-27S, 2001.
90. Smalley WE, Griffin MR: The risks and costs of upper gastrointestinal complications attributable to NSAIDS, *Gastointerol Clin North Am* 25:373-379, 1996.
91. Raney LH: *Evidence-based use of NSAIDS in the ED*, 2001. Available at http://www.emedhome.com. Accessed March 16, 2005.
92. Garcia Rodriguez LA, Cattaruzzi C, Troncon MG et al: Risk of hospitalization for upper gastrointestinal tract bleeding associated with ketorolac, other NSAIDs, anti-inflammatory drugs, calcium antagonists, and other antihypertensive drugs, *Arch Intern Med* 158:33-39, 1998.
93. Busson M: Update on ibuprofen: review article, *J Int Med Res* 14:53-62, 1986.
94. Cooper SA: Five studies on ibuprofen for post-surgical dental pain, *Am J Med* 77:70-77, 1984.
95. Winter L, Bass W: Analgesic activity of ibuprofen (Motrin) in post-operative oral surgical pain, *Oral Surg Oral Med Oral Pathol* 45:159-166, 1978.
96. Seymour RA, Ward-Booth P: Evaluation of different doses of ibuprofen and ibuprofen tablets in postoperative dental pain, *Br J Oral Maxillofac Surg* 34:110-114, 1996.

WWW.HIGHDENT.IR
مرجع دندانسازان و دندانپزشکان

97. Kellstein DE, Waksman JA: The safety profile of non-prescription ibuprofen in multiple dose use: a meta-analysis, *J Clin Pharmacol* 39:520-532, 1999.
98. Driessen B, Reimann W: Interaction of the central analgesic, tramadol, with the uptake and release of 5-hydroxytryptamine in the rat brain in vitro, *Br J Pharmacol* 105:147-151, 1992.
99. Moore PA: Pain management in dental practice: tramadol vs. codeine combinations, *J Am Dent Assoc* 130:1075-1079, 1999.
100. Vickers MD, O'Flaherty D: Tramadol: pain relief by an opioid without depression of respiration, *Anaesthesia* 47:291-296, 1992.
101. Mullican WS, Lacy JR: Tramadol/acetaminophen combination tablets and codeine/acetaminophen combination capsules for the treatment of pain: a comparative trial, *Clin Ther* 23:1429-1445, 2001.
102. Medve RA, Wang A: Tramadol and acetaminophen tablets for dental pain, *Anesth Prog* 48:79-81, 2001.
103. United States Department of Health and Human Services: *Agency for Health Care Policy and Research Clinical Practice Guidelines, Number 9*, Washington, DC, 1994, U.S. Government Printing Office.
104. Seymour, RA, Blair GS: Postoperative dental pain and analgesic efficacy, *Br J Oral Surg* 21:290-297, 1983.
105. Kaurich MJ, Otomo-Corgel J: Comparison of postoperative bupivacaine with lidocaine on pain and analgesic use following periodontal surgery, *J West Soc Periodontol Periodontal Abstr* 45:5-8, 1997.
106. Moore PA: Adverse drug interactions in dental practice: interactions associated with local anesthetics, sedatives and anxiolytics, *J Am Dent Assoc* 130:541-554, 1999.
107. Moore PA: Prevention of local anesthesia toxicity, *J Am Dent Assoc* 123:60-64, 1992.
108. Thomson PD, Melmo KL: Lidocaine pharmacokinetics in advanced heart failure, liver disease, and renal failure in humans, *Ann Intern* 78:499-513, 1973.
109. American Dental Association: *American Dental Association guidelines for the use of conscious sedation, deep sedation and general anesthesia for dentists*. Available at http://www.ada.org/prof/resources/positions/statements/anesthesia_guidelines.pdf. Accessed July 16, 2007.
110. Stoelting RK: *Pharmacology and physiology in anesthetic practice,* ed 4, Philadelphia, 2006, Lippincott Williams & Wilkins.
111. *Mosby's dental drug reference,* ed 8, St Louis, 2008, Elsevier.

WWW.HIGHDENT.IR

Chapter 22

Applied Anatomy for Dental Implants

Mohamed Sharawy, Carl E. Misch

The surgical anatomy of the maxilla and mandible provide the foundation required to safely insert dental implants. The anatomy is also a requisite to the understanding of complications that may inadvertently occur during surgery, such as injury to blood vessels or nerves, as well as postoperative complication such as infection. This information also provides the operator with the confidence needed to deal with these complications. This chapter addresses those issues important in the field of oral implantology.

SURGICAL ANATOMY OF THE MAXILLA AS AN ORGAN

The maxilla is pyramidal in shape with the root of the zygoma as its apex (Figures 22-1 and 22-2). The latter can be palpated in the buccal vestibule of the oral cavity. It divides the facial surface of the maxilla into anterolateral and posterolateral surfaces of the pyramid. The third surface of the pyramid is the orbital plate of the maxilla. The base of the pyramid is the lateral wall of the nose or the medial wall of the maxillary sinus. The alveolar process of the maxilla related to the anterolateral surface carries the incisors, the canines, and the premolars, whereas that of the posterolateral surface carries the molars and ends as the maxillary tuberosity. The intraoral part of the maxilla is limited by the mucobuccal fold and the orbicularis oris muscle anteriorly and by the buccinator muscle posteriorly. The posterolateral surface of the maxilla above the mucobuccal fold forms the anterior wall of the infratemporal fossa and is difficult to palpate. However, the anterolateral surface of the maxilla beyond the mucobuccal fold can be palpated easily under the skin, as can the anterior nasal spine, the anterior nasal aperture, and the frontal process of the maxilla. Intraorally, it is possible to palpate the canine eminence, the canine fossa (distal to the canine eminence and a common site for facial access to the maxillary sinus), the maxillary tuberosity, and the hamular notch. The maxilla extends as a horizontal plate medially to form the anterior two thirds of hard palate. The horizontal plate of the palatine bone forms the posterior one third of hard palate. The palatine bone has a vertical plate that articulates with the base of the maxilla; it also has a pyramidal process that interposes between the maxillary tuberosity and the pterygoid processes of the sphenoid bone. Mucosal incision at the maxillary tuberosity that extends into the hamular notch may expose the pyramidal process of the palatine bone. Distal to this point, one may expose the medial pterygoid muscle, which takes origin from the tuberosity and the lateral pterygoid plate of the sphenoid. The medial wall of the maxilla begins at the sharp edge of the anterior nasal aperture and extends posteriorly, with a concavity that bounds the nasal fossa and continues distal to the canine. Once there, it forms the medial wall of the maxillary sinus and continues all the way back to the maxillary tuberosity. The medial wall of the maxilla provides attachment to

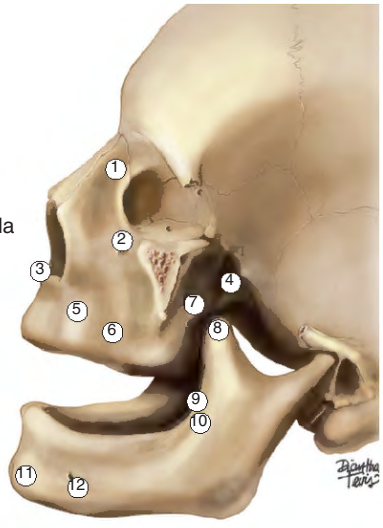
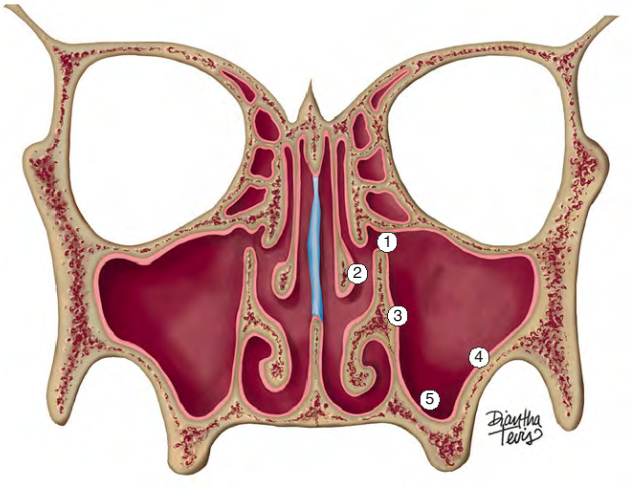


Figure 22-1 Anatomical features of the maxilla and mandible that are of clinical importance.

The authors thank Francis T. Lake for contributing to the section on blood supply of edentulous jaws and Lewis Hinley for skillful medical illustration.

WWW.HIGHDENT.IR



1. Maxillary sinus opening
2. Middle meatus
3. Medial wall of sinus
4. Lateral wall of sinus
5. Floor of sinus and alveolar recess

Figure 22-2 Anatomical features of the maxillary sinus.

the inferior nasal concha and to the vertical plate of the palatine bone. The opening of the maxillary sinus is found in the medial wall of the maxilla, close to the floor of the orbit. The opening is reduced in diameter by the uncinate process of the ethmoid bone. The latter provides the superior and middle conchae of the lateral nasal wall. The orbital plate of the maxilla forms the floor of the orbit and also the roof of the maxillary sinus. The infraorbital canal carries the infraorbital nerve and vessels, and it forms a ridge that can be seen in the sinus cavity.

Muscles Attached to the Maxilla

As the maxillary alveolar bone resorbs, the crest of the residual ridge migrates toward the muscles that take their origin from the basal bone of the maxilla. Descriptions of muscles of surgical importance to oral implantologists follow (Figures 22-3 to 22-5).

Orbicularis Oris Muscle

The orbicularis oris muscle originates from the modiolus at each corner of the mouth. The muscle fibers fan out into the upper and lower lips, where they form upper and lower peripheral portions under the skin and marginal portions under the vermilion zone of the lips. Some of the orbicularis oris fibers attach to the ala of the nose and to the nasal septum. In the midline of the upper lip, the peripheral portions from both sides interdigitate to create the philtrum. The marginal portions interdigitate and create the labial tubercle. Although unattached to the bone of the maxilla, the muscle limits the depth of the upper and lower facial vestibule. The orbicularis oris receives innervation from the buccal and mandibular branches of the facial nerve.

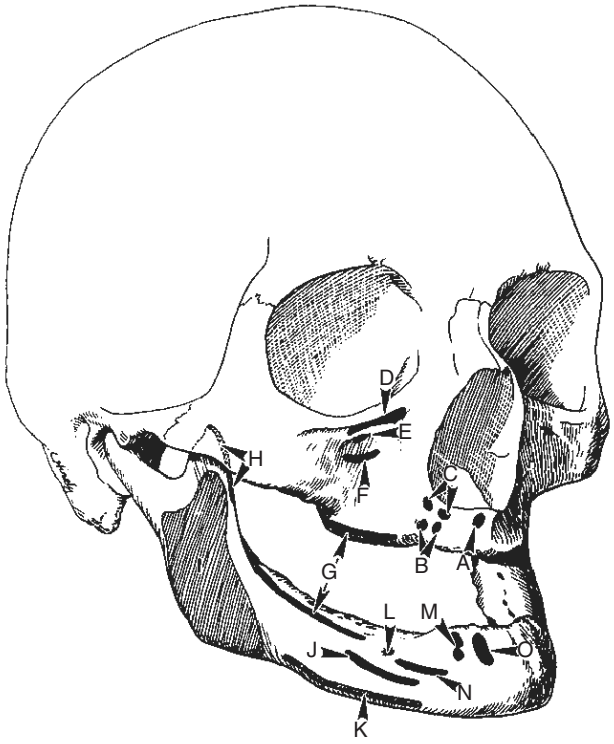


Figure 22-3 *A,* Origin of depressor septi muscle; *B,* origin of superior incisivus muscle; *C,* origin of nasalis muscle; *D,* origin of levator labii superioris muscle; *E,* infraorbital foramen; *F,* origin of levator anguli oris (caninus) muscle; *G,* origin of buccinator muscle; *H,* insertion of lateral tendon of temporalis muscle; *I,* insertion of masseter muscle; *J,* origin of depressor anguli oris (triangularis) muscle; *K,* insertion of platysma muscle; *L,* mental foramen; *M,* origin of inferior incisivus muscle; *N,* origin of depressor labii inferioris muscle; *O,* origin of mentalis muscle.

Incisivus Labii Superioris Muscle

The incisivus labii superioris muscle originates from the floor of the incisive fossa of the maxilla above the eminence of the lateral incisor and deep to the orbicularis oris. To expose the bone of the premaxilla between the canines, a mucoperiosteal flap reflection may detach the incisivus labii superioris. It may also detach the septalis and oblique fibers of nasalis muscle. The first is attached to the skin of the nasal septum and the latter to the ala of the nose. These small muscles will reattach after placement of the flap. However, if the muscles were damaged, then drooping of the septum and flaring of the ala of the nose may result.

Buccinator Muscle

The buccinator muscle originates from the base of the alveolar process opposite to the first, second, and third molar of both jaws. This muscle also takes origin from the pterygoid hamulus of the medial pterygoid plate of the sphenoid bone and therefore bridges the gap between the maxillary tuberosity anteriorly and the hamulus posteriorly. Extension of a subperiosteal frame design into the pterygoid plates may interfere with the fibers of these muscles without adding too much

WWW.HIGHDENT.IR

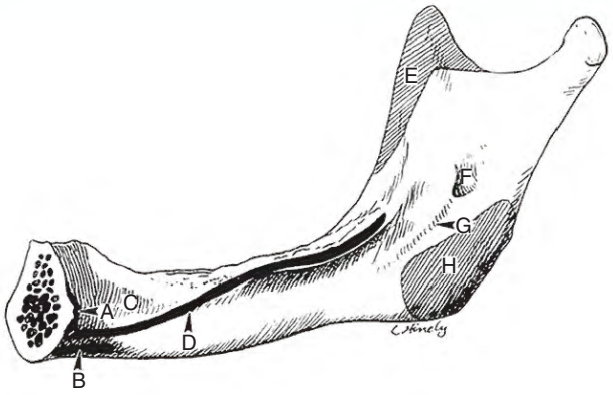


Figure 22-4 *A,* Genial tubercles, site of origin of genioglossus (superior tubercle) and geniohyoid (inferior tubercle) muscles; *B,* digastric fossa, site of origin of anterior belly of digastric muscle; *C,* sublingual fossa, location of sublingual gland; *D,* mylohyoid line, site of origin of mylohyoid muscle; *E,* insertion of medial tendon of temporalis muscle; *F,* mandibular foramen; *G,* mylohyoid groove, formed by mylohyoid nerve; *H,* site of insertion of medial pterygoid muscle.

1. Maxillary n.
2. Pterygopalatine ganglion
3. Infraorbital n.
4. Posterior superior alveolar n.
5. Middle superior alveolar n.
6. Anterior superior alveolar n.
7. Buccal n.
8. Mandibular n.
9. Lingual n.
10. Inferior alveolar n.
11. Nerve to mylohyoid
12. Auriculotemporal n.
13. Mental branch of inferior alveolar n.

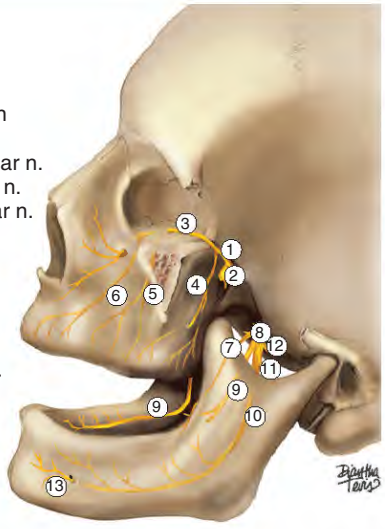


Figure 22-6 Sensory innervation of the maxilla and mandible.

1. Temporalis
2. Nasalis
3. Levator labii superioris alaque nasi
4. Levator labii superioris
5. Zygomaticus minor
6. Levator anguli oris
7. Zygomaticus major
8. Oblique portion of nasalis
9. Orbicularis oris
10. Buccinator
11. Risorius
12. Medial tendon of temporalis
13. Lateral tendon of temporalis
14. Masseter
15. Mentalis
16. Depressor labii inferioris
17. Depressor anguli oris

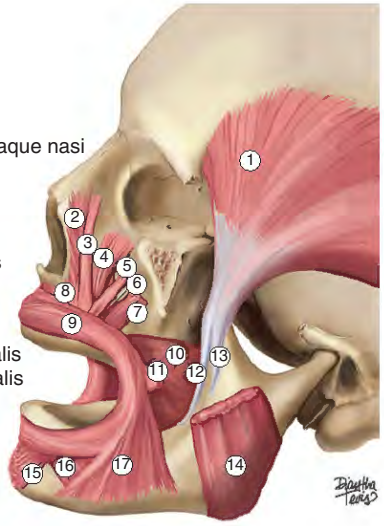


Figure 22-5 Muscles attached to the maxilla and mandible.

to the retention of the implant. When incising and reflecting the mucosa overlying the areas of the maxillary tuberosity and hamular notch before taking impressions for maxillary subperiosteal implants, avoid injuring the tendon of the tensor veli transact muscle, which passes around the pterygoid hamulus. The tendon moves on an underlying bursa whenever the soft palate moves; therefore it may become irritated by the subperiosteal frame and result in inflammation and pain. Fibers of the buccinator and medial pterygoid muscles are also found in the area of reflection. The majority of the fibers of the medial pterygoid muscle originate from the medial surface of the lateral pterygoid plate of the sphenoid bone, whereas the rest of the fibers form the tuberal head, which takes origin from the maxillary tuberosity. Near the pterygoid hamulus, a fibrous tissue raphe or, in some cases, a broad fascialike structure is found between the transaction and the superior pharyngeal constrictor muscles. In some cases, no raphe or fascia is found. Injury to the latter muscle should be avoided during reflection of the mucosa, particularly on the palatal aspect of the area of the hamulus.

Levator Labii Superioris Muscle

The levator labii superioris muscle takes origin from the infraorbital margin above the infraorbital foramen and therefore is rarely of concern to the implant surgeon. The zygomatic branch of the facial nerve innervates this muscle.

Levator Anguli Oris (Caninus) Muscle

The levator anguli oris muscle originates in the maxilla below the infraorbital foramen. The infraorbital nerve and vessels arise between this muscle and the levator labii superioris. In the severe atrophic division D maxilla, the infraorbital foramen is relatively close to the crest of the ridge. Reflection of the tissues for autogenous grafts and implant placement into sinus grafts may approximate this region and cause parasthesia. In subperiosteal implant cases that require extensive framework extension for retention, the operator should be aware of the location of the infraorbital neurovascular bundle in relation to the caninus and levator labii superioris muscles. The zygomatic branch of the facial nerve innervates the caninus muscle.

Sensory Innervation of the Maxilla

The maxillary nerve (V2) innervates the maxilla (Figure 22-6). The nerve leaves the middle cranial fossa by passing through the foramen rotundum and appears in the pterygopalatine fossa. It exits the fossa and passes briefly into the infratemporal fossa; from there it enters the floor of the orbit or the roof of the maxillary sinus by passing through the infraorbital fissure. It

WWW.HIGHDENT.IR

then exits the orbit via the infraorbital foramen. The pterygopalatine portion of the maxillary nerve provides the descending palatine and sphenopalatine branches. The sphenopalatine nerve enters the nasal cavity from the pterygopalatine fossa by passing through the sphenopalatine foramen. It supplies the nasal cavity and becomes the incisive nerve that supplies the palatine mucosa opposite to the upper six anterior teeth. The descending palatine nerve terminates as the great palatine nerve, which supplies the mucosa of the hard palate, and the lesser palatine nerves, which supply the mucosa of soft palate. These sensory nerves also carry parasympathetic fibers from the sphenopalatine ganglion that innervate the mucous glands of the palate. The infratemporal portion of V2 branches into the posterior alveolar nerve and zygomatic nerve. The latter divides into the zygomaticofacial and zygomaticotemporal cutaneous nerves. The posterior superior alveolar nerve supplies the buccal gingiva, buccal alveolar bone, second and third molars, and two roots of first molar. The infraorbital portion of V2 gives rise to anterior superior alveolar and occasionally middle superior alveolar nerves. These nerves run in bony grooves in the facial wall of the maxillary sinus under the Schneiderian membrane. The nerves supply the sinus wall and the premolars; the canine, lateral, and central incisor on the same side; and also the central incisor at the contralateral side. The infraorbital nerve exits the maxilla at the infraorbital foramen and supplies cutaneous branches to the lower eyelid, side of the nose, and upper lip. Implantologists often need to block V2 or several of its branches. Luckily these can be achieved by an intraoral route. V2 can be reached via the great palatine foramen and descending palatine canal, or via the pterygomaxillary fissure by following the slope of the posterolateral surface of the maxilla into the pterygopalatine fossa.

Posterior Superior Alveolar (Dental) Nerve

The nerve arises within the pterygopalatine fossa, courses downward and forward, passing through the pterygomaxillary fissure, and enters the posterior aspect of the maxilla. It runs between the bone and the lining of the maxillary sinus. This nerve supplies the sinus, the molars, the buccal gingiva, and the adjoining portion of the cheek; it may be injured during a sinus augmentation with a lateral approach. Clinically this does not appear to be of major consequence.

Infraorbital Nerve

This nerve is a continuation of the main trunk of the maxillary division. It leaves the pterygopalatine fossa by passing through the inferior orbital fissure to enter the floor of the orbit. It runs in the infraorbital groove and then in the infraorbital canal. The nerve exits the orbit through the infraorbital foramen to give cutaneous branches to the lower eyelid, the ala of the nose and the skin, and the mucous membrane of the lip and cheek. The infraorbital foramen is located between the levator labii superioris muscle, which takes origin above the foramen, and the levator anguli oris (caninus) muscle, which takes origin below the foramen. This foramen and neurovascular contents are within 5 to 10 mm of an extremely resorbed maxilla. When applying onlay grafts, which expose the entire maxilla, the implant dentist must be very aware of this situation. Fixation screws or implants may cause paresthesia when inserted through the graft and into this structure. Subperiosteal implants designed for an atrophied maxilla should not extend into the site of the infraorbital nerve and vessels. In some cases of maxillary sinus disorder, the site of the infraorbital foramen becomes tender, probably as a result of inflammation of the infraorbital nerve. This is an important diagnostic test for possible postoperative involvement after sinus augmentation procedures.

Middle Superior Alveolar (Dental) Nerve

This branch of the infraorbital nerve is given off as the infraorbital nerve passes through the infraorbital groove. The middle superior alveolar nerve runs downward and forward in the lateral wall of the sinus to supply the maxillary premolars. This region is routinely violated for the lateral approach to sinus grafts, with apparently no consequence.

Anterior Superior Alveolar (Dental) Nerve

This branch of the infraorbital nerve arises within the infraorbital canal. It initially runs laterally within the sinus wall and then curves medially to pass beneath the infraorbital foramen. It turns downward to supply the maxillary anterior teeth. A nasal branch passes into the nasal cavity to supply the mucosal lining of a portion of the nasal cavity. Before elevation of nasal mucosa and placement of grafts, this nerve must be anesthetized. The infraorbital nerve block or V2 block anesthesia is suggested. Implant dentists must also anesthetize this branch before placement of implants in the incisor region. The anterior, middle, and posterior superior alveolar nerves intermingle to form the superior dental plexus. The posterior, middle, and anterior superior alveolar nerves run in the facial wall of the maxillary sinus between its lining membrane and the bone. During antrostomy procedures to augment the floor of the sinus, the operator should be aware of these structures, which are present even in the absence of teeth.

Palatine Nerve

The greater (anterior) and lesser (posterior) palatine nerves supply the hard and soft palate, respectively. They exit the pterygopalatine fossa through the superior opening of the descending palatine canal, travel downward, and enter the oral cavity by way of the greater and lesser palatine foramina. The greater palatine nerve runs forward in a groove on the inferior surface of the hard palate to supply the palatal mucosa as far forward

WWW.HIGHDENT.IR

as the incisor teeth. Here the nerve communicates with the nasopalatine nerve. The nerve supplies the gingiva, mucous membrane, and glands of the hard palate. The greater palatine artery and vein accompany the nerve during its course in the hard palate. As the maxillary alveolar process atrophies, it shifts to the palate and brings the crest of the ridge closer to the groove where the greater palatine neurovascular bundle is found. The restoring dentist should be aware that an incision too palatal to the crest of the ridge in the atrophied maxilla might injure these vital structures. This foramen is entered for a V2 block anesthesia. One may find it by taking a blunt instrument and pressing firmly along the alveolar palatal bone angle. The instrument will depress over the foramen when in the correct position.

Nasopalatine (Sphenopalatine) Nerve

The nasopalatine nerve leaves the pterygopalatine fossa through the sphenopalatine foramen located in the medial wall of the fossa. It enters the nasal cavity and supplies portions of the lateral and superior aspects of the nasal cavity. The longest branch reaches the nasal septum, where it turns downward and forward, traveling on the surface of the septum. While on the septum it forms a groove on the vomer bone. The nerve supplies the nasal mucosa, descends to the floor of the nose near the septum, passes through the nasopalatine canal, and then exits onto the hard palate through the incisive foramen. The latter opening is deep to the incisive papilla. The nerve communicates with the greater palatine nerve. The incisive nerve should be anesthetized before elevation of the mucosa of the floor of the nose for subnasal grafts or implants that engage the nasal floor in the incisor region.

Arterial Supply to the Maxilla

The majority of arterial blood supply (Figure 22-7) comes from the maxillary artery, which is one of the terminal branches of the external carotid artery. The artery starts deep to the neck of the mandibular condyle (mandibular portion) and then proceeds either superficial or deep to the lateral pterygoid muscle (pterygoid portion). It then branches close to the pterygomaxillary fissure, where one branch enters the fossa (pterygopalatine portion). The other branch, called the *infraorbital artery*, enters the floor of the orbit via the infraorbital fissure; it proceeds in the infraorbital canal and exits on the face by passing through the infraorbital foramen. Branches of the maxillary artery are as follows:

1. Mandibular portion: deep auricular, tympanic, middle meningeal, and inferior alveolar arteries
2. Pterygoid portion: deep temporal, lateral pterygoid, medial pterygoid, and masseteric arteries
3. Pterygopalatine portion: posterior superior alveolar, descending palatine, and sphenopalatine arteries

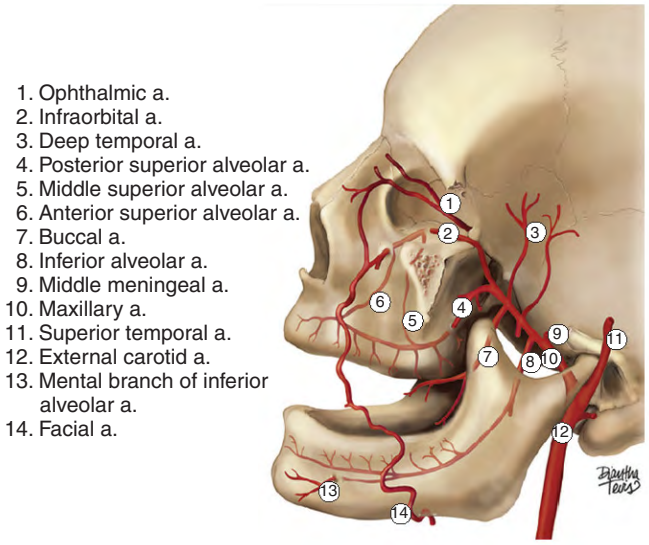


Figure 22-7 Arterial supply of the maxilla and mandible.

4. Infraorbital portion: anterior and middle superior alveolar, palpebral, nasal, and labial arteries

Supplemental arterial blood supply reaches the maxilla via two branches from the cervical portion of the facial artery (ascending palatine and tonsillar arteries), two dorsolingual arteries from the lingual artery, and the ascending pharyngeal branch of external carotid artery. All the collateral circulation reaches the maxilla from the area of the soft palate. During orthognathic surgery to correct maxillary prognathism, the surgeon often cuts the posterior, middle, and anterior superior alveolar arteries, as well as the descending palatine arteries, without compromising the blood supply to the maxilla because of the presence of supplemental blood supply from the branches mentioned previously. It is important to note that the maxillary artery supplies blood to the bone of the mandible via its inferior alveolar artery and its branches to the muscles of mastication. Detaching the masseter and medial pterygoid muscles without reattaching it could result in necrosis of the ramus of the mandible. In addition, all the arterial branches mentioned previously arise from the external carotid; therefore bilateral arteriosclerosis of the carotids, which is common in old age and in uncontrolled diabetic patients, may compromise the blood supply to the maxilla and could result in delay of healing after insertion of implants or bone grafting to the area. More detailed consideration of applied anatomy of the arterial supply to both the maxilla and mandible is presented at the end of this chapter.

Venous Drainage of the Maxilla

The veins follow the arteries and carry the same names. The maxilla drains into the maxillary vein. The latter communicates freely with the pterygoid plexus of veins and then joins the superficial temporal vein to form the

WWW.HIGHDENT.IR

posterior facial vein within the parotid gland. Infection from the maxilla may follow the maxillary vein to the pterygoid plexus veins and then to the cavernous sinuses via emissary veins, causing infected cavernous sinus thrombosis. Adequate arterial supply and healthy venous drainage are essential for bone regeneration and remodeling of bone grafts.

Lymphatic Drainage

The maxilla, including the maxillary sinuses, drains its lymphatics into the submandibular lymph nodes. In addition, the posterior portion of the maxilla and soft palate drain into the deep facial lymph nodes, part of the deep cervical nodes. Palpation of lymph nodes is an essential part of the physical examination of the head and neck.

SURGICAL ANATOMY OF THE MANDIBLE

The clinician should be familiar with the anatomical features of dentulous and edentulous mandibles, not only from radiographs but also from physical examination (see Figures 22-1 to 22-4). The symphysis, inferior border, premasseteric notch, gonial angle, lateral pole of condyle, and coronoid process are all palpable under the skin. Intraoral palpable features of the mandible from the facial surface include the external oblique ridge and retromolar triangle, with the coronoid process at its tip, the external oblique ridge bordering it laterally, and the internal oblique ridge bordering it medially. The latter is called the *temporal crest* because this is the site for insertion of the medial tendon of the temporalis muscle. The mental foramen can be located at the midpupillary line at the apices of the premolars. From the lingual aspect, palpate the internal oblique ridge and torus mandibularis at the premolar region. Reflection of mucoperiosteal flap beyond the mucobuccal fold facially exposes the mentalis muscles lateral to the midline, the mental foramen with the mental neurovascular bundle, the depressor labii inferioris and triangularis close to the inferior border in the premolar region, the transaction at the base of the alveolar process opposite to the molars, and the temporalis tendons at the anterior border of the ramus. An atrophied edentulous mandible loosens the alveolar process, and the crest of the ridge may be found at the same level as the external and internal oblique ridge. It is possible to palpate the superior genial tubercles with its genioglossus muscle attachment. Reflection of the mucoperiosteal flap after midcrest incision may expose the mental neurovascular bundle, which is abnormally located at or occasionally lingual to the crest of the ridge. The transact muscle may loose its attachment to the external oblique ridge, whereas the mylohyoid may rise above the level of the ridge. The lingual nerve, which has a close relationship to the alveolar bone of the third molar in the dentulous mandible, may run close to the crest of the edentulous ridge; in some cases it may be found under the retromolar pad.

Muscle Attachment to the Mandible

The loss of teeth begins a cascade of events that leads to alveolar bone loss in width and height. As the mandibular alveolar bone resorbs, the residual ridge migrates toward many of the muscles that originate or insert on the mandible (see Figures 22-3 to 22-5). The origin, insertion, innervation, and function of the muscles of surgical importance to the implant dentist are discussed.[1-7]

Lingual or Medial Attachments

Mylohyoid Muscle

The mylohyoid muscle is the main muscle of the floor of the mouth. It takes origin from the entire length of the mylohyoid lines on the medial aspect of the mandible bilaterally. The most posterior fibers of the mylohyoid insert into the body of the hyoid bone, whereas the other fibers meet in the midline to form a median raphe that extends from the mandible to the hyoid bone. The structures above the mylohyoid muscle are sublingual or intraoral in location, and the structures below the mylohyoid muscle are extraoral or subcutaneous. With a severely resorbed residual ridge, the origin of the mylohyoid muscle approximates the crest of the ridge, especially in the posterior mandible. In these cases surgical manipulation at the crest of the ridge may injure the mylohyoid muscle. A mandibular periosteal reflection for subperiosteal implant often reflects this muscle to the second molar region. The substructure of the implant then has a permucosal site in the first molar area and a lingual primary strut above and below the mylohyoid muscle. Surgical manipulation of the tissue of the floor of the mouth may lead to edematous swelling of the sublingual space (above the mylohyoid muscle), swelling of the submandibular space (below the mylohyoid muscle), or both. Ecchymosis resulting from blood accumulation may occur subcutaneously and/or submucosally. In some cases, infection may start and spread lingually and lead to an abscess or cellulitis either sublingually (intraoral) or submandibularly (extraoral), depending on the site of origin of the infection in relation to the origin of the mylohyoid muscle. Extensive bilateral cellulitis of the sublingual spaces may push the tongue backward or compress the pharynx, which may result in airway obstruction and necessitate a tracheotomy or cricothyroidotomy to maintain the airway. Functionally, the mylohyoid muscle raises the hyoid bone and floor of the mouth, or it can depress the mandible if the hyoid bone is fixed. The mylohyoid nerve that innervates the muscle is a motor branch of the inferior alveolar nerve. The latter is a branch of the mandibular nerve (V3).

WWW.HIGHDENT.IR

Genioglossus Muscle

The genioglossus muscle forms the bulk of the tongue. It takes origin from the superior genial tubercle. The anterior fibers insert into the dorsal surface of the tongue from the root to its tip, and the posterior fibers insert into the body of the hyoid bone. The genioglossus muscle is the main protruder of the tongue. The genial tubercles, particularly the superior pair, may be located near the crest of the alveolar ridge in Divisions C to D atrophic mandible. During the elevation of the lingual mucosa and before making an impression for a subperiosteal implant, one should be aware of the origin of this structure to avoid causing injury during the procedure. A portion of this muscle may be reflected from the genial tubercle. However, the muscle should not be completely detached from the tubercle, because this may result in retrusion of the tongue and possible airway obstruction. A branch of the hypoglossal nerve (cranial nerve XII) supplies the genioglossus.

Medial Pterygoid Muscle

The majority of the fibers of the medial pterygoid muscle take origin from the medial surface of the lateral pterygoid plate of the sphenoid bone. A small slip of muscle originates from the tuberosity of the maxilla. The muscle inserts on the medial surface of the angle of the mandible. The medial pterygoid muscle bounds the pterygomandibular space medially. This space is entered when an inferior dental nerve block is administered. Furthermore, during surgical procedures medial to the medial tendon of the temporalis muscle, such as in preparation for the insertion of unilateral subperiosteal implant, the pterygomandibular space is usually involved. Infection of this space is dangerous because of its proximity to the parapharyngeal space and the potential for spread of the infection to the mediastinum. Surgical exposure of tissue posterior to the maxillary tuberosity may also involve the medial pterygoid muscle because a portion of the muscle takes origin from the maxillary tuberosity. However, the numbers of fibers originating from the tuberosity are few in comparison with the fibers from the medial surface of the lateral pterygoid plate. A branch of the mandibular division (V3) of the trigeminal nerve innervates the muscle.

Lateral Pterygoid Muscle

Although the lateral pterygoid muscles rarely are involved in surgery for implants, their possible action in mandibular flexure or adduction during opening, as well as the effect of this phenomenon on subperiosteal implants or prosthetic full-arch splitting of mandibular implants in the molar region, warrants their consideration. The lateral pterygoid muscle consists of superior and inferior heads. The superior head takes origin from the infratemporal surface and crest of the greater wing of the sphenoid bone (roof of the infratemporal fossa), whereas the inferior head takes origin from the lateral surface of the lateral plate of the pterygoid process of the sphenoid bone. The fibers of the superior head run downward to insert on the anterior band of the temporomandibular joint (TMJ) disk (about 15% of its fibers) and the pterygoid fovea on the neck of the mandible. The fibers of the inferior head run upward to insert on the pterygoid fovea and also on the medial pole of the condyle, median capsule, and median collateral ligament of the TMJ disk. Because of the angulation of the lateral pterygoid muscles, many authors believe that the mandibular flexure causing alteration in the mandibular arch width, and sometimes pain in patients with a full-arch subperiosteal implant or prosthetic splint, may be caused by contraction of the lateral pterygoid muscles. The muscles normally function in protraction of the mandible and are innervated by a branch of the mandibular nerve (V3).

Temporalis Muscle

The temporalis is a fan-shaped muscle of mastication. It takes origin from the temporal fossa and inserts into the coronoid process of the mandible and the anterior border of the ramus as far inferiorly as the last molar at the site of the retromolar fossa. The muscle has two tendons that insert into the mandible. The superficial tendon is located laterally, and the deep tendon is inserted medially. The temporalis tendons and their associated fascia project anteromedially and inferiorly and serve as a common point for attachment for the temporalis, masseter, and medial pterygoid muscles, as well as for the transaction and superior pharyngeal constrictor muscles. The long buccal nerve and vessels are also located in this area. This temporalis tendon-fascial complex extends into what is traditionally called the *retromolar triangle*. Surgical exposure of the mandibular ramus medially would involve this tendon-fascial complex, with its contents of muscle fibers, nerves, and vessels, and may lead to transaction and postoperative pain. Incisions placed on the anterior ascending ramus for subperiosteal implants or harvesting bone from the external oblique and ramus should be inferior to the insertion of the two tendons of the temporalis muscle. The temporalis muscle is a powerful elevator and retractor of the mandible and, like all the major muscles of mastication, is innervated by a branch of V3.

Buccal or Facial Muscle Attachments

Mentalis Muscle

The external surface of the mandible in the midline presents a ridge indicative of the location of the symphysis menti (see Figure 22-5). The ridge leads inferiorly to a triangular elevation known as the *mental protuberance*. The base of the triangle is raised on either side into the mental tubercles. The mentalis muscles take origin from the periosteum of the mental tubercles and the sides of the mental eminence and insert into the skin of the chin and superiorly interdigitate with the orbicularis oris of the lower lip. Above the mentalis origin, the incisivus muscles take origin from small fossae called the *incisivus fossae*. Complete reflection of the mentalis muscles for the purpose of extension of a subperiosteal

WWW.HIGHDENT.IR

implant or symphyseal intraoral graft may result in "witch's chin," probably caused by the failure of muscle reattachment. If the muscle is completely detached to expose the symphysis, then an elastic bandage is applied externally to the chin for 4 days to help in the reattachment of the muscle. Another approach is to incise the muscle and leave a proximal portion attached to bone and reflect the distal portion. The distal and proximal portions should be approximated with resorbable sutures before suturing the mucosa. The mentalis muscle receives its nerve supply from the marginal (mandibular) branch of the facial nerve.

Buccinator Muscle

The fibers of the transaction muscle (cheek muscle) take origin from the lateral surfaces of the alveolar processes of the maxilla and the mandible in the area of the molars, the maxillary tuberosity, the pterygoid hamulus, the pterygomandibular raphe, and the retromolar fossa of the mandible. The insertion of the muscle is complex. The upper and lower fibers of the transaction blend with the fibers of the orbicularis oris at the upper and lower lips. The central fibers decussate at the modiolus before they insert into the orbicularis oris. The modiolus is the site of crossing and intermingling of fibers from the transaction muscle with fibers from the elevator and depressor muscles of the angle of the mouth. The modiolus forms a palpable node inside the angle of the mouth opposite the upper first premolar tooth. The parotid duct opposite the maxillary second molar pierces the transaction muscle. The buccopharyngeal fascia, which is a part of the visceral fascia of the neck, covers the muscle. Lateral to the fascia is the buccal pad of fat.

Some patients wearing lower subperiosteal implants complain of episodic swelling and pain at the site of origin of the transaction muscle, particularly after periods of heavy mastication or bruxism. Incision of these swellings does not usually yield exudate or purulence. The condition responds well to heat application, transactionary drugs, and rest. Although the cause for this condition is not known, one may speculate that myositis of a detached transaction muscle may cause it. The process of muscle reattachment to the implant surface or to a new site should be investigated. The buccal branch of the facial nerve innervates the muscle.

Masseter Muscle

This strong muscle of mastication covers the lateral surface of the ramus and angle of the mandible. The masseter has a dual origin from superficial and deep heads. The superficial head takes origin from the anterior two thirds of the lower border of the zygomatic arch. The deep head originates from the posterior one third of the zygomatic arch and the entire deep surface of the arch. The muscle inserts into the outer surface of the ramus of the mandible from the sigmoid notch to the angle. However, the muscle can be deflected easily during surgery to expose the bone for the ramus extension needed for lateral support of a subperiosteal implant. The space between the masseteric fascia and the muscle is a potential surgical space, known as the *masseteric space*, into which an infection may spread, causing myositis and trismus. The masseter is one of the main elevators of the jaw. The masseteric nerve provides the innervation of the muscle and is a branch of the mandibular division (V3) of the trigeminal nerve.

Innervation of the Lower Jaw and Associated Structures

Inferior Alveolar (Dental) Nerve

This nerve arises as a branch of the mandibular nerve (V3) in the infratemporal fossa (see Figure 22-6). It appears at the inferior border of the inferior head of the lateral pterygoid muscle, courses downward, and enters the mandibular foramen on the medial aspect of the ramus. Before the nerve enters the mandibular foramen, it gives numerous sensory branches that innervate the mandibular bone. These small nerves are in association with small vessels in neurovascular channels. The inferior dental nerve can run as one unit through the mandibular canal until it reaches the premolar region, where it divides into the mental and the incisive nerves. The mental nerve exits the canal through the mental foramen. In an excessively resorbed ridge, the mental foramen, with its contents of mental nerve and vessels, can be found on the crest of the ridge. When making an incision or reflection of the mucosa in this area, avoid injury to these vital structures. Knowledge of the position of the inferior dental canal in vertical and buccolingual dimensions is of paramount importance during site preparation for implants. The potential use of reconstruction techniques on computed tomographic scans and magnetic resonance imaging may increase clinicians' ability to locate the inferior dental canal precisely within the jawbone. Much less expensive techniques using panoramic cross-sectional tomographic imaging are also available. In some cases the inferior dental nerve may divide into two or three rami that occupy separate canals as the nerve travels in the mandible to supply the bone. These variations can be determined by conventional radiographic techniques, and the operator should modify the surgical approach and type of implant to avoid injury to the portion of the nerve that exits the foramen. Injury to the portion of the inferior alveolar nerve that remains in the atrophied bone and does not innervate soft tissues is of far less consequence. The nerves in the bone, when in contact with an implant, may account for the rare but occasional observation of tenderness, even though the implant is rigid and appears healthy. In addition, the fibrous tissue around these nerves may cause an increase in the amount of fibrous tissue around an implant that is inserted in contact with these structures.

Lingual Nerve

The lingual nerve is a branch of the mandibular nerve that is given off in the infratemporal fossa. It appears

WWW.HIGHDENT.IR

at the inferior border of the inferior head of the lateral pterygoid muscle anterior to the inferior alveolar nerve. It passes downward and forward between the ramus of the mandible and the medial pterygoid muscle. The nerve enters the oral cavity above the posterior edge of the mylohyoid muscle close to its origin at the third molar region. Because the nerve lies just medial to the retromolar pad, incisions in this region should remain lateral to the pad, and the mucosal reflection should be done with the periosteal elevator in constant contact with bone to prevent injury to the nerve. The nerve proceeds on the surface of the hyoglossus muscle and then crosses the duct of the submandibular gland medially to enter the floor of the mouth and the tongue. While in the infratemporal fossa, the nerve is joined by the chorda tympani nerve, which is a branch of cranial nerve VII. The chorda tympani nerve carries taste fibers from the anterior two thirds of the tongue and parasympathetic preganglionic fibers to the submandibular autonomic ganglion. The ganglion is connected to the lingual nerve on the surface of the hyoglossus muscle. The postganglionic neurons from the submandibular ganglion supply the submandibular and sublingual salivary glands. The branches of the lingual nerve in the oral cavity carry sensory information from the lingual mucosa, the mucosa of the floor of the mouth, and the anterior two thirds of the tongue. Improper reflection of a lingual mucoperiosteal flap may injure the lingual nerve and produce ipsilateral paresthesia or anesthesia of the innervated mucosa, loss of taste, and reduction of salivary secretion. The extent of involvement depends on the degree of injury to the nerve.

Nerve to the Mylohyoid

The mylohyoid motor branch of the inferior dental nerve is given off just before the nerve enters the mandibular foramen. This branch descends in a groove on the medial surface of the mandibular ramus and then appears in the submandibular triangle at the posterior border of the mylohyoid muscle. The nerve supplies the mylohyoid muscle and then proceeds on its surface with the submental artery (branch of the facial artery) until it reaches the anterior belly of the digastric muscle, which it also supplies. Because the nerve is so closely related to the ramus of the mandible, surgical intervention in this area may lead to injury of this important motor nerve.

Long Buccal Nerve

This nerve is a sensory branch of the mandibular division of the trigeminal nerve and is distributed to the skin and mucous membrane of the cheek and the buccal transact opposite the mandibular molar region. The nerve courses between the two heads of the lateral pterygoid muscle, then precedes medial to, or sometimes within, the medial temporalis tendon to gain access to the surface of the buccinator muscle. The nerve supplies the skin of the cheek and runs down to the level of the external oblique ridge, penetrates the buccinator, and spread its branches under the cheek mucosa, alveolar mucosa, and attached gingivae opposite to molar teeth. The implantologist who is planning to access the ramus for the purpose of excising a block graft should be aware of the buccal nerve and avoid injuring it. In addition, surgical manipulation in this area (e.g., during insertion of a subperiosteal implant) may injure this nerve.

BLOOD SUPPLY TO THE MAXILLA AND MANDIBLE

The head and neck region has an abundant blood supply, with many anastomoses (see Figure 22-7). The upper and lower jaws are no exception. The blood supply to both the mandible and maxilla is derived from a common source, the external carotid artery. The external carotid artery is a branch of the common carotid artery, which is a direct branch off the arch of the aorta on the left side and a branch of the brachiocephalic artery on the right side of the body.

The main artery supplying the mandible is the inferior alveolar (dental) artery, which serves as the nutrient artery to the bone and other tissues within the lower jaw. The bone tissue of the maxillae is supplied by branches of two major vessels, the posterior superior alveolar (dental) artery, and the infraorbital artery. The major branch of the infraorbital artery that supplies the maxilla is the anterior superior alveolar (dental) artery. The posterior superior alveolar and infraorbital arteries are branches of the maxillary artery, which is one of the two terminal branches of the external carotid artery.

General Concepts

The circulation of blood within long bones is centrifugal; that is, the blood circulates from the marrow (medullary) region outward through the cortical bone to end in vessels located in the periosteum and soft tissues associated with the bone.[8,9] The blood supply to the medullary region is by way of nutrient arteries, which are relatively large vessels that pass through the bone by way of nutrient canals to enter the marrow spaces. Within the marrow spaces, the nutrient artery forms a network of vessels called the *endosteal* or *medullary plexus*. Vessels from this plexus enter the cortical bone through Volkmann's canals and eventually reach the surface of the bone. While blood is passing through the cortical bone, numerous vessels are given off at right angles to these intraosseous vessels within Volkmann's canals. These branches are the vessels that are found within the haversian canals of the osteons.[8,9] Osteonal bone is the major type of bone found in the cortical bone of the jaws. Once the intraosseous vessels reach the outer surface of the bone, they anastomose with

WWW.HIGHDENT.IR

vessels within the fibrous layer of the periosteum or with arteries supplying the soft tissues. The network of vessels associated with the periosteum is called the *periosteal plexus*. The periosteal plexus in turn communicates with vessels that are supplying arterial blood to muscles and other soft tissues in the area.

The mandible and maxilla are membrane bones and as such do not develop in the same manner as long bones. Most researchers agree that the circulation of blood within the body of the mandible[10] and in the maxilla[11,12] is centrifugal under normal conditions. As in the long bones, endosteal and periosteal plexuses exist that are connected with one another.[12,13] In addition to these vascular networks, a periodontal plexus is found associated with the teeth.[12,13] When teeth are present, intraosseous vessels send branches into the alveolar processes (intraalveolar arteries), to the teeth (apical arteries), and to branches of the periodontal plexus. The intraalveolar arteries and periodontal plexus in turn connect with vessels of the periosteal plexus, as well as with vessels within the soft tissues surrounding the bone. Once a tooth is removed, its periodontal plexus is lost. When abnormal circulatory conditions exist within the mandible or maxilla, such as occlusion of the nutrient artery, the blood supply to the bone is reversed so that the direction of flow is from the outside to the inside of the bone.[10,11,14] This is called *centripetal circulation*.

Maxilla

The vessels that supply the maxilla are branches of the third part of the maxillary artery. The posterior superior alveolar artery leaves the maxillary artery and travels on the infratemporal portion of the maxilla, where it divides into several branches. Some of the branches enter alveolar canals within the posterior aspect of the maxilla to become intraosseous arteries, which supply the molar and premolar teeth and the lining of the maxillary sinus. Other branches of the posterior superior alveolar artery travel on the surface of the maxilla to supply the transact of the posterior maxillary teeth. Injury to this artery within the bone during lateral-approach sinus elevation procedures may cause hemorrhage, which requires coagulation or the use of bone wax to control the bone bleeding.

The infraorbital artery leaves the maxillary artery and enters the orbital cavity by way of the inferior orbital fissure. The artery runs in the infraorbital groove and later in the infraorbital canal. Both of these structures are located in the floor of the orbit. The infraorbital canal opens on the face as the infraorbital foramen. Within the canal the artery gives off the anterior superior alveolar artery, which descends through anterior alveolar canals to supply the maxillary anterior teeth and the mucous membrane of the maxillary sinus. The anterior and posterior superior alveolar arteries join together to form an arterial loop. The middle superior alveolar artery is rarely a separate branch.[15] The infraorbital artery also supplies branches to the maxillary sinus.[16]

Gingival, buccal, labial, palatal, nasal, and maxillary sinus blood vessels anastomose with the arterial networks associated with the maxilla. These vessels not only join with the periosteal plexus, but also penetrate the bone to connect with vessels of the endosteal and periodontal plexuses. In addition, abundant midline crossover is possible in the soft tissues of the palate and face.[13]

The mucoperiosteum of the anterior maxilla is supplied by branches of the infraorbital artery and the branches of the superior labial artery, which is a major branch of the facial artery.[13] The buccal mucoperiosteum of the maxilla is supplied by vessels of the posterior superior alveolar, anterior superior alveolar, and buccal arteries. Branches from the greater (anterior) palatine and the nasopalatine arteries supply the mucoperiosteum of the hard palate. The lesser (posterior) palatine artery supplies the soft palate. Communications of the lesser palatine arteries with the ascending pharyngeal branch of the external carotid artery and the ascending palatine branch of the facial artery are critical in many of the surgical orthognathic procedures that are performed on the maxilla.[12] In these surgical procedures, the major nutrient arteries to the maxilla are sometimes severed, but the blood supply is maintained by means of the anastomoses present in the soft palate. The vessels of the soft palate unite with vessels of the hard palate, which in turn communicate with the periosteal, periodontal, and endosteal plexuses of the upper jaw. Thus the vitality of the tissues of the maxilla is maintained through an arterial supply derived entirely from vessels that normally supply the soft palate.

Mandible

The major artery supplying the blood of the mandible is the inferior alveolar artery. The artery enters the medial aspect of the ramus of the mandible and courses downward and forward within the mandibular canal to enter the body of the mandible. The artery branches in the premolar region to give rise to two terminal branches: the mental and incisive arteries. The incisive artery continues medially within the body to anastomose with the artery of the opposite side. This artery is often severed during the harvest of a monocortical symphyseal block of bone for grafting resorbed ridges. Crushing bone around the vessel or using bone wax easily controls the bleeding. The mental artery exits the body of the mandible through the mental foramen and supplies the region of the chin and anastomoses with the submental and inferior labial arteries. Near its origin the inferior alveolar artery gives off a lingual branch, which supplies blood to the oral mucosa.[16]

Studies in animals have demonstrated that the coronoid process, the condylar process, and the angle of the mandible are supplied by arteries that provide blood

WWW.HIGHDENT.IR

to the muscles that attach to these sites.[16] Studies of human cadaver material show that the condylar process is supplied by the vascular network of the TMJ joint capsule and the lateral pterygoid muscle. In addition, researchers found that vessels from the temporalis muscle supplied the coronoid process exclusively, and the inferior alveolar artery supplied the angle of the mandible, as well as the muscles attached to the area. The same researchers found that the vessels that supply the pterygomasseteric sling (i.e., the medial pterygoid and masseter muscles) also supply the anterior portion of the ramus.[17] Empirical findings from mandibular osteotomy procedures in humans support many of these findings.[18] Thus the repositioning of the inferior alveolar artery laterally, a procedure that may be needed in some cases before implant insertion, should not eliminate the blood supply to the bone in this region (see the discussion that follows).

Changes in Blood Supply to the Mandible with Age

Although the normal circulation within the body of the mandible is centrifugal in young individuals, the direction of blood flow may reverse with aging. It has been shown that the inferior alveolar artery is susceptible to arteriosclerotic changes and tends to become tortuous and narrow with age.[19,20] Blockage of the inferior alveolar artery occurs years before any clinical evidence of blockage in the carotid vessels is found. Angiographic studies of living human subjects of all ages demonstrated blockage of the inferior alveolar artery in 79% of all individuals studied, and in 33% of the patients arterial flow was absent.[20] The incidence of absence of flow in the inferior alveolar artery increased with age. The reduction or absence in flow within the inferior alveolar artery may be associated with tooth extraction.[20] Studies in completely edentulous humans indicate that the inferior alveolar artery degenerates to such an extent as to be negligible in the supply of blood to the mandible.[19] In these cases the blood supply to the bone and internal structures was dependent on the connections with the external blood supply located within the periosteum and soft tissues associated with the mandible.[19,20] Major arteries that probably supply blood to the mandible after the interruption of the inferior alveolar artery blood flow include the mental artery,[14] the mandibular branch of the sublingual artery,[14] the facial artery,[10] and muscular branches of the maxillary artery. These anastomoses are critical in surgical procedures in which mucoperiosteal flaps are created in the mandible. The changes in pattern of blood flow to the atrophic mandible are of special importance to implant dentistry. Mucoperiosteal flap reflection for subperiosteal implant usually exposes 75% of the body of the mandible and approximately 50% of the inferior one third of the rami. Dehiscence of the mucosa at the incision lines has been reported. The reduction in atrophied bone blood supply may be a contributing factor. Onlay grafts from the iliac crest to severely atrophied mandibles are also associated with occasional incision line opening postoperatively. Limitation of surgical reflection of muscles that attach to the bone improves blood supply but may complicate primary closure without tension. However, muscle attachments at the basal bone of the mandible, which are not in the way of the graft placement, should not be reflected. In addition, endosteal implants placed in an atrophied anterior mandible may have less blood supply to the interface and may require longer time for load-bearing bone to develop. Misch has suggested 5 months of healing in very dense bone when found in an atrophied anterior mandible.[21] These speculations, of course, require experimental verification. Similar blood flow reversal with age has not been reported in the maxilla, but final comment concerning blood flow in the aged edentulous maxilla awaits further investigation.

Implantologists may encounter neurovascular bundles such as the infraorbital, incisal, greater palatine, anterior, middle, and posterior superior alveolar nerves in the maxilla (e.g., during antral augmentation procedures or mucoperiosteal flap reflection) and the mental nerve, inferior dental nerve, and the lingual nerve in the mandible (e.g., during placement of root or blade implants or reflection of microperiosteal flaps). Stretching, compression, partial resection, or total transaction can mechanically injure the nerve.

Factors that affect nerve response to mechanical injury include the following:

1. Size and number of funiculi (nerve bundles) within the nerve trunk
2. Funicular pattern: The branching within the nerve trunk will lead to an increase in density or number of nerve fibers per cross section of the nerve. Therefore injury to the nerve at one spot may cause damage to more fibers than to the adjacent area of the nerve that has less funicular branching.
3. The amount of epineural tissue: The connective tissue that surrounds the nerve is called the *epineurium*. The thinner the epineurium is, the greater the possibility is that partial injury to the nerve could damage the nerve fibers.
4. Position of the nerve fibers in the nerve trunk: The peripheral fibers leave the nerve first, whereas the central fibers innervate the most distal tissue. If a patient develops paresthesia of the lower lip after surgical placement of implants in the molar region, then it will mean that the nerve damage went through the center of the nerve to affect the mental nerve fibers.
5. Physiologic susceptibility: For an unknown reason, the motor fibers respond differently when subjected to mechanical deformation as compared with sensory fibers.

Nerve fibers of the peripheral nervous system (PNS) show greater capacity for regeneration than nerves of

WWW.HIGHDENT.IR

the central nervous system (CNS). The axons of both myelinated and unmyelinated fibers in the PNS are surrounded by Schwann cells, which are covered by basal lamina. They later provide a continuous tube, even after the nerve fiber is cut into distal and proximal segments. The proximal segment is still connected to a living nerve cell body, whereas the distal segment gradually degenerates and eventually disappears. This is known as *wallerian degeneration*. The Schwann cells of the distal segment proliferate and form a cell strand known as a *Schwann cell column* or *band of Biingner* within the basal lamina tube.[22] The Schwann cells also become phagocytic, and along with macrophages they clean the distal segment from degenerating axons. Sprouting of new axons takes place approximately 4 weeks after injury, and it takes 5 weeks for a sizeable number of axons to occupy the distal segment. The excess sprouts degenerate, and one fiber finds its way into the distal segments. If regenerating axons evade the Schwann cell column and enter the connective tissue, then they cease to grow after elongation of a few millimeters. The Schwann cells and the basal lamina are indispensable for axonal regeneration because they retain growth factors. The Schwann cells also provide new myelin for the regenerating fibers, although the conduction property is less efficient and the functional recovery may never be complete. The sprouting stage may cause pain to the patient, and the area may be sensitive to touch. The rate of recovery will depend on the type of injury (e.g., a crushed nerve regenerates faster than a severed nerve).

References

1. Sharawy M: *Companion of applied anatomy,* ed 4, Augusta, Ga, 1999, Medical College of Georgia Printing Service.
2. Hickey JC, Zarb GA, Bolender CL: *Boucher's prosthetic treatment for edentulous patients,* ed 9, St Louis, 1982, Mosby.
3. Atwood DA, Coy WA: Clinical cephalometric and densitometric study of reduction of residual ridges, *J Prosthet Dent* 26:280-299, 1977.
4. DuBrul EL: *Sicher's oral anatomy,* St Louis, 1982, Mosby.
5. Atwood DA: Some clinical factors related to rate of resorption of residual ridges, *J Prosthet Dent* 12:441-450, 1962.
6. Atwood DA: Reduction of residual ridges: a major oral disease entity, *J Prosthet Dent* 29:266-279, 1971.
7. Bays RA: The pathophysiology and anatomy of edentulous bone loss. In Fonseca RJ, Davis WH: *Reconstructive preprosthetic oral and maxillofacial surgery,* Philadelphia, 1986, WB Saunders.
8. Brookes M: *The blood supply of bone,* London, 1971, Butterworths.
9. Rhinelander FW: Circulation of bone. In Bourne GH, editor: *The biochemistry and physiology of bone,* New York, 1972, Academic Press.
10. Hellem S, Ostrup LT: Normal and retrograde blood supply to the body of the mandible in the dog. II. The role played by periosteo-medullary and symphyseal anastomoses, *Int J Oral Surg* 10:31-42, 1981.
11. Bell WH, Levy BM: Revascularization and bone healing after anterior mandibular osteotomy, *J Oral Surg* 28:196-203, 1970.
12. Bell WH: Biologic basis for maxillary osteotomies, *Am J Phys Anthropol* 38:279-290, 1973.
13. Bell WH: Revascularization and bone healing after anterior maxillary osteotomy: a study using adult rhesus monkeys, *J Oral Surg* 27:249-255, 1969.
14. Castelli WA, Nasjleti CE, Diaz-Perez R: Interruption of the arterial inferior alveolar flow and its effects on mandibular collateral circulation and dental tissues, *J Dent Res* 54:708-715, 1975.
15. Perint J: Surgical anatomy and physiology: detailed roentgenologic examination of the blood supply in the jaws and teeth by applying radiopaque solutions, *J Oral Surg* 2:2-20, 1949.
16. Williams PL, Warwick R: *Gray's anatomy,* ed 36, Philadelphia, 1980, WB Saunders.
17. Castelli W: Vascular architecture of the human adult mandible, *J Dent Res* 42:786-792, 1963.
18. Bell WH: Biologic basis for modification of the sagittal ramus split operation, *J Oral Surg* 35:362-369, 1977.
19. Bradley JC: Age changes in the vascular supply of the mandible, *Br Dent J* 132:142-144, 1972.
20. Bradley JC: A radiological investigation into the age changes of the inferior dental artery, *Br J Oral Surg* 13:82-90, 1975.
21. Misch CE: Density of bone, its effect on treatment planning, surgery, healing, and progressive bone loading, *Int J Oral Implantol* 6:23-31, 1990.
22. Ide C: Peripheral nerve regeneration, *Neurosci Res* 25: 101-121, 1996.

WWW.HIGHDENT.IR

Chapter 23

Spread of Dental Infection in the Head and Neck

Mohamed Sharawy, Carl E. Misch

SIGNIFICANT COMPLICATIONS AND IMPLANT DENTISTRY

Few significant complications are associated with implant dentistry. However, identification of potentially life-threatening problems is in the best interest of all concerned. Death in dental practice has been related to air emboli and spread of infection. These complications have a similar foundation: the vascular-lymphatic network and fascial planes. Routes associated with these potential situations are reviewed in this chapter.

The three stages in the development of infection in the head and neck region are the development, extension, and complication stages. The development stage permits the infection to spread through the bone and form an abscess under the periosteum (subperiosteal abscess). This may occur with teeth or implants. When related to an endosteal implant, it is usually associated with a device that is splinted to rigid implants or teeth in the same prosthesis. A subperiosteal implant also may be associated with this complication without the infection coming from within the bone. Most often, before subperiosteal abscess develops, purulent exudate may be found around the permucosal regions of the implant. On rare occasions, a fibrous tissue at the interface between implant and bone may become infected, and the infection may spread through the bone from a retrograde direction. For the most part, implant infection begins from the permucosal region and spreads in an apical direction. Also, an implant placed in an infected immediate extraction site may become involved in retrograde infection.

The extension stage of the development of infection occurs when the subperiosteal abscess penetrates the periosteum and extends to the fascial spaces, producing a cellulitis or fascial abscess. Mandibular subperiosteal implants most often produce this entity in the posterior region, along the posterior body of the mandible. In maxillary subperiosteal or sinus graft surgeries, this stage of infection may develop along the lateral aspect of the maxilla just below the zygomatic arch. The source of the infection is primarily from the maxillary sinus and often has an anaerobic component. On rare occasions, this extension stage has been observed in the submental region from endosteal implants placed through the inferior border of the mandible.

The complication stage results when the infection spreads and causes cavernous sinus thrombosis, brain abscess, neck and mediastinal involvement, pleurisy, or pericarditis. These complications have been reported following sinus surgeries, primarily when the antrum was infected at the time of surgery.

The principal routes for the spread of dental infection are through the following four mechanisms:

1. Bacteremia permits the infectious process to spread by way of blood.
2. Infection may spread to the walls of the veins, which may also thrombose and create a condition referred to as *thrombophlebitis*. Lack of valves in the head venous system allows retrograde flow of blood and may involve the cavernous sinus, pterygoid, and pharyngeal plexuses with infected thrombi.
3. The lymph vessels are very prevalent in the head and neck. They commonly drain the infected site and carry the infection to regional lymph nodes. The nodes become tender, enlarged, soft, and mobile on palpation. This is called *lymphadenitis*.
4. Once the infection is outside the bone, the loose areolar connective tissue produces a path of least resistance into the various surgical spaces of the head and neck and mediastinum. The muscle attachment and fascial compartments limit or direct the path of infection. Infections that may occur after surgeries involving reflection of muscle attachments may permit the infection to spread more easily into these surgical spaces. Therefore it is prudent to prophylactically cover patients with antibiotics when larger

WWW.HIGHDENT.IR

regions of soft tissues are reflected beyond muscle attachments.

Subperiosteal implants often extend beyond muscle attachments or fascial planes. Infections associated with primary struts should be closely supervised and aggressively treated because the infection may spread by continuing beyond these structures. Endosteal implants are most often positioned beyond the apices of natural teeth. As a result, infections beyond their apices may perforate bone beyond the usual limiting borders associated with the roots of teeth and may result in subcutaneous rather than submucosal infection.

PATHOLOGIC ENTITIES

The definitions of specific terms that describe infections of the head and neck provide keys to methodology of treatment and improved communications. An acute cellulitis involves diffuse inflammation of the areolar connective tissue and loose subcutaneous tissues. Lymphadenitis is a condition in which the regional lymph nodes become inflamed, enlarged, and tender. The node may become suppurated, break through the capsule, and involve the surrounding tissues. Abscess formation results when tissues break down and leukocytes die, thus forming pus. Staphylococci and streptococci are the usual bacteria involved in this process; however, a more varied population is prevalent in sinus infections. Phlegmon is any cellulitis that does not go on to suppuration. In this condition, the inflammatory infiltration of the subcutaneous tissue leads to accumulation of foul-smelling, brownish exudate. Hemolytic streptococci are usually present. A chronic cellulitis follows an acute cellulitis and may be the result of inadequate treatment or a subvirulent organism with no suppuration. A chronic abscess is a well-encapsulated entity caused by a subvirulent organism. In this case, a bone sequestrum or retained root tip is the more common source of the infection. A chronic skin fistula is a sign of retained focus of infection and, in some cases, a more serious condition of bone and bone marrow inflammation called *osteomyelitis* (Figure 23-1). This has been observed in the mandible and is associated with infection of both endosteal and subperiosteal implants in patients who have poor dental awareness and lack of implant maintenance. A noma starts as a gangrenous stomatitis and spreads to adjacent bone and muscles, causing lysis and necrosis of tissue. This rare condition perforates the cheek, floor of the mouth, or both, and is usually seen in debilitated individuals.

MAXILLARY INFECTIONS

Acute suppurative periapical abscesses in the maxilla may penetrate the alveolar bone and form a subperiosteal abscess under the periosteum. If the abscess penetrates the periosteum above the attachment of the buccinator muscle, it causes a buccal abscess.

If the abscess continues to spread, it may involve the skin, forming a cutaneous abscess. The infection may progress superiorly and involve the temporal region. Downward extension may involve the submandibular space. If the abscess perforates the buccal alveolar bone and periosteum below the attachment of the buccinator muscle, the abscess wall appears intraorally in the buccal vestibule and forms a gingival or alveolar abscess.

Palatal extension of the infection is rare. Usually it is from lingual roots of the upper molars, forming a palatal abscess. On occasion, an implant may perforate the palatal bone and cause an infection that may be evident at a later date.

The maxillary sinus may become invaded by an infection of the maxillary teeth, resulting in an acute sinusitis. Radiographs reveal a clouded sinus resulting from accumulation of inflammatory exudates within the sinus cavity. Sinus graft surgeries may penetrate an already infected antrum, or one that becomes infected shortly after surgery, with subsequent extension of the infection into the whole implanted region. The risk of sinus infection immediately following surgery is approximately 3%.

The loose, fat-containing connective tissue of the lips and cheeks is continuous and is traversed by the muscles of facial expression, which arise from the bones of the face, traverse the subcutaneous tissue, and end in the skin. These muscles, with their thin perimysium, play a role in directing the spread of infection. Dental or implant abscesses that erode and perforate the facial alveolar compact bone sometimes find their way through the subcutaneous tissue and produce remarkable swelling of the upper lip and cheek and may spread to the lower and upper eyelids due to the lack of fascial barriers on the face (Figure 23-2).

Most often, maxillary dental infections involve four regions of the maxilla: the upper lip, canine fossa, buccal space, and infratemporal space. The most common

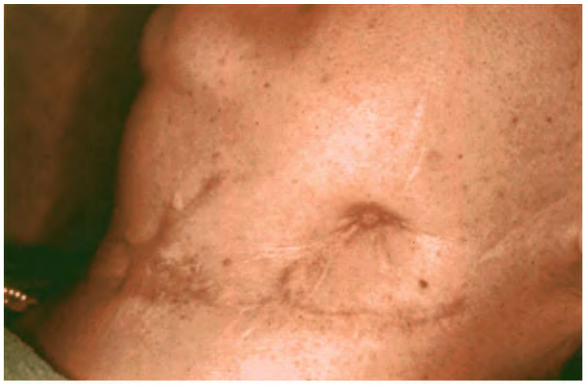


Figure 23-1 Multiple sinuses in a case of osteomyelitis of the mandible. The patient has a failing subperiosteal implant.

WWW.HIGHDENT.IR

Figure 23-2 Facial infection secondary to spread of dental infection. Once infection reaches the facial space, it can spread to the eyelids.

maxillary implant-associated infection is in the buccal space (Figure 23-3).

Lip

The lip contains weak and small muscles, but this does not limit the spread of infection. Infection of the base of the upper lip is most often caused by infection of the maxillary central region, which first produces a collateral edema and then a cellulitis of the upper lip. The abscess usually forms on the oral side of the orbicularis oris muscle in the buccal sulcus and may extend upward between the levator anguli oris and levator labii superioris. The side of the nose and lower lid of the eye may then swell. In these cases, a potential exists for the development of a cavernous sinus thrombosis by spread of infection through the angular vein to the superior ophthalmic vein to the cavernous sinus. Infected maxillary subperiosteal implants may result in infected onlay grafts and would have the potential for spreading into facial veins and the cavernous sinus.

Treatment is usually by incision and drainage. A horizontal incision is made down to the bone in the apex area of the involved tooth, implant, or piece of bone graft. The loose bone is then removed, or the tooth is treated endodontically and a rubber drain is inserted within the incision. An implant is evaluated for mobility and, if present, is usually extracted. A stable implant may remain and be evaluated after the infection is controlled.

Infection of Canine Fossa

Infection of the canine fossa is commonly caused by the spread of infection from the maxillary canine and premolars. It may also be found when sinus grafts are performed in this region. The nasolabial fold is obliterated, and edema of the eyelids results. The infection forms between the levator labii superioris and orbicularis oris anteriorly and the buccinator posteriorly. This same space can be drained intraorally by making an incision at the fluctuant swelling in the buccal sulcus and the insertion of a clamp to drain the canine space superiorly. Sinus-related infections are most often controlled with antibiotics. However, if the infection becomes localized, drainage is indicated.

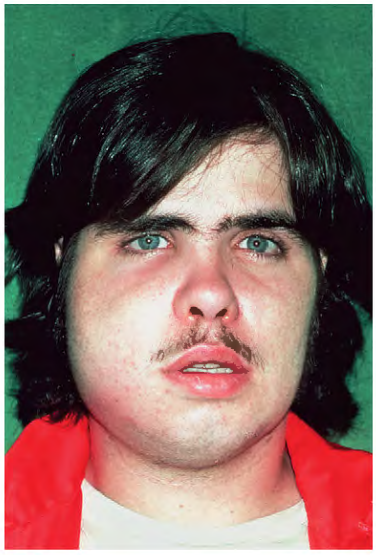


Figure 23-3 Buccal surgical space infection with spread to the submandibular space. *(From Hupp JR, Tucker MR, Ellis E:* Contemporary oral and maxillofacial surgery, *ed 5, St Louis, 2009, Mosby.)*

Infection of Buccal Space

The buccal space lies between the skin of the cheek and buccinator and contains a fat pad that extends upward and inward between the muscles of mastication, filling the retrozygomatic fossa and infratemporal space.

Abscesses of the second and third maxillary molars above the attachment of the buccinator frequently involve the buccal space. The infection may extend superiorly to the temporal space, inferiorly to the submandibular space, or posteriorly to the masticatory spaces (Figure 23-4).

Skin incision at the highest point of fluctuance, with blunt dissection to obtain adequate drainage of the space, is recommended. Anesthesia for this procedure is difficult to achieve, and a referral to an experienced practitioner is strongly suggested. A decision has to be made in individual cases as to whether the maxillary implants should be removed or conservatively treated by curettage of the infected granulation tissue with close monitoring of the progress of healing. When infections from implants extend to the infratemporal space, strong consideration should be given to their removal, rather than attempting to salvage the implants and prosthesis.

Infratemporal Space

The infratemporal space lies below a horizontal plane drawn through the zygomatic arch and is bounded laterally by the ramus of the mandible and medially

WWW.HIGHDENT.IR

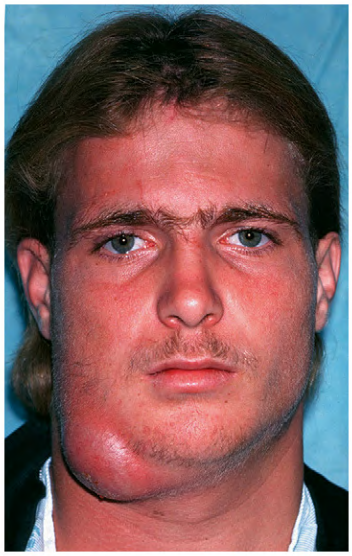


Figure 23-4 This patient had trismus and pain on swallowing. Infection spread to the submandibular, and pterygomandibular spaces. This patient needs to be admitted to the hospital for multiple incisions and drainage. *(From Hupp JR, Tucker MR, Ellis E:* Contemporary oral and maxillofacial surgery, *ed 5, St Louis, 2009, Mosby.)*

by the medial pterygoid muscle. The infratemporal space includes the pterygomandibular space between the ramus and medial pterygoid muscle, postzygomatic space, and pterygomaxillary fossa.

Infections of the maxillary teeth or endosteal implants extending above the attachment of the buccinator muscle can spread into the postzygomatic or infratemporal space. Infections of the mandibular second and third molars are more likely to infect the pterygomandibular part. Infection of this space, fortunately, is not common. It is serious because of the complications that can follow.

Infratemporal space infection may involve the pterygoid plexus of veins and subsequently the cavernous sinus. The infection may also spread through the infraorbital fissure and cause periorbital infection.

The temporal fossa, which is almost completely filled by the temporalis muscle and covered by the temporal fascia, may become involved from extension of an infection of the infratemporal space. Such involvement causes a painful swelling of the region, elevating the temporalis fascia and preventing opening of the jaw.

Incision and drainage constitute the treatment of choice. Whether an intraoral or extraoral incision is made will depend on the presence or absence of trismus, whether the intraoral approach is adequate for drainage of deep pockets of pus, and whether the maintenance of drainage from the intraoral site would constitute a health hazard from aspiration.

Incision and drainage by an intraoral approach is usually made by incising the mucosa medial and parallel to the ascending ramus along the anterior border of the medial pterygoid muscle. The surgical spaces are then approached by blunt dissection in a posteromedial direction for the pterygomandibular and parapharyngeal spaces, in an upward direction for the temporalis space, and posterolaterally into the masseteric space. Extraoral incision at the angle of the mandible is recommended for more adequate drainage of these spaces. The temporalis space may also be drained by a skin incision parallel and superior to the zygomatic arch to avoid injury to the temporal and zygomatic branches of the facial nerve.

MANDIBULAR INFECTIONS

In mandibular dental or implant infections, the mylohyoid muscle attachment on the medial surface of the mandible and the buccinator attachment on the outer surface of the mandible play an important part in limiting or directing the spread of infection.

The line of origin of the mylohyoid muscle begins close to the lower border of the mandible and ascends posteriorly and diagonally across the inner surface of the mandible to the socket of the last molar. The apical level of the roots of the incisors, canines, premolars, and first molar is always above the mylohyoid line, whereas the third molar consistently reaches below the line and the second molar does so sometimes. If a periapical abscess of the incisors, canines, and premolars perforates the lingual plate of the mandible, it will involve the floor of the mouth above the mylohyoid muscle and cause sublingual abscess or cellulitis. If the infection originates from the third molar (and sometimes the first and second molars) below the mylohyoid muscle origin, the subcutaneous connective tissue of the submandibular space is invaded with the potential for the infection to spread posteriorly and medially to the parapharyngeal space, causing a descending cervical cellulitis. A lingual perforation of the periapical abscess of the mandibular molars is an exception because of the lingual inclination of these teeth; the root tips are positioned closer to the buccal plate. Endosteal implants in the anterior and posterior regions usually extend beyond the mylohyoid muscle attachment and often are positioned more lingually than the roots of teeth. Infections may therefore penetrate the bone and appear in the submandibular space. Subperiosteal implants also extend below the muscle attachments and favor the spread of infection into the submandibular space.

Mandibular infection has the potential to spread to the submandibular space, the sublingual spaces, the parotid space, the masticatory spaces, and the parapharyngeal space. The infection may spread from the head to the neck region by following the carotid sheath, the perivisceral space, and perivertebral space—all of which communicate with the superior mediastinum. When infections extend into the sublingual space, very aggressive monitoring of the tongue size and position is needed to avoid possible obstruction of the airway. Implantologists need to be aware of these life-threatening complications, and early implant extraction may be indicated. It is often more prudent to remove

WWW.HIGHDENT.IR

the implants rather than place the patient at risk with the ailing, failing device.

Circumscribed Mandibular Abscess

Circumscribed mandibular abscess generally is caused by a mandibular premolar and is usually seen at the side of the mandible, anterior to the facial artery. It is not usually palpable under the skin and at times is closely adherent to the mandible. The abscess is usually drained by a skin incision made parallel with the lower border of the affected area of the mandible, followed by a blunt dissection with a hemostat and placement of a rubber drain. This region may also be involved with a mandibular subperiosteal implant.

Subperiosteal implants may cause swelling in the region not associated with infection. Commonly, traumatic inflammation caused by the implant casting rubbing against the buccinator muscle or bone may cause local swelling. Incision and drainage is not indicated. The condition is usually caused by hyperfunction resulting from overly aggressive eating episodes. Heat application coupled with brief avoidance of mastication on the affected side usually reduces the swelling and returns the region to normal within 1 to 3 days.

Infection of the Submandibular Space

The submandibular space is frequently involved in the spread of dental infections, osteomyelitis, and fracture of the angle of the mandible. The submandibular space is found between the mylohyoid muscle and the skin and is bound by the posterior part of the mandible laterally.

The involvement of this space is usually from infection related to deciduous or permanent molars extending through the bone below the mylohyoid muscle or descending beneath the periosteum, from a periodontal abscess or pericoronitis, or from the area around the outer aspect of the mandible. Subperiosteal or endosteal implants may be placed into or close to the sublingual space. The lingual strut of the first molar post of a subperiosteal implant should be placed above the mylohyoid attachment when possible to limit any potential infection from spreading directly into the submandibular space. Sublingual infection may also migrate on the surface of the mylohyoid muscle and descend beyond its posterior border to the submandibular space. Conversely, infection from the submandibular space may spread around the posterior part of the mylohyoid into the sublingual space to the adjacent parotid space and parapharyngeal space. When both the submandibular and sublingual spaces are involved, the floor of the mouth and tongue are elevated. In these cases, the tongue may retrude and cause difficulty in breathing.

Submandibular infection usually is drained by a skin incision 1 cm below the inferior border of the mandible, posterior to the facial artery. The areas anterior and posterior to the submandibular gland are explored by a hemostat. A rubber drain is placed.

Infection of the Masticatory Space

The masseteric space is found between the fascia and the masseter muscle (see Figure 23-4). The lateral wings of the subperiosteal implant extend along the lateral portion of the ramus. Infection of the ramus permucosal posts may extend into this region. The pterygomandibular space is found between the ramus of the mandible and the medial pterygoid muscle. The temporalis space is found between the thick temporalis fascia and the temporalis muscle.

Infection in the buccal space may spread in a posterolateral direction to involve the masseteric space or in a posteromedial direction to involve the pterygomandibular space, or it may migrate superiorly along the temporalis tendons to involve the temporalis space. Infection of the muscles will lead to myositis. The patient usually complains of trismus and pain on opening the jaw. Infection from the pterygomandibular space commonly spreads to the parapharyngeal space and may cause pain on swallowing.

Infection of the Sublingual Space

Two sublingual spaces lie above the mylohyoid on each side of the median raphe. There is no natural barrier to the spread of infection from one sublingual space to the other. The sublingual abscesses are generally derived from an infected tooth in the anterior part of the jaw. The infection emerges on the lingual side of the mandible above the mylohyoid. This will lead to swelling of the floor of the mouth, elevation of the tongue, and difficulty and pain on swallowing.

The sublingual space is drained by an incision made at the base of the alveolar process of the mandible in the lingual sulcus so that the sublingual gland, lingual nerve, and submandibular duct will not be injured. A hemostat is passed down between the sublingual gland and the geniohyoid muscle. If no pus is found, a subperiosteal abscess on the inner surface of the mandible should be investigated; if found, incision of the periosteum is made. The drain should be sutured, as it may become displaced by the tongue. The sublingual spaces can also be accessed by a skin incision in the area of the submental triangle. The mylohyoid muscles, which form the floor of the triangle, should be incised, and a hemostat can be introduced in the midline to drain the sublingual spaces (Figure 23-5).

Infection of the Submental Space

The submental space lies within the boundaries of the submental triangle, which is bound by the anterior

WWW.HIGHDENT.IR

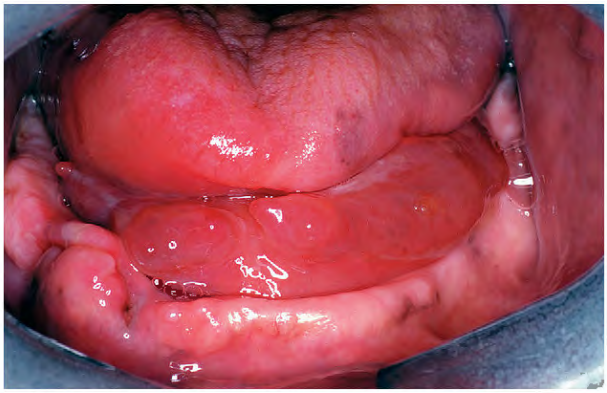


Figure 23-5 Sublingual abscess spreading into right and left surgical spaces. *(From Hupp JR, Tucker MR, Ellis E:* Contemporary oral and maxillofacial surgery, *ed 5, St Louis, 2009, Mosby.)*

bellies of digastric muscles laterally and the hyoid bone inferiorly. Pus accumulates between the skin and platysma inferiorly and the mylohyoid muscle covered by suprahyoid fascia superiorly. Posteriorly, the space extends to the submandibular space. The infection forms an abscess or cellulitis emerging below the chin.

This space is drained by transverse incision below the mandible along skin folds, and a hemostat is inserted forward and backward to open the entire area.

Ludwig's Angina

The distinct clinical entity of Ludwig's angina is characterized by a deep, tender swelling of both the submandibular and sublingual spaces, swelling of the floor of the mouth, and elevation of the tongue.

This condition is usually a nonsuppurative cellulitis that originates in the submandibular space and spreads rapidly toward the floor of the mouth. It is caused almost exclusively from mandibular molar teeth because their root apices may reach below the mylohyoid ridge. An apical infection may spread directly to the submandibular space, and from there it may extend to adjacent regions by continuity. The subperiosteal implant does not extend below this structure beyond the first molar, so the risk of infection below the muscle is reduced. The main danger from this infection is asphyxiation as a result of rapidly increasing respiratory embarrassment caused by swelling and displacement of the tongue backward and edema of the glottis. Early in the disease there is no pus, and thus no fluctuation. If pus does form, it will be found between the mylohyoid and geniohyoid or between the geniohyoid and genioglossus. This will make swallowing and speech more difficult.

Bilateral incisions of the submandibular spaces with a blunt dissection into the submental region are needed. An incision into the submental space through the mylohyoid muscle should also be made. A clamp is inserted toward the base of the tongue to explore the sublingual space. A through-and-through drain should be placed between the submandibular spaces.

Infection of the Parotid Space

The parotid space contains the parotid gland and lies between two layers of deep fascia. It is in direct communication with the parapharyngeal and submandibular spaces. The infection may start within the parotid, as in septic parotitis, or result from spread of dental infection. The condition causes pain on eating and swallowing, and there is purulent discharge from the parotid duct.

To establish drainage of the parotid space, an incision is made around and below the angle of the mandible. The space is entered with a clamp, and a drain is placed.

Infection of the Parapharyngeal Space

The parapharyngeal space extends upward between the lateral wall of the pharynx and the medial surface of the medial pterygoid muscle. Behind the medial pterygoid muscle, the parapharyngeal space widens considerably and reaches laterally to the styloid process with its muscles, and to the deep surface of the parapharyngeal space around the anterior and posterior borders of the medial pterygoid muscle. The patient usually experiences difficulty and pain during swallowing. The swelling of the tonsillar area can usually be seen intraorally. The infections may easily disseminate either upward through various foramina at the base of the skull, producing brain abscesses, meningitis, or sinus thrombosis, or downward along the carotid sheath or the visceral space toward the mediastinum.

This space is more commonly involved from acute infection around the mandibular third molar or from extension of pterygomandibular abscess, pharyngitis, tonsillitis, and parotitis.

The lateral or parapharyngeal abscess can be drained by intraoral incision of the lateral pharyngeal wall if there is no trismus or by skin incision at the anterior border of the sternocleidomastoid muscle at the level of the hyoid bone, followed by dissection along the posterior border of the posterior belly of the digastric muscle to the parapharyngeal space.

Infection of Perivisceral and Perivertebral Surgical Spaces of the Neck

Deep cervical fascias surround the pharynx, larynx, trachea, esophagus, and thyroid gland and extend through the thoracic inlet into the mediastinum. There is a potential space between the fascia and the viscera, called the *perivisceral space*. The peripharyngeal, peritracheal, and periesophageal areas are all parts of the perivisceral space. Cervical vertebrae and perivertebral muscles are similarly surrounded by a deep cervical fascia known

WWW.HIGHDENT.IR

as the *perivertebral fascia*. There is a potential space between the perivertebral fascia and the perivertebral muscles known as the *perivertebral space,* or "dangerous space." The space continues into the mediastinum. Infection in the perivisceral or perivertebral spaces commonly spreads into the thoracic mediastinum and may cause pleurisy, pericarditis, and lung abscesses. Patients who suffer from parapharyngeal space infection should be referred to a specialist for urgent incision and drainage. Those dangerous spaces can be accessed through an incision of the skin along the anterior border of sternocleidomastoid muscle below the hyoid bone, followed by retraction of the muscle and carotid sheath, blunt dissection of the spaces, and placement of a drain. A tracheostomy or cricothyroidotomy is also performed if there is respiratory obstruction caused by the spread of infection to the peritracheal space.

Infection of the Carotid Sheath

The carotid sheath is a fascial condensation around the internal jugular vein, the vagus nerve, and the common and internal carotid arteries. It lies deep to the sternocleidomastoid.

The infection could spread to the carotid sheath from the submandibular space, infratemporal space, parotid space, and parapharyngeal space. Involvement is frequently associated with thrombosis of the internal jugular vein. This condition is serious, as there is no anatomical barrier to downward spread of infection to the mediastinum. An incision is made over, and extending below, the region involved along the anterior border of the sternocleidomastoid muscle, generally above the omohyoid muscle. If the internal jugular vein is thrombosed, it is ligated below its lowest limit of involvement to prevent further descent of the infection.

SIGNIFICANT COMPLICATIONS OF INFECTIONS IN THE FACE AND NECK

Cavernous Sinus Thrombosis

Cavernous sinus thrombosis is one of the major complications of infection of the head and neck. It may occur from infection of the upper or lower jaw. The prognosis was poor before the advent of penicillin; 90% to 100% of patients died of advanced toxemia or meningitis in 5 days. The dentist should be able to recognize the early signs of cavernous sinus thrombosis and refer the patient immediately for hospital care by a specialist.

Cavernous sinus thrombosis may be caused by direct extension from the infratemporal space through the foramina or through the bone by osteomyelitis caused by staphylococcal infection or by venous thrombi originating in the venous system. One of three pathways is usually involved: the facial vein, angular vein, or superior ophthalmic vein to cavernous sinuses; the deep facial vein, pterygoid plexus of veins, and emissary veins to cavernous sinuses; or the maxillary vein (drains upper and lower jaws) to pterygoid plexus of veins to cavernous sinuses.

The clinical signs and symptoms usually include all or a combination of some of the following: edema of the eyelids; hemorrhagic skin of nose and eyelids; ophthalmoplegia caused by involvement of cranial nerves III, IV, and VI; burning and tingling sensation of the forehead caused by involvement of nerve V_1; proptosis caused by periorbital edema; or chills, fever, restlessness, and severe headache.

Brain Abscess

A brain abscess may result from bacteremia or by direct spread of infection and may occur as single or multiple abscesses, not always on the same side as the involved tooth or implant. Clinical signs consist of convulsive seizure, headache, stupor, slurred speech, and hemiparesis. Meningitis may develop, and death may occur in 2 to 6 weeks. Brain abscess has been reported after sinus surgery.

LYMPHATIC SPREAD OF INFECTION

The lymphatic circulation begins with blind-ended capillaries that drain the tissue fluid and carry it to larger vessels and eventually to two main lymphatic ducts (left thoracic and right common lymphatic ducts), which drain finally into the venous blood at the root of the neck. The lymphatic vessels are interrupted by lymph nodes. Each region of the body has a primary lymph node group that drains it. The knowledge of the regional nodes permits the diagnosis of the hitherto hidden site of infection. Although the sites of regional lymph nodes are fairly constant, there is great variability in their number and size.

Submental Lymph Nodes

The unpaired group of submental nodes lies between the hyoid bone and the anterior bellies of the digastric muscles in the submental triangle. They drain the middle part of the lower lip, the skin of the chin, and the tip of the tongue. Lymph from the lower incisors and the gingiva in this region flows at least partly into the submental nodes.

Submandibular Lymph Nodes

The submandibular lymph nodes are located in the submandibular triangle between the two bellies of the digastric muscle and the lower border of the mandible. They can be subdivided into an anterior, a middle, and

WWW.HIGHDENT.IR

a posterior group, each represented by one large or two or more smaller lymph nodes. Some of these nodes located in the submandibular niche (the space between the mylohyoid muscle and the medial surface of the mandible) are hidden by the body of the mandible and can be palpated only by pressing the finger upward on the inner surface of the lower jaw. This manipulation is facilitated with the patient's head tilted forward and toward the side being examined.

The anterior submandibular node or nodes are found along the submental vein close to the chin. The middle group is always represented by two or three small nodes situated around the anterior facial vein and facial artery, above the submandibular salivary gland. The posterior group of submandibular lymph nodes is located behind the anterior facial vein.

The submandibular lymph nodes collect the lymph of the upper and lower teeth with the exception of the incisors of the lower jaw; the lymph from the upper and lower lips with the exception of the middle part of the lower lip; the lymph from the anterior parts of the nasal cavity and palate; and finally, the lymph from the body of the tongue. Because of their relation to the teeth, the submental and submandibular lymph nodes are sometimes described as *dental lymph nodes*. The submandibular lymph nodes drain into the superficial and deep cervical lymph nodes.

Accessory lymph nodes are frequently associated with the submandibular lymph nodes and are closely related to the submandibular salivary gland. They are situated inside the capsule of the gland or even in its interlobular connective tissue (paramandibular lymph nodes).

Cervical Lymph Nodes

Cervical lymph nodes are found along the external and internal jugular veins. A subdivision into superficial and deep groups is made according to the relation of the nodes to the deep fasciae of the neck. The superficial cervical lymph nodes are, as a rule, restricted to the upper region of the neck and are found in the angle between the mandibular ramus and the sternocleidomastoid muscle. These superficial nodes receive the lymph directly from the ear lobe and the adjacent part of the skin and are secondary to the preauricular and postauricular lymph nodes.

The deep cervical lymph nodes may be subdivided into an upper and lower group; the latter sometimes are termed *supraclavicular lymph nodes.* If a continuous chain of nodes accompanies the internal jugular vein, the omohyoid muscle is taken as the arbitrary boundary between the upper and lower deep cervical lymph nodes. In addition, each of these two groups again is subdivided into an anterior (or medial) and a posterior (or lateral) group. Superior and inferior deep cervical lymph nodes, situated in front of or covered by the sternocleidomastoid muscle, are classified as anterior or medial deep cervical lymph nodes; those situated in the posterior triangle of the neck behind the sternocleidomastoid are posterior or lateral deep cervical lymph nodes.

The deep cervical lymph nodes are primary sites for the drainage of the base of the tongue, the sublingual region, and the posterior part of the palate. They are secondary and tertiary nodes into which the lymph of the auricular, submental, and submandibular and accessory nodes of the face empty, as well as the lymph from the viscera of the neck. The two lymph nodes that are normally palpable are the jugulodigastric (tonsil) lymph node and the juguloomohyoid (tongue) lymph nodes.

The superior deep cervical lymph nodes send their lymph into the inferior deep cervical or supraclavicular lymph nodes. The lymph on the right side is then collected by the right common lymphatic duct, and the left side by the thoracic duct. The two main lymphatic vessels empty on either side into the "venous angle," where the internal jugular and subclavian veins unite; thus the lymph enters the venous system by way of brachiocephalic veins.

SUMMARY

The complications from spread of dental infection are described in detail in this chapter to make the implantologist aware of the potential danger of involving the surgical spaces of the head with infection from dental origin. The early signs of infection need to be recognized and considered urgently, especially when the patient's swallowing or breathing is compromised. Also, early signs of cavernous sinus thrombosis need to be recognized and the patient referred immediately to a specialist.

Endosteal implants are usually inserted beyond the apex position of natural teeth. Subperiosteal implants traverse natural barriers of infection when extended beyond muscle attachments. Intraoral infections may extend to the base of implants, which may cause more concern than infections of natural teeth.

The implant dentist must be aware of the changes in the patient's symptoms as infection progresses and, when indicated, refer the patient immediately to a specialist for treatment. As discussed in this chapter, the infection may start as a painful swelling in the face region with little or no change in the ability of the patient to open the mouth, swallow, or breathe, and with only mild signs of toxemia. The dentist has to be extremely alert to the possibility of a progression of the infection to involve the masticatory, parapharyngeal, perivertebral, and perivisceral spaces and similar areas, with accompanying signs and symptoms, such as the inability to open the mouth and the compromise of

WWW.HIGHDENT.IR

vital signs, such as breathing. When this occurs, the patient should be hospitalized without hesitation. It is important that extraoral incisions and management of infection of the head and neck be handled by the appropriate specialists.

BIBLIOGRAPHY

Flynn TR: Anatomy of oral and maxillofacial infection. In Topazian RG, Goldberg MH, Hupp JR, editors: *Oral and maxillofacial infection*, ed 4, Philadelphia, 2002, WB Saunders.

Hollinshead WH: Fascia and fascial spaces of the head and neck. In Hollinshead WH, editor: *Anatomy for surgeons, vol 1: the head and neck*, ed 3, Philadelphia, 1982, Harper & Row.

Sharawy M: Knowledge of the lymphatics of the head and neck essential to the accurate examination of lymph nodes, *J Okla Dent Assoc* 92:48-49, 2002.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 24

Biomaterials for Dental Implants

Jack E. Lemons, Francine Misch-Dietsh

COMPATIBILITY OF SURGICAL BIOMATERIALS AND THE ROLE OF SYNTHETIC MATERIALS

The biocompatibility profiles of synthetic substances (biomaterials) used for the replacement or augmentation of biological tissues have always been a critical concern within the health care disciplines. Special circumstances are associated with dental implant prosthetic reconstruction of the oral-maxillofacial areas because the devices extend from the mouth, across the protective epithelial zones, and onto or into the underlying bone. The functional aspects of use also include the transfer of force from the occlusal surfaces of the teeth through the crown and bridge and neck-connector region of the implant into the implant for interfacial transfer to the supporting soft and hard tissues. This situation represents a very complex series of chemical and mechanical environmental conditions.

This most critical aspect of biocompatibility is, of course, dependent on the basic bulk and surface properties of the biomaterial. All aspects of basic manufacturing, finishing, packaging and delivering, sterilizing, and placing (including surgical placement) must be adequately controlled to ensure clean and nontraumatizing conditions. The importance of these considerations has been reemphasized through the concept and practice of osteointegration of endosteal root form implant systems.

The disciplines of biomaterials and biomechanics are complementary to the understanding of device-based function. The physical, mechanical, chemical, and electrical properties of the basic material components must always be fully evaluated for any biomaterial application, because these properties provide key inputs into the interrelated biomechanical and biological analyses of function. It is important to separate the roles of macroscopic implant shape from the microscopic transfer of stress and strain along biomaterial-tissue interfaces. The macroscopic distribution of mechanical stress and strain is predominantly controlled by the shape and form of the implant device. One important material property related to design (shape and form) optimization is the elastic strain (one component of the elastic modulus) of the material.

The localized microscopic strain distribution is controlled more by the basic properties of the biomaterial (e.g., surface chemistry, microtopography, modulus of elasticity) and by whether the biomaterial surface is attached to the adjacent tissues. Engineering analyses of implant systems include optimization considerations related both to the design and to the biomaterial used for construction. Therefore the desire to positively influence tissue responses and to minimize biodegradation often places restrictions on which materials can be safely used within the oral and tissue environments. Designs are often evolved for specific biomaterials because of the imposed environmental or restorative conditions.

BULK PROPERTIES

History of Materials and Designs

Over the past several decades, definitions of material biocompatibilities have evolved and reflect an ever-changing opinion related to philosophies of surgical implant treatment. In general, the definition of biocompatibility has been given as an appropriate response to a material (biomaterial) within a device (design) for a specific clinical application.[1] Metallic and nonmetallic implantable materials have been studied in the field of orthopedics since the turn of the twentieth century.[2-7] In the 1960s, emphasis was placed on making the biomaterials more inert and chemically stable within biological environments. The high-purity ceramics of aluminum oxide (Al_2O_3), carbon, and carbon-silicon compounds and extra-low-interstitial (ELI) grade alloys are classic examples of these trends. In the 1970s, biocompatibility was defined in terms of minimal harm to the host or to the biomaterial. The importance of a stable interaction then moved into central focus for both the research and the clinical communities. In the 1980s the focus transferred to bioactive substrates intended to positively influence tissue responses,

WWW.HIGHDENT.IR

whereas in the last two decades, emphasis on chemically and mechanically anisotropic substrates combined with growth (mitogenic) and inductive (morphogenic) substances. Today many biomaterials are being constituted, fabricated, and surface modified to directly influence short- and long-term tissue responses. Bioactive coatings on most classes of biomaterials have continued to evolve from human clinical trials to acceptable modalities of surface preparation, and research focus has shifted to combinations of active synthetic and biological implants.

Of interest, dental implants have significantly influenced these trends. In the 1960s, dental devices were recognized as being in a research and development phase, and critical longitudinal reviews of clinical applications were strongly recommended.[8] During this time, longevity studies of various devices demonstrated that the longest duration of clinical applications were for orthopedic prostheses. In the 1980s, controlled clinical trials showed that dental implants provided functional longevities that exceeded most other types of functional tissue replacement modalities.[9,10] Clearly, these clinical studies have strongly influenced both the research and development and the clinical application processes. At the present time, the exponential growth of implant use and related scientific reports support the views expressed by early visionaries several decades ago.

The evolution of any implant modality is a multipart story in which significant roles have been played by biomaterials; biomechanical analyses of designs, tissues, and function; wound healing along interfaces; surgical methods to minimize mechanical, chemical, and thermal trauma; prosthodontic and periodontal restorative and maintenance treatment modalities; and protocols for controlled multidisciplinary clinical trials. The interdependence of all phases of basic and applied research should be recognized. All interrelate and must evolve to provide a level of better understanding of the basic physical and biological phenomena associated with the implant systems before the longer clinical outcomes will be fully described.

Evaluations of endosteal and subperiosteal dental implants raise interesting questions with respect to the interrelationships between material and design selection. Opportunities exist to select a material from a number of systems, such as metals, ceramics, carbons, polymers, or composites. In addition, only the available anatomical dimensions and the requirement to attach some form of intraoral restorative device limit implant shape and form (design). Because of the wide range of biomaterial properties demonstrated by the classes of materials available, it is not advisable to fabricate any new implant design without a thorough biomechanical analysis. Another approach now often used is to determine a specific design based on clinical considerations and then to select the biomaterial of choice from computer-based analyses. The safety of these combinations can then be demonstrated through laboratory and animal investigations. Controlled clinical trials following prospective protocols, of course, provide the final evaluation for both safety and effectiveness. Long-term success is thus determined clinically in investigator follow-up studies and is clearly an area that should be emphasized for many available dental implant systems.

Research and Development

Basic studies within the physical and biological sciences have been supportive of the development of surgical implant systems. One example is the continued progress from materials that have been available for industrial applications to the new classes of composites that have evolved for biomedical applications. This same situation exists within a broad area; for example, surface science and technology, mechanics and biomechanics of three-dimensional structures, pathways and processes of wound healing along biomaterial interfaces, and the description of the first biofilms that evolve on contact with blood or tissue fluids.[11-14] The progressive move from materials to quantitatively characterized biomaterials has been extremely important to the biomedical applications of surgical implants. Dental implant investigations now play a leadership role within selected areas of this overall process, and all phases of medicine and dentistry should benefit.

PHYSICAL, MECHANICAL, AND CHEMICAL REQUIREMENTS FOR IMPLANT MATERIALS

Physical and Mechanical Properties

Forces exerted on the implant material consist of tensile, compressive, and shear components. As for most materials, compressive strengths of implant materials are usually greater than their shear and tensile counterparts. A hypothesis that dental implants are less affected by alternating stresses than implants of the cardiovascular and locomotor systems because of the significantly lower number of loading cycles must be qualified because of the special concern that dental implants are considerably smaller in physical dimension. All fatigue failures obey mechanical laws correlating the dimensions of the material to the mechanical properties of said material.[11,15] In addition, when present, parafunction (nocturnal and/or diurnal) can be greatly detrimental to longevity because of the mechanical properties, such as maximum yield strength, fatigue strength, creep deformability, ductility, and fracture. Limitations of the relevance of these properties are mainly caused by the variable shape and surface features of implant designs. A recurring problem exists between the mechanical strength and deformability of the material and the recipient bone. A different approach to match more closely the implanted material and hard tissue properties led to the

WWW.HIGHDENT.IR

انجمن علمی کارشناسان دندانسازان و دندانپزشکان

experimentation of polymeric, carbonitic, and metallic materials of low modulus of elasticity.[16,17]

Because bone can modify its structure in response to forces exerted on it, implant materials and designs must be designed to account for the increased performance of the musculature and bone in jaws restored with implants. The upper stress limit decreases with an increasing number of loading cycles sometimes reaching the fatigue limit after 10^6 to 10^7 loading cycles.[11,15,18] In other words, the higher the applied load, the higher the mechanical stress—and therefore the greater the possibility for exceeding the fatigue endurance limit of the material.

In general, the fatigue limit of metallic implant materials reaches approximately 50% of their ultimate tensile strength.[11,18] However, this relationship is only applicable to metallic systems, and polymeric systems have no lower limit in terms of endurance fatigue strength. Ceramic materials are weak under shear forces because of the combination of fracture strength and no ductility, which can lead to brittle fracture. Metals can be heated for varying periods to influence properties, modified by the addition of alloying elements or altered by mechanical processing such as drawing, swagging, or forging, followed by age or dispersion hardening, until the strength and ductility of the processed material are optimized for the intended application.

The modifying elements in metallic systems may be metals or nonmetals. A general rule is that constitution or mechanical process hardening procedures result in an increased strength but also invariably correspond to a loss of ductility. This is especially relevant for dental implants. Most all consensus standards for metals (American Society for Testing and Material [ASTM], International Standardization Organization [ISO], American Dental Association [ADA]) require a minimum of 8% ductility to minimize brittle fractures. Mixed microstructural phase hardening of austenitic materials with nitrogen (e.g., stainless steels) and the increasing purity of the alloys seem most indicated to achieve maximum strength and maintain this high level of possible plastic deformation.[1,15,19-23]

Corrosion and Biodegradation

Corrosion is a special concern for metallic materials in dental implantology, because implants protrude into the oral cavity where electrolyte and oxygen compositions differ from that of tissue fluids. In addition, the pH can vary significantly in areas below plaque and within the oral cavity. This increases the range of pH that implants are exposed to in the oral cavity compared with specific sites in tissue.[24-29]

Plenk and Zitter[15] state that galvanic corrosion (GC) could be greater for dental implants than for orthopedic implants. Galvanic processes depend on the passivity of oxide layers, which are characterized by a minimal dissolution rate and high regenerative power for metals such as titanium. The passive layer is only a few nanometers thick and usually composed of oxides or hydroxides of the metallic elements that have greatest affinity for oxygen. In reactive group metals such as titanium, niobium, zirconium, tantalum, and related alloys, the base materials determine the properties of the passive layer. The stability zones of the oxides of passivable elements cover the redox potentials and pH values typical of the oral environment. However, titanium, tantalum, and niobium oxides cover a markedly larger zone of environmental stability compared with chromium oxides.

The risk of mechanical degradation, such as scratching or fretting of implanted materials, combined with corrosion and release into bone and remote organs has been previously considered. For example, investigators such as Laing,[30] Willert et al.,[31] and Lemons,[32,33] have extensively studied the corrosion of metallic implants. Steinemann[34] and Fontana and Greene[35] have presented many of the basic relationships specific to implant corrosion. Mears[26] addressed concerns about GC and studied the local tissue response to stainless steel and cobalt-chromium-molybdenum (Co-Cr-Mo) and showed the release of metal ions in the tissues. Williams[36] suggested that three types of corrosion were most relevant to dental implants: (1) stress corrosion cracking (SCC), (2) GC, and (3) fretting corrosion (FC).

Stress Corrosion Cracking

The combination of high magnitudes of applied mechanical stress plus simultaneous exposure to a corrosive environment can result in the failure of metallic materials by cracking, where neither condition alone would cause the failure. Williams[36] presented this phenomenon of SCC in multicomponent orthopedic implants. Others hypothesized that it may be responsible for some implant failures in view of high concentrations of forces in the area of the abutment-implant body interface.[37-39] Most traditional implant body designs under three-dimensional finite element stress analysis show a concentration of stresses at the crest of the bone support and cervical third of the implant. This tends to support potential SCC at the implant interface area (i.e., a transition zone for altered chemical and mechanical environmental conditions). This has also been described in terms of corrosion fatigue (i.e., cyclical load cycle failures accelerated by locally aggressive medium). In addition, nonpassive prosthetic superstructures may incorporate permanent stress, which strongly influences this phenomenon under loaded prostheses[37,40,41] (Figure 24-1, *A* and *B*).

Galvanic corrosion occurs when two dissimilar metallic materials are in contact and are within an electrolyte resulting in current flowing between the two. The metallic materials with the dissimilar potentials can have their corrosion currents altered, thereby resulting

WWW.HIGHDENT.IR

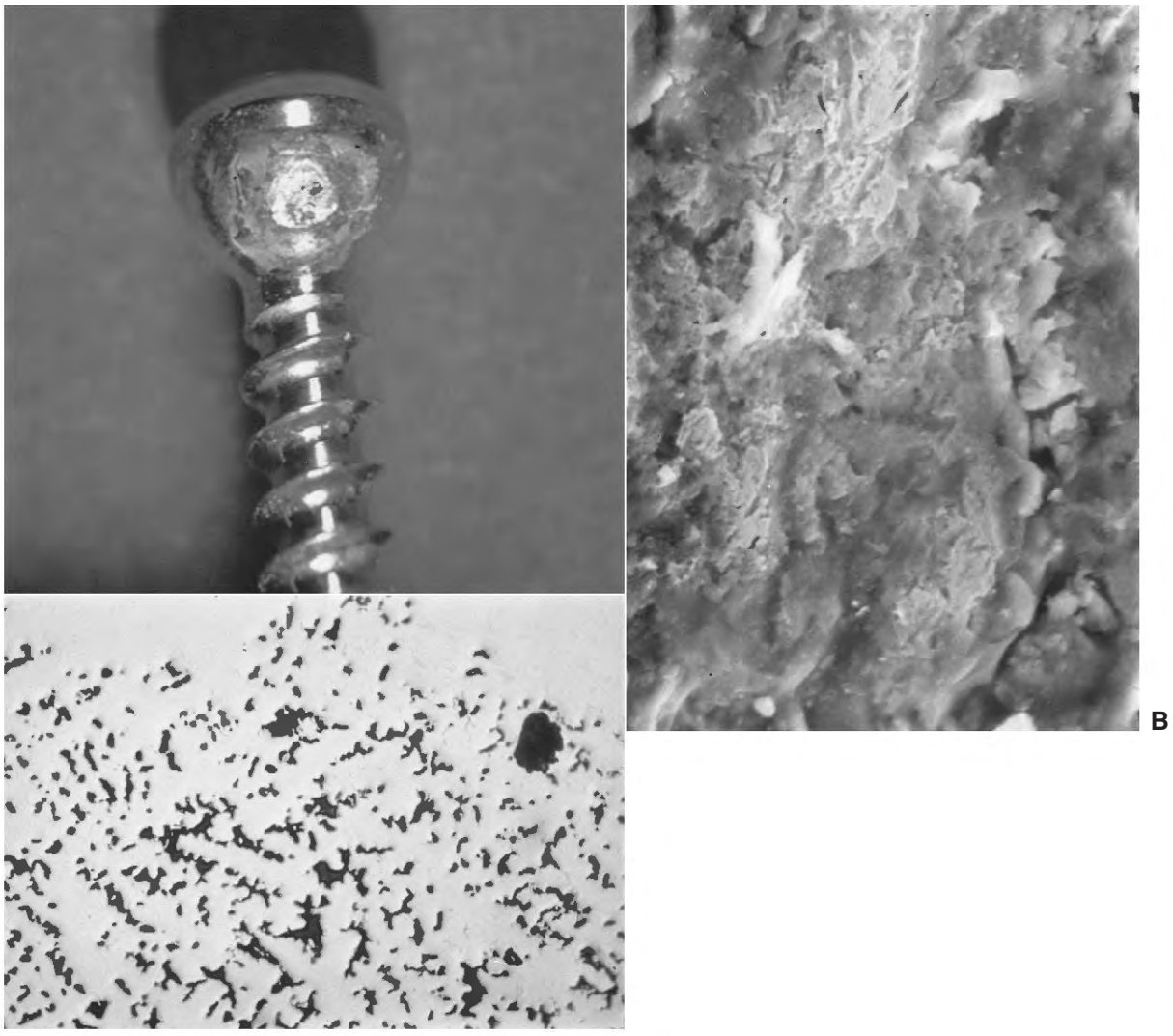


Figure 24-1 **A,** Stainless steel (316L) fracture fixation screw showing crevice corrosion after 1 year in vivo (approximately ×5). **B,** Microscopic characteristics of cobalt alloy root form surface showing environmental degradation (approximately ×100). **C,** As-polished microstructure of cobalt alloy subperiosteal showing porosity associated with galvanically assisted corrosion (approximately ×100).

in a greater corrosion rate (Figure 24-1, *C*). Fretting corrosion occurs when a micromotion and rubbing contact occurs within a corrosive environment (e.g., the perforation of the passive layers and shear-directed loading along adjacent contacting surfaces). The loss of any protective film can result in the acceleration of metallic ion loss. Fretting corrosion has been shown to occur along implant body-abutment-superstructure interfaces.

Normally the passive oxide layers on metallic substrates dissolve at such slower rates that the resultant loss of mass is of no mechanical consequence to the implant. A more critical problem is the irreversible local perforation of the passive layer that chloride ions often cause, which may result in localized pitting corrosion. Such perforations can often be observed for iron-chromium-nickel-molybdenum (Fe-Cr-Ni-Mo) steels that contain an insufficient amount of the alloying elements stabilizing the passive layer (i.e., Cr and Mo) or local regions of implants that are subjected to abnormal environments. Even ceramic oxide materials are not fully degradation resistant. Corrosion-like behavior of ceramic materials can then be compared with the chemical dissolution of the oxides into ions or complex ions of respective metallic oxide substrates. An example of this is the solubility of aluminum oxide as alumina or titanium oxide as titania. This statement is generally valid; however, most metallic oxides and nonmetallic substrates have amorphous-hydroxide inclusive structures, whereas bulk ceramics are mostly crystalline. The corrosion resistance of synthetic polymers, on the other hand, depends not only on their composition and structural form but also on the degree of polymerization. Unlike metallic and ceramic materials, synthetic polymers are not only dissolved but also penetrated by water and substances from biological

WWW.HIGHDENT.IR

environments. The resulting degree of alteration depends on the material property conditions for the manufactured component.

Toxicity and Consideration

Toxicity is related to primary biodegradation products (simple and complex cations and anions), particularly those of higher atomic weight metals. Factors to be considered include (1) the amount dissolved by biodegradation per time unit, (2) the amount of material removed by metabolic activity in the same time unit, and (3) the quantities of solid particles and ions deposited in the tissue and any associated transfers to the systemic system. For example, the quantity of elements released from metals during corrosion time (e.g., grams per day) can be calculated by using the following formula[15]:

$$\text{TE (g/day)} = \frac{\text{TEA(\%)} \times \text{CBR(g/cm}^2 \times \text{day)} \times \text{IS(cm}^2\text{)}}{100}$$

where *TE* = toxic element, *TEA* = toxic elements in alloy, *CBR* = corrosion biodegradation, and *IS* = implant surface.

It is of little importance for the formula whether or not the metallic substrate is exposed because the passive layer is dissolved. The critical issue is that the surface represents the "finished" form of the implant. The formula is also valid for ceramic materials and for substances transferred from synthetic polymers. Therefore it appears that the toxicity is related to the content of the materials' toxic elements and that they may have a modifying effect on corrosion rate.[15]

The transformation of harmful primary products is dependent on their level of solubility and transfer. It is known that chromium and titanium ions react locally at low concentrations, whereas cobalt, molybdenum, or nickle can remain dissolved at higher relative concentrations and thus may be transported and circulated in body fluids. Several studies have documented the relative toxicity of titanium and its alloys and are addressed within the section on titanium.

Lemons[32] reported on the formation of electrochemical couples as a result of oral implant and restorative procedures and stressed the importance of selecting compatible metals to be placed in direct contact with one another in the oral cavity to avoid the formation of adverse electrochemical couples. The electrochemical behavior of implanted materials has been instrumental in assessing their biocompatibility.[42] Zitter and Plenk[43] have shown that anodic oxidation and cathodic reduction take place in different spaces but must always balance each other through charge transfer. This has been shown to impair both cell growth and transmission of stimuli from one cell to another. Therefore an anodic corrosion site can be influenced by ion transfer but also by other possibly detrimental oxidation phenomena. Charge transfer appears to be a significant factor specific to the biocompatibility of metallic biomaterials. Passive layers along the surfaces of titanium, niobium, zirconium, and tantalum increase resistance to change transfer processes by isolating the substrate from the electrolyte, in addition to providing a higher resistance to ion transfers. On the other hand, metals based on iron, nickel, or cobalt are not as resistant to transfers through the oxidelike passive surface zones.

METALS AND ALLOYS

To date, most of the dental implant systems available within the United States are constructed from metals or alloys. These materials are reviewed in this chapter by separating the metals and alloys according to their elemental compositions, because a growing proportion have modified surface characteristics that are addressed in the second section of this chapter.

Several organizations have provided guidelines for the standardization of implant materials.[44] ASTM Committee F4 (ASTM F4) and ISO (ISOTC 106, ISOTR 10541) have provided the basis for such standards.[19,20] To date, a multinational survey by ISO indicated that titanium and its alloy are mainly used. The most widely used nonmetallic implants are oxidic, carbonitic, or graphitic oxidelike materials.[45]

The major groups of implantable materials for dentistry are titanium and alloys, cobalt chromium alloys, austenitic Fe-Cr-Ni-Mo steels, tantalum, niobium and zirconium alloys, precious metals, ceramics, and polymeric materials.

Titanium and Titanium–6 Aluminum–4 Vanadium (Ti-6Al-4V)

This reactive group of metals and alloys (with primary elements from reactive group metallic substances) form tenacious oxides in air or oxygenated solutions. Titanium oxidizes (passivates) on contact with room temperature air and normal tissue fluids. This reactivity is favorable for dental implant devices. In the absence of interfacial motion or adverse environmental conditions, this passivated (oxidized) surface condition minimizes biocorrosion phenomena. In situations in which the implant would be placed within a closely fitting receptor site in bone, areas scratched or abraded during placement would repassivate in vivo. This characteristic is one important property consideration related to the use of titanium for dental implants.[37,46-48] Some reports show that the oxide layer tends to increase in thickness under corrosion testing,[48] and that breakdown of this layer is unlikely in aerated solutions.[49]

Bothe et al.[50] studied the reaction of rabbit bone to 54 different implanted metals and alloys and showed that titanium allowed bone growth directly adjacent to the oxide surfaces. Leventhal[51] further studied the

WWW.HIGHDENT.IR

Table 24-1 Engineering Properties of Metals and Alloys Used for Surgical Implants*

MATERIAL	NOMINAL ANALYSIS (w/o)	MODULUS OF ELASTICITY GN/m^2 (psi × 10^6)	ULTIMATE TENSILE STRENGTH MN/m^2 (ksi)	ELONGATION TO FRACTURE (%)	SURFACE
Titanium	99+Ti	97 (14)	240-550 (25-70)	>15	Ti oxide
Titanium-aluminum-vanadium	90Ti-6Al-4V	117 (17)	869-896 (125-130)	>12	Ti oxide
Cobalt-chromium-molybdenum (casting)	66Co-27Cr-7Mo	235 (34)	655 (95)	>8	Cr oxide
Stainless steel (316L)	70Fe-18Cr-12Ni	193 (28)	480-1000 (70-145)	>30	Cr oxide
Zirconium	99^+Zr	97 (14)	552 (80)	20	Zr oxide
Tantalum	99^+Ta	—	690 (100)	11	Ta oxide
Gold	99^+Au	97 (14)	207-310 (30-45)	>30	Au
Platinum	99^+Pt	166 (24)	131 (19)	40	Pt

*Minimum values from the American Society for Testing and Materials Committee F4 documents are provided. Selected products provide a range of properties.
GN/m^2, Giganewton per meter squared; *ksi*, thousand pounds per inch squared; *MN/m^2*, meganewton per meter squared; *psi*, pounds per inch squared; *w/o*, weight percent.

application of titanium for implantation. Beder et al.,[52] Gross et al.,[53] Clarke et al.,[54] and Brettle[55] were able to expand indications of these materials. In all cases, titanium was selected as the material of choice because of its inert and biocompatible nature paired with excellent resistance to corrosion.[1,56-60]

Specific studies in the literature addressed the corrosion of titanium implants and are reported in the surface characteristics section. Unfortunately most are for in vitro and unloaded conditions, and few identify precisely the type of titanium and titanium surface studied.

The general engineering properties of the metals and alloys used for dental implants are summarized in Table 24-1. Titanium shows a relatively low modulus of elasticity and tensile strength when compared with most other alloys. The strength values for the wrought soft and ductile metallurgic condition (normal root forms and plate form implants) are approximately 1.5 times greater than the strength of compact bone. In most designs in which the bulk dimensions and shapes are simple, strength of this magnitude is adequate. Because fatigue strengths are normally 50% weaker or less than the corresponding tensile strengths, implant design criteria are decidedly important. The creation of sharp corners or thin sections must be avoided for regions loaded under tension or shear conditions. The modulus of elasticity of titanium is five times greater than that of compact bone, and this property places emphasis on the importance of design in the proper distribution of mechanical stress transfer. In this regard, surface areas that are loaded in compression have been maximized for some of the newer implant designs. Four grades of unalloyed titanium and titanium alloy are the most popular. Their ultimate strength and endurance limit vary as a function of their composition.

The alloy of titanium most often used is titanium-aluminum-vanadium. The wrought alloy condition is approximately six times stronger than compact bone and thereby affords more opportunities for designs with thinner sections (e.g., plateaus, thin interconnecting regions, implant-to-abutment connection screw housing, irregular scaffolds, porosities). The modulus of elasticity of the alloy is slightly greater than that of titanium, being about 5.6 times that of compact bone. The alloy and the primary element (i.e., titanium) both have titanium oxide (passivated) surfaces. Information has been developed on the oxide thickness, purity, and stability as related to implant biocompatibilities.[9,14,19] In general, titanium and alloys of titanium have demonstrated interfaces described as *osteointegrated* for implants in humans. In addition, surface conditions in which the oxide thickness has varied from hundreds of angstroms of amorphous oxide surface films to 100% titania (titanium dioxide [TiO_2] rutile form ceramic) have demonstrated osteointegration.

The possible influences of aluminum and vanadium biodegradation products on local and systemic tissue responses have been reviewed from the perspectives of basic science and clinical applications.[61] Extensive literature has been published on the corrosion rate of titanium within local tissue fluids[62-64] and the peri-implant accumulation of "black particles."[65] A few adverse effects have been reported.[66] Increased titanium concentrations were found in both peri-implant tissues and parenchymal organs,[67,68] mainly the lung and much lesser concentrations in the liver, kidney, and

WWW.HIGHDENT.IR

spleen.[25,66-70] However, alloy compositions were not well defined or controlled. Corrosion and mechanical wear have been suggested as possible causes.[48,67,68] Authors who still caution about the applicability of these results to the presently available titanium alloys have developed other alloys using iron, molybdenum, and other elements as primary alloying agents.[17] More recently, several new titanium alloys of higher strength have been introduced.[33,71]

Although many basic science questions remain, clinical applications of these alloys in dental and orthopedic surgical systems have been very positive, especially in light of improved strength, and the titanium alloys have not demonstrated significant numbers of identifiable negative sequelae.[19]

Electrochemical studies support the selection of conditions in which elemental concentrations would be relatively low in magnitude.[11] Electrochemically, titanium and titanium alloy are slightly different in regard to electromotive and galvanic potentials when compared with other electrically conductive dental materials. Results of these electrochemical potentials and how they relate to in vivo responses have been published previously.[9,42,63] In general, titanium- and cobalt-based systems are electrochemically similar; however, comparative elements imitating the conditions in an aeration cell revealed that the current flow in titanium and titanium alloys is several orders of magnitude lower than that in Fe-Cr-Ni-Mo steels or Co-Cr alloys.[15] Gold-, platinum-, and palladium-based systems have been shown to be noble, and nickel-, iron-, copper, and silver-based systems are significantly different (subject to galvanic coupling and preferential in vivo corrosion).

Mechanically, titanium is much more ductile (bendable) than titanium alloy. This feature has been a very favorable aspect related to the use of titanium for endosteal plate form devices. The need for adjustment or bending to provide parallel abutments for prosthetic treatments has caused manufacturers to optimize microstructures and residual strain conditions. Coining, stamping, or forging followed by controlled annealing heat treatments are routinely used during metallurgic processing. However, if an implant abutment is bent at the time of implantation, then the metal is strained locally at the neck region (bent), and the local strain is both cumulative and dependent on the total amount of deformation introduced during the procedure. This is one reason, other than prior loading fatigue cycling, why reuse of implants is not recommended. In addition, mechanical processes can sometimes significantly alter or contaminate implant surfaces. Any residues of surface changes must be removed before implantation to ensure mechanically and chemically clean conditions.

The emerging techniques to cast titanium and titanium alloys remain limited for dental implant application because of high melting points of the elements and propensity for absorption of oxygen, nitrogen, and hydrogen, which may cause metallic embrittlement. A high vacuum or ultrapure protective gas atmosphere allows the production of castings in titanium and its alloys at different purity levels,[72,73] although microstructures and porosity are relatively unfavorable related to fatigue and fracture strengths.[9,32] Typical strengths of cast commercially pure (CP) titanium grade 2 and Ti-6Al-4V after heat treatment and annealing can be in the range of those of wrought titanium alloys used for dental implants.[74]

Cobalt-Chromium-Molybdenum–Based Alloy

The cobalt-based alloys are most often used in an as-cast or cast-and-annealed metallurgic condition. This permits the fabrication of implants as custom designs such as subperiosteal frames. The elemental composition of this alloy includes cobalt, chromium, and molybdenum as the major elements. Cobalt provides the continuous phase for basic properties; secondary phases based on cobalt, chromium, molybdenum, nickel, and carbon provide strength (four times that of compact bone) and surface abrasion resistance (see Table 24-1); chromium provides corrosion resistance through the oxide surface; and molybdenum provides strength and bulk corrosion resistance. All of these elements are critical, as is their concentration, which emphasizes the importance of controlled casting and fabrication technologies. Also included in this alloy are minor concentrations of nickel, manganese, and carbon. Nickel has been identified in biocorrosion products, and carbon must be precisely controlled to maintain mechanical properties such as ductility. Surgical alloys of cobalt are not the same as those used for partial dentures, and substitutions should be avoided.

In general, the as-cast cobalt alloys are the least ductile of the alloy systems used for dental surgical implants, and bending of finished implants should be avoided. Because many of these alloy devices have been fabricated by dental laboratories, all aspects of quality control and analysis for surgical implants must be followed during alloy selection, casting, and finishing. Critical considerations include the chemical analysis, mechanical properties, and surface finish as specified by the ASTM F4 on surgical implants and the ADA.[19,21] When properly fabricated, implants from this alloy group have shown to exhibit excellent biocompatibility profiles.

Iron-Chromium-Nickel–Based Alloys

The surgical stainless steel alloys (e.g., 316 low carbon [316L]) have a long history of use for orthopedic and dental implant devices. This alloy, as with titanium systems, is used most often in a wrought and heat-treated metallurgic condition, which results in a high-strength and high-ductility alloy. The ramus blade, ramus frame,

WWW.HIGHDENT.IR

stabilizer pins (old), and some mucosal insert systems have been made from the iron-based alloy.

The ASTM F4 specification for surface passivation was first written and applied to the stainless steel alloys.[19] In part, this was done to maximize corrosion-biocorrosion resistance. Of the implant alloys, this alloy is most subject to crevice and pitting biocorrosion, and care must be taken to use and retain the passivated (oxide) surface condition. Because this alloy contains nickel as a major element, use in patients allergic or hypersensitive to nickel should be avoided. In addition, if a stainless steel implant is modified before surgery, then recommended procedures call for repassivation to obtain an oxidized (passivated) surface condition to minimize in vivo biodegradation.

The iron-based alloys have galvanic potentials and corrosion characteristics that could result in concerns about galvanic coupling and biocorrosion if interconnected with titanium, cobalt, zirconium, or carbon implant biomaterials.[75-77] In some clinical conditions, more than one alloy may be present within the same dental arch of a patient. For example, if a bridge of a noble or a base-metal alloy touches the abutment heads of a stainless steel and titanium implant simultaneously, then an electrical circuit would be formed through the tissues. If used independently, where the alloys are not in contact or not electrically interconnected, then the galvanic couple would not exist, and each device could function independently. As with the other metal and alloy systems discussed, the iron-based alloys have a long history of clinical applications. Long-term device retrievals have demonstrated that, when used properly, the alloy can function without significant in vivo breakdown. Clearly, the mechanical properties and cost characteristics of this alloy offer advantages with respect to clinical applications.

Other Metals and Alloys

Many other metals and alloys have been used for dental implant device fabrication. Early spirals and cages included tantalum, platinum, iridium, gold, palladium, and alloys of these metals. More recently, devices made from zirconium, hafnium, and tungsten have been evaluated.[15,78,79] Some significant advantages of these reactive group metals and their alloys have been reported, although large numbers of such devices have not been fabricated in the United States.

Gold, platinum, and palladium are metals of relatively low strength, which places limits on implant design. In addition, cost-per-unit weight and the weight-per-unit volume (density) of the device along the upper arch have been suggested as possible limitations for gold and platinum. These metals, especially gold because of nobility and availability, continue to be used as surgical implant materials. For example, the Bosker endosteal staple design represents use of this alloy system.[80]

CERAMICS AND CARBON

Ceramics are inorganic, nonmetallic, nonpolymetric materials manufactured by compacting and sintering at elevated temperatures. They can be divided into metallic oxides or other compounds. Oxide ceramics were introduced for surgical implant devices because of their inertness to biodegradation, high strength, physical characteristics such as color and minimal thermal and electrical conductivity, and a wide range of material specific elastic properties.[81,82] In many cases, however, the low ductility or inherent brittleness has resulted in limitations. Ceramics have been used in bulk forms and more recently as coatings on metals and alloys.

Aluminum, Titanium, and Zirconium Oxides

High-strength ceramics from aluminum, titanium, and zirconium oxides have been used for root form, endosteal plate form, and pin type of dental implants.[83] The overall characteristics of these ceramics are summarized in Table 24-2. The compressive, tensile, and bending strengths exceed the strength of compact bone by three to five times. These properties, combined with high moduli of elasticity, and especially with fatigue and fracture strengths, have resulted in specialized design requirements for these classes of biomaterials.[19,84] For

Table 24-2 Engineering Properties of Some Inert Ceramics Used as Biomaterials*

MATERIAL	MODULUS OF ELASTICITY GN/m^2 ($psi \times 10^6$)	ULTIMATE BENDING STRENGTH MPa (ksi)	SURFACE
Aluminum oxide			
Polycrystalline	372 (54)	300-550 (43-80)	Al_2O_3
Single crystal (sapphire)	392 (56)	640 (93)	Al_2O_3
Zirconium oxide zirconia (PSZ)	195-210 (28-30)	500-650 (72-94)	ZrO_2
Titanium oxide (titania)	280 (41)	69-103 (10-15)	TiO_2

*These high ceramics have 0% permanent elongation at fracture.
GN/m², Giganewton per meter squared; *psi*, pounds per inch squared; *MPa*, megapascal; *ksi*, thousand pounds per inch squared.

WWW.HIGHDENT.IR
مرجع تخصصی دندانسازان و دندانپزشکان

example, the fabrication of a subperiosteal device from a high ceramic should not be done because of the custom nature of these devices, the lower fracture resistance, and the relative cost for manufacturing. The aluminum, titanium, and zirconium oxide ceramics have a clear, white, cream, or light-gray color, which is beneficial for applications such as anterior root form devices. Minimal thermal and electrical conductivity, minimal biodegradation, and minimal reactions with bone, soft tissue, and the oral environment are also recognized as beneficial when compared with other types of synthetic biomaterials. In early studies of dental and orthopedic devices in laboratory animals and humans, ceramics have exhibited direct interfaces with bone, similar to an osteointegrated condition with titanium. In addition, characterization of gingival attachment zones along sapphire root form devices in laboratory animal models has demonstrated regions of localized bonding.[9,85-89]

Although the ceramics are chemically inert, care must be taken in the handling and placement of these biomaterials. Exposure to steam sterilization results in a measurable decrease in strength for some ceramics; scratches or notches may introduce fracture initiation sites; chemical solutions may leave residues; and the hard and sometimes rough surfaces may readily abrade other materials, thereby leaving a residue on contact. Dry-heat sterilization within a clean and dry atmosphere is recommended for most ceramics.

One series of root form and plate form devices used during the 1970s resulted in intraoral fractures after several years of function.[90] The fractures were initiated by fatigue cycling where biomechanical stresses were along regions of localized bending and tensile loading. Although initial testing showed adequate mechanical strengths for these polycrystalline alumina materials,[91] the long-term clinical results clearly demonstrated a functional design-related and material-related limitation. This illustrates the need for controlled clinical investigation to relate basic properties to in vivo performance. The established chemical biocompatibilities, improved strength and toughness capabilities of sapphire and zirconia, and the basic property characteristics of high ceramics continue to make them excellent candidates for dental implants.

Bioactive and Biodegradable Ceramics Based on Calcium Phosphates

Bone Augmentation and Replacement

The calcium phosphate ($CaPO_4$) materials (i.e., calcium phosphate ceramics [CPCs]) used in dental reconstructive surgery include a wide range of implant types and thereby a wide range of clinical applications. Early investigations emphasized solid and porous particulates with nominal compositions that were relatively similar to the mineral phase of bone ($Ca_5[PO_4]_3OH$). Microstructural and chemical properties of these particulates were controlled to provide forms that would remain intact for structural purposes after implantation. The laboratory and clinical results for these particulates were most promising and led to expansions for implant applications, including larger implant shapes (e.g., rods, cones, blocks, H-bars) for structural support under relatively high-magnitude loading conditions.[92,93] In addition, the particulate size range for bone replacements was expanded to both smaller and larger sizes for combined applications with organic compounds. Mixtures of particulates with collagen, and subsequently with drugs and active organic compounds such as bone morphogenetic protein (BMP), increased the range of possible applications. Over the past 20 years, these types of products and their uses have continued to expand significantly.[93-96]

Endosteal and Subperiosteal Implants

The first series of structural forms for dental implants included rods and cones for filling tooth root extraction sites (ridge retainers)[97] and, in some cases, load-bearing endosteal implants.[98] Limitations in mechanical property characteristics soon resulted in internal reinforcement of the CPC implants through mechanical (central metallic rods) or physicochemical (coating over another substrate) techniques.[99,100]

The numbers of coatings of metallic surfaces using flame or plasma spraying (or other techniques) increased rapidly for the CPCs.[93] The coatings have been applied to a wide range of endosteal and subperiosteal dental implant designs, with an overall intent of improving implant surface biocompatibility profiles and implant longevities (and are addressed later in this chapter).[101-103]

Advantages and Disadvantages

Box 24-1 summarizes the advantages and disadvantages of CPCs.

Box 24-1 Advantages and Disadvantages of Calcium Phosphate Ceramics

Advantages	Disadvantages
Chemistry mimics normal biological tissue (C,P,O,H)	Variable chemical and structural characteristics (technology and chemistry related)
Excellent biocompatibility	Low mechanical tensile and shear strengths under fatigue loading
Attachment between CPC and hard and soft tissues	Low attachment between coating and substrate
Minimal thermal and electrical conductivity	Variable solubility
Moduli of elasticity closer to bone than many other implantable materials	Variable mechanical stability of coatings under load-bearing conditions
Color similar to hard tissues	Overuse
Extensive research	

CPC, Calcium phosphate ceramics.

WWW.HIGHDENT.IR

The recognized advantages associated with the CPC biomaterials are as follows[104]:

1. Chemical compositions of high purity and of substances that are similar to constituents of normal biological tissue (calcium, phosphorus, oxygen, and hydrogen)
2. Excellent biocompatibility profiles within a variety of tissues, when used as intended
3. Opportunities to provide attachments between selected CPC and hard and soft tissues
4. Minimal thermal and electrical conductivity plus capabilities to provide a physical and chemical barrier to ion transport (e.g., metallic ions)
5. Moduli of elasticity more similar to bone than many other implant materials used for load-bearing implants
6. Color similar to bone, dentin, and enamel
7. An evolving and extensive base of information related to science, technology, and application

Some of the possible disadvantages associated with these types of biomaterials are as follows:

1. Variations in chemical and structural characteristics for some currently available implant products
2. Relatively low mechanical tensile and shear strengths under condition of fatigue loading
3. Relatively low attachment strengths for some coating-to-substrate interfaces
4. Variable solubilities depending on the product and the clinical application (The structural and mechanical stabilities of coatings under in vivo load-bearing conditions—especially tension and shear—may be variable as a function of the quality of the coating.)
5. Alterations of substrate chemical and structural properties related to some available coating technologies
6. Expansion of applications that sometimes exceed the evolving scientific information on properties

Critical to applications are the basic properties of these substances. Table 24-3 provides a summary of some properties of bioactive and biodegradable ceramics. In general, these classes of bioceramics have lower strengths, hardnesses, and moduli of elasticity than the more chemically inert forms previously discussed. Fatigue strengths, especially for porous materials, have imposed limitations with regard to some dental implant designs. In certain instances, these characteristics have been used to provide improved implant conditions (e.g., biodegradation of particulates).

Calcium aluminates, sodium-lithium invert glasses with $CaPO_4$ additions (Bioglass or Ceravital), and glass ceramics (AW glass-ceramic) also provide a wide range of properties and have found extended applications.[96,100]

Bioactive Ceramic Properties

Physical properties are specific to the surface area or form of the product (block, particle), porosity (dense, macroporous, microporous), and crystallinity (crystalline or amorphous). Chemical properties are related to the calcium-phosphate ratio, composition, elemental impurities (e.g., carbonate), ionic substitution in atomic structure, and the pH of the surrounding region. These properties plus the biomechanical environment all play a role in the rate of resorption and the clinical application limits of the materials.

The atomic relationships of the basic elements, stoichiometric ratios, and the normal chemical names for several characterized CPCs are provided in Table 24-4. The general family of apatites has the following formula:

$$M_{10}^{2+}(XO_4^{3-})_6Z_2^{-1}$$

Very often, apatite atomic ratios are nonstoichiometric; that is, 1 mol of apatite may contain fewer than 10 mol of metallic ions (M^{2+}) and fewer than 2 mol of anions Z^{-1}.[105] The number of XO retains a number of 6. Multiple metals and anions can be substituted within this formulation. Most important, the relative physical, mechanical, and chemical properties of each final $CaPO_4$ material, including each of the apatites, are different from one another.[89,95] Additionally, the microstructure of any final product (solid structural form or coating) is equally important to the basic properties of the substance alone. The crystalline monolythic hydroxyapatite (HA) (fired ceramic $Ca_{10}[PO_4]_6[OH]_2$) of high density and purity (50 maximum ppm impurities)

Table 24-3 Properties of Bioactive and Biodegradable Ceramics*

MATERIAL	MODULUS OF ELASTICITY GPa (psi × 10^6)	ULTIMATE BENDING STRENGTH MPa (ksi)	SURFACE
Hydroxyapatite	40-120 (6-17)	40-300 (6-43)	$Ca_{10}(PO_4)_6(OH)_2$
Tricalcium phosphate	30-120 (4-17)	15-120 (2-17)	$Ca_3(PO_4)_2$
Bioglass or Ceravital	40-140 (6-20)	20-350 (3-51)	$CaPO_4$
AW ceramic	124 (18)	213 (31)	$CaPO_4$ + F
Carbon	25-40 (4-6)	150-250 (22-36)	C
Carbon-silicon (LTI)	25-40 (4-6)	200-700 (29-101)	CSi

GPa, Gigapascal; *psi*, pounds per inch squared; *MPa*, megapascal; *ksi*, thousand pounds per inch squared; *LTI*, low-temperature isotropic.
*These ceramics and carbons have 0% permanent elongation at fracture.

WWW.HIGHDENT.IR

Table 24-4 Names, Formulae, and Atomic Ratios for Some Calcium Phosphate Materials

MINERAL OR GENERAL NAME	FORMULA	CA:P RATIO	APPLICATIONS
Monetite (DVP)	$CaHPO_4$	1	Nonceramic bone substitute particulate
Brushite (DCPD)	$CaHPO_4\ 2H_2O$	1	Phase of some $CaPO_4$ biomaterials
Octacalcium phosphate (OCP)	$Ca_8(HPO_4)_2(PO_4)\ 5H_2O$	1.33	Phase of some $CaPO_4$ biomaterials
Whitlockite (WH)	$Ca_{10}(HPO_4)(PO_4)_6$	1.43	Phase of some $CaPO_4$ biomaterials
Beta-tricalcium phosphate (β-TCP)	$Ca_3(PO_4)_2$	1.48	Biodegradable $CaPO_4$ ceramic for bone substitute and coatings; also a phase of some $CaPO_4$ biomaterials
Defective hydroxyapatite (DOHA) biomaterials	$Ca_9(HPO_4)(PO_4)_5(OH)$	1.5	Component of some $CaPO_4$ biomaterials
Hydroxyapatite (HA)	$Ca_{10}(PO_4)_6(OH)_2$	1.67	Major mineral phase of bone; when fired as a ceramic, named *HA*

has provided one standard for comparison related to implant applications. The ratio of calcium to phosphorus of $Ca_{10}(PO_4)_6(OH)_2$ is 1.67, and the ceramic can be fully crystalline. Considerable differences exist between the synthetic HA ceramics (HAs) that are produced by elevated temperature processing and biological apatites (HAs).[105] Biological apatites contain trace amounts of CO_3^{2-}, sodium, magnesium, fluorine, and chlorine ions. These exist in varying ratios and distributions and of course are only one phase of calcified tissues.

The crystalline tricalcium phosphate ($\beta Ca_3[PO_4]_2$) (TCP) ceramic has also provided a high-purity (<50 ppm maximum impurities) biomaterial for comparison with other products. National standard specifications related to the basic properties and characteristics of both HA and TCP have been published.[19] These two compositions have been used most extensively as particulates for bone augmentation and replacement, carriers for organic products, and coatings for endosteal and subperiosteal implants.

One of the more important aspects of the CPCs relates to the possible reactions with water. For example, hydration can convert other compositions to HA; also, phase transitions among the various structural forms can exist with any exposure to water. This has caused some confusion in the literature, in that some CPCs have been steam autoclaved for sterilization purposes before surgical implantation. Steam or water autoclaving can significantly change the basic structure and properties of CPCs (or any bioactive surface) and thereby provide an unknown biomaterial condition at the time of implantation. This is to be avoided through the use of presterilized or clean, dry heat or gamma sterilized conditions.

Forms, Microstructures, and Mechanical Properties

Particulate HA, provided in a nonporous (<5% porosity) form as angular or spherically shaped particles, is an example of a crystalline, high-purity HA biomaterial[106] (Figure 24-2, *A*). These particles can have relatively high compressive strengths (up to 500 MPa), with tensile strengths in the range of 50 to 70 MPa. Usually, dense polycrystalline ceramics consisting of small crystallites exhibit the highest mechanical strength, apart from monocrystalline ceramics free of defects (e.g., single-crystal sapphire implants). Ceramics are brittle materials and exhibit high compressive strengths compared with tensile strengths. However, less resistance to tensile and shear stresses limit their application as dental implants because of mechanical constraints of implant form and volume. Nonresorbable, "bioinert" ceramics exhibiting satisfactory load-bearing capability are limited to dense monocrystalline and polycrystalline aluminum, zirconium, and titanium oxide ceramics. These same mechanical characteristics exist for the solid portions of several porous HA particulates and blocks. The macroporous (>50 µm), or microporous (<50 µm) particulates have an increased surface area per unit volume. This provides more surface area for solution- and cell-mediated resorption under static conditions and a significant reduction in compressive and tensile strengths (Figure 24-2, *B* and *C*; Figure 24-3). The porous materials also provide additional regions for tissue ingrowth and integration (mechanical stabilization) and thereby a minimization of interfacial motion and dynamic (wear-associated) interfacial breakdown. The strength characteristics after tissue ingrowth would then become a combination of the ceramic and the investing tissues.[107]

A number of the CPCs are phase mixtures of HA and TCP, whereas some compounds are composites or mechanical mixtures with other materials[93] (see Table 24-4). These classes of bioactive ceramics, including glasses, glass-ceramics, mixtures of ceramics, combinations of metals and ceramics, or polymers and ceramics, exhibit a wide range of properties. In general, these biomaterials have shown acceptable biocompatibility profiles from

WWW.HIGHDENT.IR

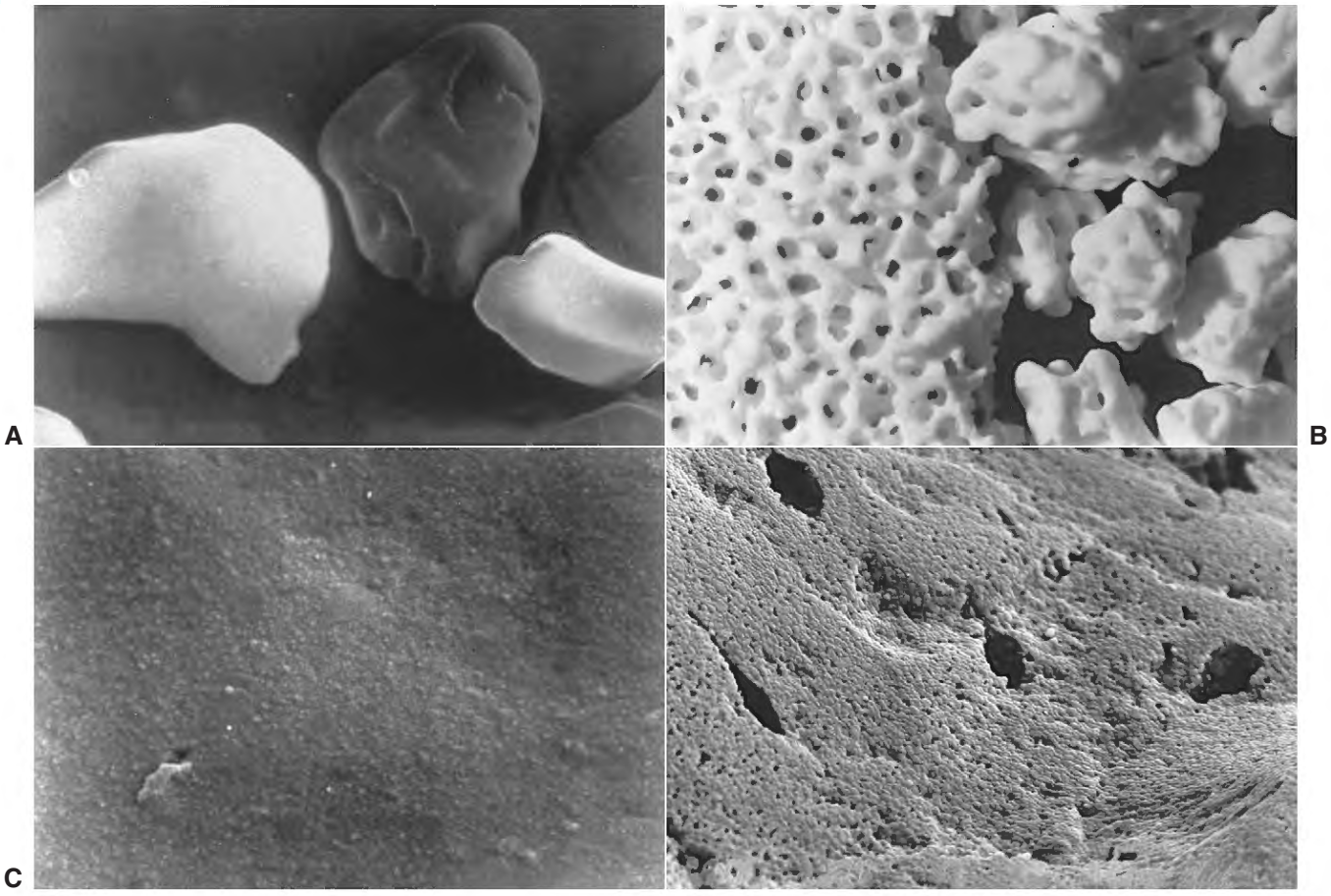


Figure 24-2 **A,** Particulate dense hydroxyapatite (HA) presents as a crystalline nonporous material with angular or spherical particles. **B,** Macroporous and microporous **(C)** particulate offer the advantage of increased surface area per unit volume, which facilitates solution and cell-mediated resorption. *(Courtesy Ceramed Corp, Denver, Colo.)*

laboratory and clinical investigations. Bulk-form implant designs made from CPCs, which were contraindicated for some implant designs because of poor mechanical performance, have found a wide range of indications as coatings of stronger implant materials.

The coatings of CPCs onto metallic (cobalt- and titanium-based) biomaterials have become a routine application for dental implants. For the most part these coatings are applied by plasma spraying, have average thickness between 50 and 70 µm, are mixtures of crystalline and amorphous phases, and have variable microstructures (phases and porosities) compared with the solid portions of the particulate forms of HA and TCP biomaterials.[93,108] At this time, coating characteristics are relatively consistent, and the quality control and stricter quality assurance programs from the manufacturers have greatly improved the consistency of coated implant systems. (A more detailed discussion of surface treatment options is presented in the next section.)

Concerns continue to exist about the fatigue strengths of the $CaPO_4$ coatings and coating-substrate interfaces under tensile and shear loading conditions. There have been some reports of coating loss as a result of mechanical fracture, although the numbers reported remain small.[89] This has caused some clinicians and manufacturers to introduce designs in which the coatings are applied to shapes (geometric designs) that

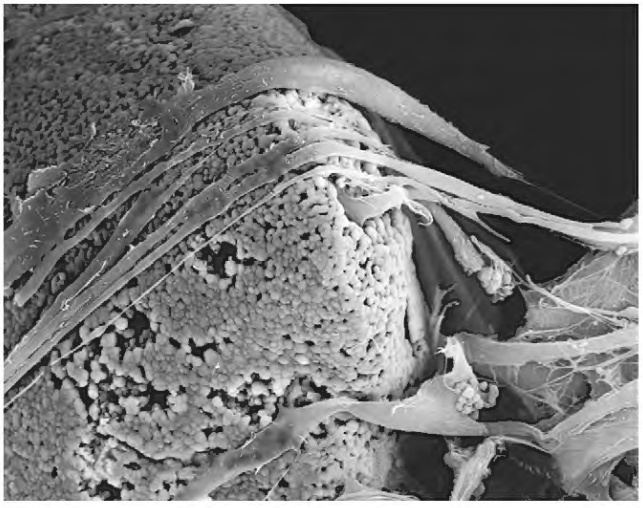


Figure 24-3 Scanning electron microscopy of cells, which actively endocytosed fragments of granules (×1500). *(Courtesy Ceramed Corp, Denver, Colo.)*

WWW.HIGHDENT.IR

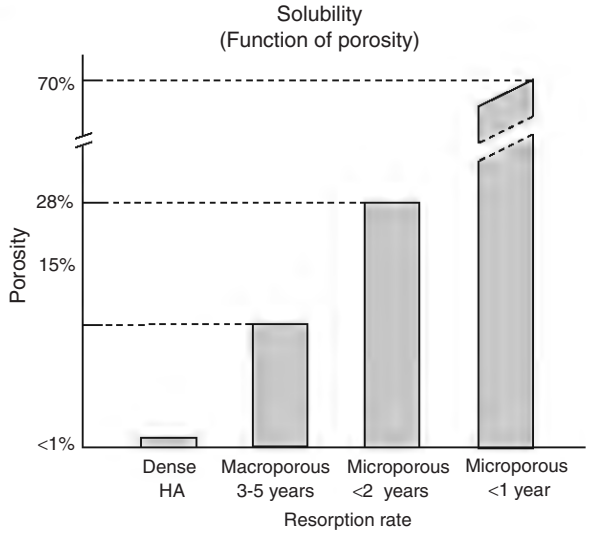


Figure 24-4 Diagram of solubility of hydroxyapatite (HA) in function of percent porosity.

minimize implant interface shear or tensile loading conditions (e.g., porosities, screws, spirals, plateaus, vents). From theoretic considerations, the coating of mechanically protected areas seems most desirable.

Density, Conductivity, and Solubility

Bioactive ceramics are especially interesting for implant dentistry because the inorganic portion of the recipient bone is more likely to grow next to a more chemically similar material. The bioactive (bioreactive) categorization includes $CaPO_4$ materials such as TCP, HA, calcium carbonate (corals), and calcium sulfate–type compounds and ceramics. A chemical-biochemical contact between the host bone and grafted material may be developed, as well as a possible stimulus of bone activity.[95] Their limitations have been associated with the material forms that have lower strengths (i.e., similar to or less than bone).[95]

The very technique-sensitive fabrication steps related to phase transition and thermal expansion during cooling might cause the final product of $CaPO_4$-type coatings to be more or less resorbable. In addition, the original categories of resorbable versus nonresorbable for these materials must be carefully weighed as a function of their particle size, porosity, chemical structure, and environmental exposure conditions.

Dissolution characteristics of bioactive ceramics have been determined for both particulates and coatings.[109,110] In general, solubility is greater for TCP than for HA. Each increase relative to increasing surface area per unit volume (porosity) and the CPC solubility profiles depend on the environment (e.g., pH, mechanical motion).

If one considers a uniform material chemistry, then the larger the particle size is, the longer the material will remain at an augmentation site. Thus 75-µm particles will be resorbed more rapidly than 3000-µm particles. In addition, the porosity of the product affects the resorption rate. Tofe et al.[111] reported on the porosity of dense, macroporous and microporous $CaPO_4$. Some of the dense HA lacks any macro- or microporosity within the particles. The longest resorption rate occurred with the dense nonporous HA type because osteoclasts may only attack the surface and cannot penetrate the nonporous material. Macroporous $CaPO_4$ (e.g., corallin HA) demonstrated 100-µm or 500-µm pores, which comprised 15% or more of the total material volume. Minimal porosity was found in the HA bulk material that surrounded the large pores. Microporous apatites often have their origin from bovine or human bone. The porosity observed in these materials is approximately 5 µm or less and comprises less than 28% of the total volume. The pores or holes are regions where blood components and organic materials can reside when placed within bone, and they represent the regions where living material existed before the processing of the implant material. The greater the porosity is, the more rapid is the resorption of the graft material. For example, clinical observation shows dense crystalline forms of HA may last longer than 15 years in the bone, the macroporous 5 years, and the microporous HA as short as 6 months (Figure 24-4).

The crystallinity of HA also affects the resorption rate of the material. The highly crystalline structure is more resistant to alteration and resorption. An amorphous product has a chemical structure that is less organized with regard to atomic structure. The hard or soft tissues of the body are more able to degrade the components and resorb the amorphous forms of grafting materials. Thus crystalline forms of HA are found to be very stable over the long term under normal conditions, whereas the amorphous structures are more likely to exhibit resorption and susceptibility to enzyme- or cell-mediated breakdown.[112] Therefore, in general, the less crystalline the material, the faster its resorption rate.[92,93,95,112,113]

The purity of the HA bone substitutes may also affect the resorption rate. The resorption of the bone substitute may be cell or solution mediated. Cell-mediated resorption requires processes associated with living cells to resorb the material, similar to the modeling and remodeling process of living bone, which demonstrates the coupled resorption and formation process. A solution-mediated resorption permits the dissolution of the material by a chemical process. Impurities or other compounds in bioactive ceramics, such as calcium carbonate, permit more rapid solution-medicated resorption, which then increases the porosity of the bone substitute. Although the coralline HA does not demonstrate micropores along the larger holes, the HA may have carbonates incorporated within the material, which hastens the resorption process.

WWW.HIGHDENT.IR

The pH in the region in which the bone substitutes are placed also affects the rate of resorption. As the pH decreases (e.g., because of chronic inflammation or infection) the components of living bone, primarily $CaPO_4$, resorb by a solution-mediated process (i.e., they become unstable chemically).

The $CaPO_4$ coatings are nonconductors of heat and electricity. This can provide a relative benefit for coated dental implants, where mixtures of conductive materials may be included in the overall prosthetic reconstruction. In combination with color (off white), these properties are considered to be advantageous.

In most applications within bone, solubilities are higher during the first few weeks, then decrease with continued in vivo exposure and the apposition of mineralized structures.[109,110] However, some investigators have shown situations in which osteoclastic resorption has removed localized zones of $CaPO_4$ coatings.[114] This raises interesting questions about long-term in vivo stabilities. At this time, clinical results have been favorable, and expanded applications have continued.

Current Status and Developing Trends

The CPCs have proved to be one of the more successful high technology–based biomaterials that have evolved within the past decades. Their advantageous properties strongly support the expanding clinical applications and the enhancement of the biocompatibility profiles for surgical implant uses.

Within the overall theme for new-generation biomaterials to be chemically (bonding to tissue) and mechanically (nonuniform, multidirectional properties) anisotropic, the CPCs could be the biomaterial surfaces of choice for many device applications.[115,116]

Carbon and Carbon Silicon Compounds

Carbon compounds are often classified as *ceramics* because of their chemical inertness and absence of ductility; however, they are conductors of heat and electricity. Extensive applications for cardiovascular devices, excellent biocompatibility profiles, and moduli of elasticity close to that of bone have resulted in clinical trials of these compounds in dental and orthopedic prostheses. One two-stage root replacement system (Vitredent) was quite popular in the early 1970s.[10] However, a combination of design, material, and application limitations resulted in a significant number of clinical failures and the subsequent withdrawal of this device from clinical use.

Ceramic and carbonitic substances continue to be used as coatings on metallic and ceramic materials. Advantages of coatings as mentioned in an earlier section include tissue attachment; components that are normal to physiological environments; regions that serve as barriers to elemental transfer, heat, or electrical current flow; control of color; and opportunities for the attachment of active biomolecules or synthetic compounds. Possible limitations relate to mechanical strength properties along the substrate-coating interface; biodegradation that could adversely influence tissue stabilities; time-dependent changes in physical characteristics; minimal resistance to scratching or scraping procedures associated with oral hygiene; and susceptibility to standard handling, sterilizing, or placing methodologies. Greater uses of surface-coated dental implants have been developed by the research and development communities.

POLYMERS AND COMPOSITES

The use of synthetic polymers and composites continues to expand for biomaterial applications. Fiber-reinforced polymers offer advantages in that they can be designed to match tissue properties, can be anisotropic with respect to mechanical characteristics, can be coated for attachment to tissues, and can be fabricated at relatively low cost. Expanded future applications for dental implant systems, beyond inserts for damping force transfers such as those used in the IMZ (Interpore, Inc.) and Flexiroot (Interdent Corp.) systems, are anticipated as interest continues in combination synthetic and biological composites.

Structural Biomedical Polymers

The more inert polymeric biomaterials include polytetrafluoroethylene (PTFE), polyethylene terephthalate (PET), polymethylmethacrylate (PMMA), ultrahigh-molecular-weight polyethylene (UHMW-PE), polypropylene (PP), polysulfone (PSF), and polydimethylsiloxane (PDS, or silicone rubber [SR]). These are summarized in Table 24-5. In general, the polymers have lower strengths and elastic moduli and higher elongations to fracture compared with other classes of biomaterials. They are thermal and electrical insulators, and when constituted as a high-molecular-weight system without plasticizers, they are relatively resistant to biodegradation. Compared with bone, most polymers have lower elastic moduli with magnitudes closer to soft tissues.

Polymers have been fabricated in porous and solid forms for tissue attachment, replacement, and augmentation and as coatings for force transfer to soft tissue and hard tissue regions. Cold-flow characteristics and creep and fatigue strengths are relatively low for some classes of polymers (e.g., SR and PMMA) and have resulted in some limitations. In contrast, some are extremely tough and fatigue cycle resistant (e.g., PP, UHMW-PE, PTFE) and afford opportunities for mechanical force transfer within selected implant designs. Most uses have been for internal force distribution connectors for osteointegrated implants, where the connector is intended to better simulate biomechanical conditions for normal tooth functions.

WWW.HIGHDENT.IR

Table 24-5 Engineering Properties of Polymers (Some Medical Grades)*

MATERIAL	MODULUS OF ELASTICITY GPa (Psi × 10^5)	ULTIMATE TENSILE STRENGTH MPa (ksi)	ELONGATION TO FRACTURE (%)
PTFE	0.5-3 (0.07-4.3)	17-28 (2.5-4)	200-600
PET	3 (4.3)	55 (8)	50-300
PMMA	3 (4.3)	69 (10)	2-15
PE	8 (1.2)	48 (7)	400-500
PP	9 (1.3)	35 (5)	500-700
PSF	3.5 (5)	69 (10)	20-100
SR	0.1 (0.014)	5 (1.1)	300-900
POM	3 (4.3)	70 (10.1)	10-75

GPa, Gigapascal; *psi*, pounds per inch squared; *MPa*, megapascal; *ksi*, thousand pounds per inch squared; *PTFE*, polytetrafluoroethylene; *PET*, polyethylene terephthalate; *PMMA*, polymethylmethacrylate; *PE*, polyethylene; *PP*, polypropylene; *PSF*, polysulfone; *SR*, silicone rubber; *POM*, polyoxymethylene (IME insert).
*Polymer properties exhibit a wide range depending on processing and structure. These values have been taken from general tables.

The indications for PTFE have grown exponentially in the last decade because of the development of membranes for guided tissue regeneration techniques. However, PTFE has a low resistance to contact abrasion and wear phenomena.

Composites

Combinations of polymers and other categories of synthetic biomaterials continue to be introduced. Several of the more inert polymers have been combined with particulate or fibers of carbon, Al_2O_3, HA, and glass ceramics. Some are porous, whereas others are constituted as solid-composite structural forms.[117,118]

In some cases, biodegradable polymers, such as polyvinyl alcohol (PVA), polylactides or glycolides, cyanoacrylates, or other hydratable forms have been combined with biodegradable $CaPO_4$ particulate or fibers.[119] These are intended as structural scaffolds, plates, screws, or other such applications. Biodegradation of the entire system, after tissues have adequately reformed and remodeled, has allowed the development significantly advantageous procedures such as bone augmentation and peri-implant defect repairs.

In general, polymers and composites of polymers are especially sensitive to sterilization and handling techniques. If intended for implant use, then most cannot be sterilized by steam or ethylene oxide. Most polymeric biomaterials have electrostatic surface properties and tend to gather dust or other particulate if exposed to semiclean air environments. Because many can be shaped by cutting or autopolymerizing in vivo (PMMA), extreme care must be taken to maintain quality surface conditions of the implant. Porous polymers can be deformed by elastic deformation, which can close open regions intended for tissue ingrowth. In addition, cleaning of contaminated porous polymers is not possible without a laboratory environment. In this regard, talc or starch on surgical gloves, contact with a towel or gauze pad, or the touching of any contaminated area must be prevented for all biomaterials.

Long-term experience, excellent biocompatibility profiles, ability to control properties through composite structures, and properties that can be altered to suit the clinical application make polymers and composites excellent candidates for biomaterial applications, as the constant expansion of the applications of this class of biomaterials can verify.

Inserts and Intramobile Elements

Relatively low moduli of elasticity (compared with metals and ceramics), high elongations to fracture, and inherent toughnesses have resulted in use of selected polymers for connectors or interpositional spacers for dental implants. One popular polymer insert system was included in Table 24-5 for general reference purposes. The most significant limitation has been the polymeric materials resistance to cyclical-load creep and fatigue phenomena. Retrieved transfer systems, in some clinical retrievals, have shown significant plastic deformation and fracture.[120] Although the desire to achieve such a stress-damping effect seems well founded, the inadequate long-term performance of the materials and high time and cost associated with maintenance of these devices have limited their field of application, and they are used less today than during the last decade.

FUTURE AREAS OF APPLICATION

Synthetic substances for tissue replacement have evolved from selected industrial grade materials such as metals, ceramics, polymers, and composites. This situation offers opportunities for improved control of basic properties. The simultaneous evolution of the biomechanical sciences also provides optimization of design and material concepts for surgical implants. Knowledge of tissue properties and

WWW.HIGHDENT.IR

computer-assisted modeling and analyses also support the present developments. The introduction of anisotropy with respect to mechanical properties; chemical gradients from device surface to center, with bonding along the tissue interfaces; and control of all aspects of manufacturing, packaging, delivering, placing, and restoring enhance the opportunities for optimal application and, it is hoped, device treatment longevities. Health care delivery would benefit from better availability and decreased per-unit costs.

Combinations to provide compositions with bioactive surfaces, the addition of active biomolecules of tissue-inductive substances, and a stable transgingival attachment mechanism could improve device systems. An integrated chemical and physical barrier at the soft tissue transition region would, at least theoretically, enhance clinical longevities. Devices that function through bone or soft tissue interfaces along the force transfer regions could be systems of choice, depending on the clinical situation.[9]

Unquestionably, the trend for conservative treatment of oral diseases will continue. Thus it can be anticipated that dental implants will frequently be a first-treatment option. Therefore increased use of root form systems is to be expected. Clearly the true efficacy of the various systems will be determined by controlled clinical studies with 10- to 20-year follow-up periods, which include statistically significant quantitative analyses.

SURFACE CHARACTERISTICS

Many aspects of biocompatibility profiles established for dental surgical implants have been shown to depend on interrelated biomaterial, tissue, and host factors. For discussion purposes, the biomaterial characteristics can be separated into categories associated with either (1) surface or (2) bulk properties. In general, the biomaterial surface chemistry (purity and critical surface tension for wetting), topography (roughness), and type of tissue integration (osseous, fibrous, or mixed) can be correlated with shorter- and longer-term in vivo host responses. Additionally, the host environment has been shown to directly influence the biomaterial-to-tissue interfacial zone specific to the local biochemical and biomechanical circumstances of healing and longer-term clinical aspects of load-bearing function. The interfacial interaction between recipient tissues and implanted material are limited to the surface layer of the implant and a few nanometers into the living tissues. The details of the integration (hard or soft tissue) and force transfer that results in static (stability) or dynamic (instability or motion) conditions have also been shown to significantly alter the clinical longevities of intraoral device constructs.

Many of the conference proceedings cited have focused on biomaterial-to-tissue interfacial interactions, which strongly supports the value of scrutinizing the surface characteristics of dental implants. This was one consistent recommendation from the 1978 and 1988 consensus conferences on the benefit and risk aspects of dental implant–based clinical treatments.[9,10,121]

The synthetic biomaterials used for the construction of dental implants and the associated abutments that contact subepithelial zones of oral tissues can be classified into *metallic*, *ceramic*, and *surface-modified* (coated, reacted, or ion-implanted) groups. It has long been recognized that synthetic biomaterials should be mechanically and chemically clean at the time of surgical placement. Surface properties are chemical in nature and have been described in terms of atomic structural characteristics with extensions to the subatomic scale. These characteristics are critical to the surface composition, corrosion resistance, cleanliness, surface energy, flexure, and tendency to interact, such as the ability to denature proteins.

Surface characteristics are the theme of this section, with emphasis on metallic, ceramic, and surface-modified dental implant biomaterials.

SURFACE CHARACTERIZATION AND TISSUE INTERACTION

Metal and Alloy Surfaces

Standard grades of alpha (unalloyed) titanium and alpha-beta and beta-base alloys of titanium exist with an oxide surface at normal temperatures, with ambient air or normal physiologic environments that act as oxidizing media. A formation of a thin oxide exists via dissociation of and reactions with oxygen or other mechanisms such as oxygen or metal ion diffusion from and to the metallic surface, especially for titanium. Independent from the fabrication process, the oxide is primarily TiO_2, with small quantities of Ti_2O_3 and TiO, with some minor variable stoichiometry.[122-126] This thin layer of amorphous oxide will rapidly reform if removed mechanically. Surface properties are the result of this oxide layer and differ fundamentally from the metallic substrate.[63,123] Therefore the oxidation parameters such as temperature, type and concentration of the oxidizing elements, and eventual contaminants all influence the physical and chemical properties of the final implant product. The type of oxide on surgical implants is primarily amorphous in atomic structure (brookite) if formed in normal-temperature air or tissue fluid environments and is usually very adherent and thin in thickness dimensions (<20 nm). In contrast, if unalloyed titanium (alpha) substrates (titanium grades 1 to 4) are processed at elevated temperatures (above approximately 350° C [660° F]) or anodized in organic acids at higher voltages (above 200 mV), then the oxide forms a crystalline atomic structure (rutile or anatase) and can be 10 to 100 times thicker. The grain structure of

WWW.HIGHDENT.IR

the metal and the oxidation conditions also condition the microstructure and morphology of the surface oxides. Porosity, density, and general homogeneity of the substrate are all related to this process. Low-temperature thermal oxides are relatively homogeneous and dense[127]; with increasing temperatures they become more heterogeneous and more likely to exhibit porosity as scale formations, and some have glasslike surface oxide conditions (semicrystalline).[125,127]

Depending on the mechanical aspects of polishing and the chemical and electrochemical aspects of cleaning and passivating, these amorphous or crystalline oxides can exhibit microscopically smooth or rough topographies at the micrometer level. However, surface macroscopic roughness is normally introduced into the substrate beneath the oxide zone by mechanical (grinding), particulate blasting (resorbable blast media or other), or chemical (acid etching) procedures. The surface topography and roughness obtained by such techniques is characteristic of each fabrication process.[11,128] The oxide dimension (thickness) along these rougher surfaces remains relatively constant and within nanometer dimensional thicknesses under normal temperature and environmental exposure conditions.

The titanium alloys used for dental implant components include microstructural phases of alpha and beta or room temperature stabilized beta (only). The alpha-phase surface regions of the alloy are similar to unalloyed titanium in atomic arrangement (close-packed hexagonal), whereas the beta phases demonstrate a different atomic structure (body-centered cubic) and elemental chemistry. However, the beta phase oxide formation kinetics, chemistry, dimensions, and environmental stabilities are relatively similar to the alpha-phase regions. Electrochemical investigations have shown that the alpha- and beta-phase oxides provide substrate coverage and a high degree of chemical and biochemical inertness (resistance to corrosion and ion transfer) for titanium and alloys of titanium. Both titanium and Ti-6Al-4V have been reported to contain small amounts of titanium nitride along their surface oxide.[124,129,130] Ions, carbon, and substances other than alloying elements may be picked up in the oxide through the preparation process, similar to that found at the surface of CP titanium.[126,131-134] Nevertheless, in the cases of titanium and titanium alloy, the oxide layer grows homogeneously and a well-controlled inert coating of very stable insoluble oxide normally contacts the living tissues.

Considerable research has been conducted on the roles of alloying elements in titanium alloys and how these elemental compositions may influence oxide properties and host tissue compatibility. This is dependent on the amount of the ions available to the tissues and relative rates of ion transfers, which could result in host tissue toxicity. In general, adequately processed and finished titanium alloys have shown integration with bone and soft tissue environments for a wide range of dental and medical implant devices.

Surface analysis studies have shown that the titanium alloy exhibits a similar oxide layer and as such is able to interact with surrounding bone in ways that are similar to unalloyed titanium.[135] Predictable results can be achieved with titanium alloy implant with a similar degree of bone integration.[136] In addition, electrochemical measurements of corrosion and ion release rates strongly support the chemical-biochemical stability properties of titanium alloys.

Some reports have expressed concerns because titanium alloy surface oxides contain significant amounts of alloying elements and exhibit different morphology and crystallization.[129,137-140] Aluminum in particular has been reported in both the outermost and the innermost layers. At the innermost layer, it was found especially over mixed (alpha) and (beta) phases grains of the alloy.[129] The different surface oxides are then argued to be responsible for a "lesser" quality of osseointegration in particular because of the potential of corrosion products containing aluminum and vanadium.[141-143] The orthopedic and dental literature specific to in vivo animal and human studies have also documented long-term success with titanium alloys that demonstrated close physical adaption of the bone to the surface of the alloy.[144-151]

Tissue Interactions

Oxide modification during in vivo exposure has been shown to result in increased titanium oxide layer thickness of up to 200 nm.[152-154] The highest oxide growth area corresponded to a bone marrow site, whereas the lowest growth was associated with titanium in contact with cortical regions of bone. Increased levels of calcium and phosphorus were found in the oxide surface layers and seemed to indicate an active exchange of ions at the interface. Hydrogen peroxide environmental conditions have been shown to interact with Ti and form a complex gel.[155-157] "Titanium gel conditions" are credited with attractive in vitro properties such as low apparent toxicity, inflammation, bone modeling, and bactericidal characteristics. The authors restricted their studies to CP titanium exclusively and not titanium alloys.

Other elements interacting with the surface layer of several implanted materials are calcium and phosphorus,[158-159] exhibiting a $CaPO_4$ structure somewhat similar to apatite on the titanium surface. However, the low percentage of these elements along the material surface indicates this was the result of transfer and adsorption of these elements from tissue fluids, not an osteointegration process per se.

The surface biointeraction processes may be slow or activated by local reactions and may cause ion release and oxide alteration of the substrate. Local and systemic increases of the ion concentration have been reported.[160-161]

WWW.HIGHDENT.IR

In vitro studies showed that both titanium or titanium alloy were released in measurable quantities of the substrate elements at the surface.[23,162] Especially high rates of ion release were observed in ethylenediamine tetraacetic acid (EDTA) and sodium citrate solutions and varied as a function of the corroding medium.[162] Ion release corresponds to an oxide layer thickness growth with inclusions of calcium, phosphorus, and sulfur in particular. This is especially a concern for larger orthopedic or porous implants, where such ion release may be a part of the origin of implant failure and allergic reactions and has even proposed to be a local or systemic reason for the formation of tumors. In addition, free-titanium ions have been shown to inhibit the growth of HA crystals (i.e., the mineralization of calcified tissues at the interface).[163-165]

Integration with Titanium and Alloys

Although titanium is known to exhibit better corrosion resistance, independent of the surface preparation, in vivo and in vitro studies have shown that titanium may interact with the recipient living tissues over several years. This interaction results in the release of small quantities of corrosion products even though a thermodynamically stable oxide film exists.

Several studies have concentrated on the behavior of titanium and titanium alloys in simulated biological environments. Williams cautioned that although titanium can demonstrate excellent properties of its tenacious oxide film, it is usually not sufficiently stable to prevent wear and galling in bearing systems under load. Some situations have resulted in metal-to-metal contact and local welding.[36] Solar et al.[166] stated that under static conditions, titanium and titanium alloy should withstand exposure to physiologic chlorine solutions at body temperature indefinitely but would be susceptible to oxide changes caused by mechanical micromotion. Bundy et al.[167] exposed implant alloys simultaneously to tensile stress and corrosive environments (stress-applied conditions). In vivo, stainless steel and titanium alloy demonstrated cracklike features when loaded to yield stress and then reimplanted under laboratory conditions for 8 weeks. Cracklike features also were seen in stainless steel and titanium alloy loaded to or beyond the yield stress and subsequently electrochemically polarized for 38 weeks in the in vitro part of the study. None of the samples actually failed by completely cracking, but the authors presumed that it would have occurred with a longer exposure time as previously suggested.[36,168] Geis Gestorfer and Weber[39] used linear polarization methods to show that titanium showed minimal breakdown in simulated tissue fluids, whereas Ni-Ti showed rapid breakdown of passivity with increased chlorine product–related concentrations in unbuffered solutions. Therefore body fluids could be responsible for the dissolution of some metallic passive oxide films.[169]

Lemons[75] studied single-stage solid implants modified by bending or cutting and showed that damage could increase corrosion. Rostoker and Pretzel[170] studied couple corrosion in vitro for alloys and found that dissimilar metals in a combined prosthesis did not create a regional breakdown of the titanium passive layer. A second in vivo study evaluated couple and crevice corrosion of prosthetic alloys in vertebral muscles of dogs for 30 weeks (non–load-bearing, nonosseointegrated).[171] It was concluded that metals of superior corrosion resistance, such as titanium alloy, and wrought cobalt alloys can be combined with titanium alloy in one prosthesis to provide superior mechanical performance without creating additional corrosion. However, repeated oxide breakdown such as sustained abrasion was likely to damage the corrosion resistance of an alloy for any type of coupling. Results from Thompson et al.[172] did not predict accelerated corrosion for titanium alloy coupled to carbon for galvanic couples under static conditions.

Marshak et al.[173] and Marshak[174] studied the potential for existence of SCC, GC, and FC in an in vitro study of titanium alloy and gold alloy abutment implants and abutment complexes simultaneously submitted to a laterally oriented 10-kg loading and a simulated tissue fluid solution at 37° C. Stress corrosion cracking was studied in the most likely area, that is the screw-to-abutment connection, which was under constant and simultaneous tension and compression stresses. These studies showed possibilities for interactions at contact regions between the cast gold and titanium alloy and components under selected environmental conditions.

Cohen and Burdairon[175] showed that odontologic fluoride gels, which create an acidic environment, could lead to the degradation of the titanium oxide layer and possibly inhibit the osseointegration process. Deposits consistent with the presence of GC byproducts were detected on various surfaces of the experimental metal.[176-177] Liles et al.[178] investigated the GC between titanium and seven crown and bridge alloys in 1% sodium chloride (NaCl) solution. The nonprecious Ni-Co complex was likely to trigger GC. Clinically, this means that in the short term, the presence of the surface impurities such as iron found on some implant parts, as well as other contaminants related to the machining process, could result in loss of bone and integration in crestal areas exposed to corrosion products. The long-term presence of corrosion reaction products and ongoing corrosion could also lead to fracture of the affected alloy-abutment interface, the abutment, or possibly the implant body itself. This combination of stress and corrosion, possibly together with factors associated with bacteria, could be one of the reasons why implants fail at the local or individual levels

WWW.HIGHDENT.IR

rather than in a generalized fashion.[179] Protocols for manufacturing and cleaning prosthetic titanium parts (specifically abutments contacting the implant body) appear less stringent than those for implant bodies. This should not be the case, and the same standards should be applied to both implant body and prosthetic components. In addition, the short and longer clinical implications of the potential GC effect could be ideally nullified by the use of electrochemically compatible alloys for the superstructure.

Cobalt and Iron Alloys

The alloys of cobalt (Vitallium) and iron (surgical stainless steel—316L) exhibit oxides of chromium (primarily Cr_2O_3 with some suboxides) under normal implant surface–finishing conditions after acid or electrochemical passivation. These chromium oxides, as with titanium and alloys, result in a significant reduction in chemical activity and environmental ion transfers. Under normal conditions of acid passivation, these chromium oxides are relatively thin (nanometer dimensions) and have an amorphous atomic structure. The oxide atomic spatial arrangement can be converted to a crystalline order by elevated temperature or electrochemical exposures.

The chromium oxides on cobalt and iron alloys are microscopically smooth, and again, roughness is usually introduced by substrate processing (grinding, blasting, or etching). Because these oxides, similar to titanium oxides, are very thin (nanometer dimensions), the reflected light color of the alloys depends on the metallic substrate under the oxide.[33] However, as mentioned, the titanium, cobalt, and iron metallic systems depend on the surface reaction zones with oxygen (oxides) for chemical and biochemical inertness.

The cobalt and iron alloy bulk microstructures are normally mixtures of the primary alloy phases with regions of metallic carbides distributed throughout the material.[33,56,76,77] Along the surfaces the chromium oxide covers the matrix phase (metallic regions), whereas the carbides stand as secondary components (usually as mounds above the surface) at the microscopic level. In contrast to homogenization-annealed alloys, the as-cast cobalt alloys exhibit multiphasic characteristics within their microstructure, with relatively extensive regions of the alloy surfaces occupied by complex metallic carbides. Thus tissue-to-oxide and tissue-to-metallic carbide zones could be used to describe tissue integration of cobalt alloy. This is uniquely different compared with titanium implant biomaterials, where tissue-to-oxide regions predominate at the interface.[76,77]

The iron-based alloy chromium oxide and substrate are more susceptible to environmental breakdown, in comparison with cobalt- and titanium-based biomaterials. This has been discussed in the literature related to crevice and pitting corrosion biodegradation phenomena for stainless steel implant systems.[59,76,77] In general, if stainless steel implant surfaces are mechanically altered during implantation, or if the construct introduces an interface that is subjected to biomechanical fretting, then the iron alloy will biodegrade in vivo, and the fatigue strength of surgical stainless steel can be significantly decreased in a corrosive environment.[180] In some cases this has resulted in implant loss. However, in the absence of surface damage, the chromium oxides on stainless steel biomaterials have shown excellent resistances to breakdown, and multiple examples of tissue and host biocompatibility have been shown for implants removed after long-term (beyond 30 years in vivo) implantations.

Dental implants and implant abutments have also been fabricated from gold alloy with many abutments fabricated from palladium or Co-Cr-Ni-Mo alloys.[37] The minimally alloyed gold and palladium systems are noble electrochemically and do not depend on surface oxides for chemical and biochemical inertness. This would be the case for the high-noble alloys (major compositions of gold, platinum, palladium, iridium, and ruthenium). However, some palladium alloys and other lower noble element content alloys gain chemical and biochemical inertness from complex metallic surface oxides.[37] As mentioned, the multicomponent (wrought) cobalt-based alloys, as with other base-metal systems, depend on chromium oxide surface conditions for inertness.

In general, the noble-metal alloys do not demonstrate the same characteristics of tissue interaction when compared with the base-metal (Ti and Co alloy) systems. The ultrastructural aspects of tissue integration have not been extensively investigated for noble-alloy systems, although some have presented results describing osteointegration of gold alloys. The noble alloys, when used in a polished condition, are resistant to debris accumulation on a relative basis compared with other alloys. This has been listed as an advantage for their use in intraoral abutment systems. In addition, mechanical finishing of the more noble alloys can result in a high degree of polish and a minimal concern about damaging or removing surface oxides.

Ceramics

Aluminum oxide ceramics have been extensively investigated related to surface properties and how these properties relate to bone and soft tissue integration.[92,93,104-117] Aluminum oxide ceramics are fully oxide materials (bulk and surface), thereby affording advantages related to tissue interface–related investigation. In addition, studies have included the polycrystalline (alumina) and single-crystalline (sapphire) forms of the oxide structure. These forms have introduced very different surface

WWW.HIGHDENT.IR

Figure 24-5 Machined surfaces exhibit an irregular surface with grooves, ridges, and pits including a nanometer scale. *(Brånemark fixture, Nobel Biocare.)*

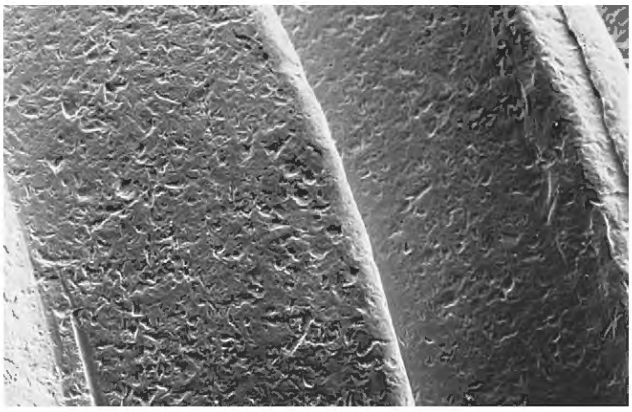


Figure 24-6 Titanium implants may be etched with a solution of nitric and hydrofluoric acids. *(Screw-vent implant, Zimmer.)*

roughness values for the same material substrate plus bulk properties where ion transfer and electrochemical phenomena are minimal influences. Bone and soft tissue integration have been demonstrated for this oxide material over the long term in humans and laboratory animals. Direct relationships have been established between the interfacial events of tissue integration for metallic surface oxides of titanium and chromium and the Al_2O_3 systems. As mentioned previously, surface quality can be directly correlated with tissue integration and clinical longevity. Because the Al_2O_3 ceramics are crystalline and extend throughout the surface and bulk zones, biomechanical instabilities do not alter the chemical aspects of biomaterial properties. (No electrochemical change is introduced if the surface is removed.) Ceramic coatings (e.g., Al_2O_3) have been shown to enhance the corrosion resistance and biocompatibility of metal implants, in particular surgical stainless steel and Ni-Cr, Co-Cr alloys.[181] However, the Ni-Cr and steel alloys can be subject to crevice corrosion. Studies in orthopedics caution that the Al_2O_3 coating may cause a demineralization phenomenon caused by a high local concentration of substrate ions in the presence of metabolic bone disease.[182] This remains to be established within the use of Al_2O_3 implants for clinical applications.

Hydroxyapatite

In addition to the bulk Al_2O_3 biomaterials, $CaPO_4$–based ceramic or ceramic-like coatings have been added to titanium and cobalt alloy substrates to enhance tissue integration and biocompatibility. These coatings, for the most part, are applied by plasma spraying small-size particles of crystalline HA ceramic powders. The process of coating and the coating dimensions and property characteristics are addressed further in the next section.

The surface topography is characteristic of the preparation process. Variations in the roughness and porosity of the surface (<100 µm) can be categorized in function of the surfacing process. Machined implants exhibit an irregular surface with grooves, ridges, and pits including a nanometer scale.[183,184] Proponents of such a surface argue that it is the most conducive to cell attachment[122-124] (Figure 24-5).

Surface roughening by particulate blasting can be achieved by different media. Sandblasting provides irregular rough surfacing with <10-µm scales and a potential for impurity inclusions. Researchers used a titanium alloy Ti-6Al-4V to improve the mechanical properties and elected to electropolish the surface to reduce surface roughness to be only in the 0.1-mm scale by controlled removal of the surface layer by dissolution.[183,185,186] Titanium implants may be etched with a solution of nitric and hydrofluoric acids to chemically alter the surface and eliminate some types of contaminant products (Figure 24-6). The acids very rapidly attack metals other than titanium, and these processes are electrochemical in nature. Proponents of this technique argue that implants treated by sandblasting and acid etch provide superior radiographic bone densities along implant interfaces compared with titanium plasma-sprayed surfaces.[187] Recently, concerns have been expressed regarding embedded media from glass beading (satin finish) and grit blasting (alumina Al_2O_3) and a possible risk of associated osteolysis caused by foreign debris.[188,189] Ricci et al.[189] reported on failed retrieved implants that exhibited extensive surface inclusions consisting of silicon and/or Al_2O_3-related product, which were also present in the surrounding tissues. A relatively new process (resorbable blast media) has been said to provide a comparable roughness to an alumina grit blast finish, which can be a rougher surface than the machined, glass-beaded, or acid-etched surfaces (Figure 24-7).[190]

Porous and Featured Coatings

The implant surface may also be covered with a porous coating. These may be obtained with titanium or HA

WWW.HIGHDENT.IR

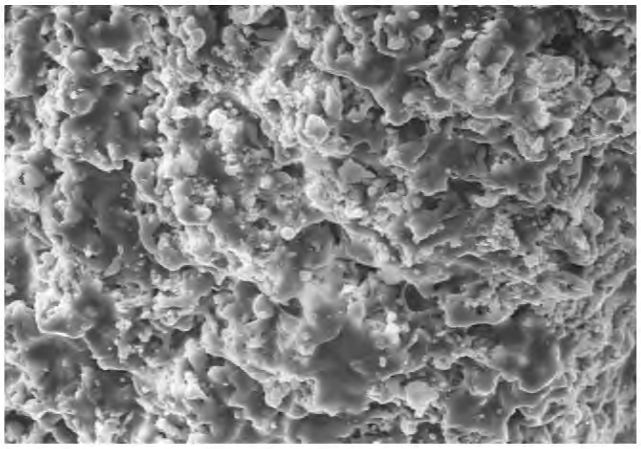


Figure 24-7 Resorbable blast media provide a comparable roughness to alumina grit blast finish, which can be rougher than machined or etched surfaces. *(D2 Maestro implant, BioHorizons.)*

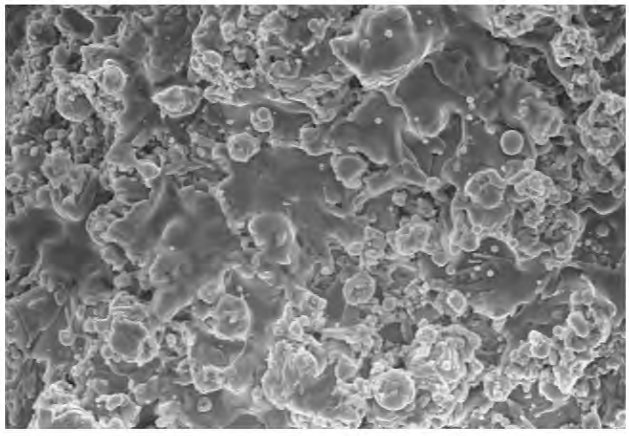


Figure 24-8 Titanium plasma-sprayed (TPS) surfaces result in increased total surface area, which may introduce a dual physical and chemical anchor system and increase load-bearing capability. (Scanning electron microscopy of BioHorizons D3 implant; ×500.)

particulate–related fabrication processes. Examples of coatings and processes for producing surface-modified implants are summarized in the following sections.

Titanium Plasma Sprayed

Porous or rough titanium surfaces have been fabricated by plasma spraying a powder form of molten droplets at high temperatures. At temperatures in the order of 15,000° C, an argon plasma is associated with a nozzle to provide very high-velocity 600 m/sec partially molten particles of titanium powder (0.05- to 0.1-mm diameter) projected onto a metal or alloy substrate.[63,191] The plasma-sprayed layer after solidification (fusion) is often provided with a 0.04- to 0.05-mm thickness. When examined microscopically, the coatings show round or irregular pores that can be connected to each other (Figure 24-8). Hahn and Palich[192] first developed these types of surfaces and reported bone ingrowth in titanium hybrid powder plasma spray-coated implants inserted in animals. Karagianes et al.[193] assessed the suitability of porous titanium and titanium alloy to achieve bone-implant bonding characteristics in miniature swine and likened it to a three-dimensional surface. Kirsch[120] conducted histologic studies for plasma flame-sprayed particulate titanium coating root form specimen (IMZ) implanted and integrated to the bone in dogs, with complete integration reported at 6 weeks. In animal experiments and histologic studies, Schroeder et al.[194] concluded that the rough and porous surfaces showed a three-dimensional interconnected configuration likely to achieve bone-implant attachment for stable anchorage. Other animal studies concluded that a porous titanium surface from various fabrication methods may increase the total surface area (up to several times), produce attachment by osteoformation, enhance attachment by increasing ionic interactions, introduce a dual physical and chemical anchor system, and increase the load-bearing capability 25% to 30%.[100,120,195-200] In vitro studies of fibroblast attachment conducted by Lowenberg et al.[201] showed superior attachment to surface-ground titanium alloy disks compared with porous titanium but with a better cell orientation on porous forms of titanium.

In 1981, Clemow et al.[202] showed that the rate and percentage of bone ingrowth into the surface was inversely proportional to the square root of the pore size for sizes greater than 100 µm and that the shear properties of the interface were proportional to the extent of bone ingrowth. The optimum pore size for bone ingrowth was determined in a study of cobalt-base alloy porous implants inserted in canine femurs. The optimum pore size was deduced from the maximum fixation strength measurements. These surface porosities ranged from 150 to 400 µm and coincidentally correspond to surface feature dimensions obtained by some plasma-spraying processes.[203-206] In addition, porous surfaces can result in an increase in tensile strength through ingrowth of bony tissues into three-dimensional features. High shear forces determined by the torque-testing methods and improved force transfer into the peri-implant area have also been reported.[207,208]

In 1985 at the Brussels Osseointegration Conference, the basic science committee did not present results that showed any major differences between smooth, rough, or porous surfaces regarding their ability to achieve osteointegration. However, proponents of porous surface preparations reported that there have been results showing faster initial healing compared with noncoated porous titanium implants and that porosity allows bone formation within the porosities even in the presence of some micromovement during the healing phase.[209,210] Such surfaces were also reported to allow the successful placement of shorter-length implants when compared with noncoated implants. The basic theory was based on increased area for bone contact. Reports in the literature caution about cracking and scaling of coatings because of stresses produced by elevated temperature

WWW.HIGHDENT.IR

processing[211,212] and risk of accumulation of abraded material in the interfacial zone during implanting of titanium plasma-sprayed implants. It may be indicated to restrict the limit of coatings in lesser bone densities that cause less frictional torque transfer during implant placement process. In addition, the present technology allows metallurgic bonding of coatings and a high resistance against mechanical separation of the coating, with many coating test values exceeding the published standard requirements.[213]

Hydroxyapatite Coating

Hydroxyapatite coating by plasma spraying was brought to the dental profession by deGroot.[92] Kay et al.[214] used scanning electron microscopy (SEM) and spectrographic analyses to show that the plasma-sprayed HA coating could be crystalline and could offer chemical and mechanical properties compatible with dental implant applications.[214] Block et al.[215] and Thomas et al.[216] showed an accelerated bone formation and maturation around HA-coated implants in dogs when compared with noncoated implants. HA coating can also lower the corrosion rate of the same substrate alloys.[217] Researchers measured the HA coating thickness after retrieval from specimens inserted in animals for 32 weeks and showed a consistent thickness of 50 mm, which is in the range advocated for manufacturing.[19,89,218,219] The bone adjacent to the implant has been reported to be better organized than with other implant materials and with a higher degree of mineralization.[220] In addition, numerous histologic studies have documented the greater surface area of bone apposition to the implant in comparison with uncoated implants,[215,221,222] which may enhance the biomechanics and initial load-bearing capacity of the system. HA coating has been credited with enabling HA-coated titanium or titanium alloy implants to obtain improved bone-implant attachment compared with machined surfaces.

Studies also demonstrated that the HA-bone attachment is superior to the HA-implant interface.[216,218,223] However, proponents of such surfaces report excellent reliability of HA-coated implants.[224,225] The most significant result is the increase in bone penetrations, which enhances fixation in areas of limited initial bone contact.[37,40,41,226] However, controversies still exist, and some authors caution that HA coatings do not necessarily represent an advantage for the long-term prognosis of the system.

Implants of solid sintered HA have been shown to be susceptible to fatigue failure.[98,219,223] This situation can be altered by the use of a CPC coating along metallic substrates. Although several methods may be used to apply CPC coatings, the majority of commercially available implant systems are coated by a plasma spray technique. A powdered crystalline HA is introduced and melted by the hot, high-velocity region of a plasma gun and propelled onto the metal implant as a partially melted ceramic (Figure 24-9).[108,191] One of the concerns regarding CPC coatings is the strength of the bond between the CPC and the metallic substrate. Investigative ion beam–sputtering coating techniques for CPC or CPC-like nonresorbable coatings to varied substrates appear to produce dense, more tenacious, and thinner coatings (a few micrometers), which would minimize the problem of poor shear strength and fatigue at the coating-substrate interface.[108] Recent reports have introduced a new type of treatment for coatings, which appear primarily amorphous in nature, and further in vivo studies are needed to determine tissue response.[227,228] Other investigations include developing new biocompatible coatings based on TCP or titanium nitride.[229]

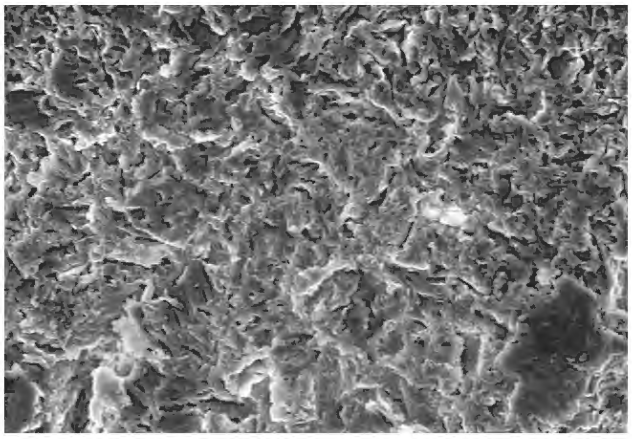


Figure 24-9 Hydroxyapatite (HA) coatings on implant surface provide several clinical properties because of the osteoconductive properties of HA. (Scanning electron microscopy [SEM] of BioHorizons D4 implant; ×500.)

It has been shown that the plasma-spraying technique can alter the nature of the crystalline ceramic powder and can result in the deposition of a variable percentage of a resorbable amorphous phase.[230] A dense coating with a high crystallinity has been listed as desirable to minimize in vivo resorption. In addition, the deposited CPC may be partially resorbed through remodeling of the osseous interface.[28,231,232] It is therefore wise to provide a biomechanically sound substructure design[230,231] that is able to function under load-bearing conditions to compensate for the potential loss of the CPC coating over years. In addition, the CPC coatings may resorb in infected or chronic inflammation areas. Animal studies also show reductions in coating thickness after in vivo function.[233] One advantage of CPC coatings is that they can act as a protective shield to reduce potential slow ion release from the Ti-6Al-4V substrate.[234] In addition, the interdiffusion between titanium and calcium (and phosphorus and other elements) may enhance the coating substrate bond by adding a chemical component to the mechanical bond.[232,235-238]

When these coatings were introduced more than two decades ago, many researchers expressed concerns about

WWW.HIGHDENT.IR

the biomechanical and gingival sulcus area biochemical stabilities. It was recommended that national and international standards for these coatings be developed, in part to provide detailed description of coating properties using consistent and uniform (standardized) test methods. Initial national standards were developed for *Beta Tricalcium Phosphate for Surgical Implantation by the ASTM Committee F4* (ASTM F4-1088). A standard specification for *Composition of Ceramic Hydroxyapatite for Surgical Implants* (ASTM F4-1185) was developed, and additional standards have been more recently approved, including *Glass and Glass-Ceramic Biomaterials for Implantation* (ASTM F4-1538), *Standard Test Method for Tension Testing of Calcium Phosphate Coatings* (ASTM F4-1501 F1147-05), *Standard Test Method for Calcium Phosphate Coatings for Implantable Materials* (ASTM F4-1609), *Test Method for Bending and Shear Fatigue Testing of Calcium Phosphate Coatings on Solid Metallic Substrates* (ASTM F4-1659 1160), and a *Standard Test Method for Shear Testing of Calcium Phosphate Coatings* (ASTM F4-1658 1044).[19] Additional standards being developed at the task group level with ASTM F4 include *Calcium Phosphate Coating Crystalline Characteristics, Mechanical Requirements for Calcium Phosphate Coatings,* and *Environmental Stability of Calcium Phosphate Coatings F1926*. An additional standard on anorganic bone (ASTM F4-1581) has also been established within the ceramics subcommittee of ASTM F4.[19]

These national and related international standards (ISO) should provide basic property information for $CaPO_4$ materials and coatings. This information should prove most useful as longer-term investigations on biocompatibility are conducted for dental implant systems. Additionally, national and international standards have been established for the surgical implant alloys, bulk ceramics, and surface finishing of metallic biomaterials.

The concerns related to $CaPO_4$ coatings have focused on (1) the biomechanical stability of the coatings and coating-substrate interface under in vivo conditions of cyclic loading and (2) the biochemical stability of these coatings and interfaces within the gingival sulcus (especially in the presence of inflammation or infection) and during enzymatic process associated with osteoclasis remodeling of the bone-coating interfacial zones. Some of these questions were addressed at an ASTM symposium on $CaPO_4$ coatings, and some researchers related that the longer-term clinical studies (less than 10 years' experience) do not support reasons for concern. It will be interesting to reevaluate these questions and answers after 20 years of clinical experience.

Other Surface Modifications

Surface modification methods include controlled chemical reactions with nitrogen or other elements or surface ion implantation procedures. The reaction of nitrogen with titanium alloys at elevated temperatures results in titanium nitride compounds being formed along the surface. These nitride surface compounds are biochemically inert (like oxides) and alter the surface mechanical properties to increase hardness and abrasion resistance. Most titanium nitride surfaces are gold in color, and this process has been extensively used for enhancing the surface properties of industrial and surgical instruments.[19] Increased hardness, abrasion, and wear resistance can also be provided by ion implantation of metallic substrates. The element most commonly used for surface ion implantation is nitrogen. Electrochemically, the titanium nitrides are similar to the oxides (TiO_2), and no adverse electrochemical behavior has been noted if the nitride is lost regionally. The titanium substrate reoxidizes when the surface layer of nitride is removed. Nitrogen implantation and carbon-doped layer deposition have been recommended to improve the physical properties of stainless steel without affecting its biocompatibility.[239] Again, questions could be raised about coating loss and crevice corrosion.

Surface Cleanliness

A clean surface is an atomically clean surface with no other elements than the biomaterial constituents. Contaminants can be particulates, continuous films (e.g., oil, fingerprints), and atomic impurities or molecular layers (inevitable) caused by the thermodynamic instability of surfaces. Even after reacting with the environment, surfaces have a tendency to lower their energy by binding elements and molecules. The typical composition of a contaminated layer depends on atmospheres and properties of surface. For example, high-energy surfaces (metals, oxides, ceramics) usually tend to bind more to this type of monolayer than polymers and carbon (amorphous).

In the earlier times of dental implantology, no specific protocol for surface preparation, cleaning, sterilization, and handling of the implants was established.[240] Researchers have respectively demonstrated adverse host responses caused by faulty preparation and sterilization, omission to eliminate adsorbed gases, and organic and inorganic debris.[122,123,137,241] According to Albrektsson,[140] implants that seem functional may fail even after years of function, and the cause may be attributed to improper ultrasonic cleaning, sterilization, or handling during the surgical placement.

A systematic study of contamination layers is not available. Lausmaa et al.[124] showed that titanium implants had large variations in carbon contamination loads (20% to 60%) in the 0.3- to 1-nm thickness range, attributed to air exposure and residues from cleaning solvents and lubricants used during fabrication. Trace amounts of Ca, P, N, Si, S, Cl, and Na were noted from other studies.[132,133,241-243] Residues of fluorine could be attributed to passivation and etching treatments; Ca, Na, and Cl to autoclaving; and Si to sand and glass beading processes.

WWW.HIGHDENT.IR

Surface Energy

Measurements of surface property values of an implant's ability to integrate within bone include contact angle with fluids, local pH, and surface topography. These are often used for the determination of surface characteristics. Numerous studies were conducted to evaluate liquid, solid, and air contact angles, wetting properties, and surface tensions as criteria to assess surface cleanliness, because these parameters have been shown to have a direct consequence on osseointegration.[12,244,245] An intrinsically high surface energy is said to be most desirable. High surface energy implants showed a threefold increase in fibroblast adhesion, and higher-energy surfaces such as metals, alloys, and ceramics are best suited to achieve cell adhesion.[12] Surface tension values of 40 dyne/cm and higher are characteristic of very clean surfaces and excellent biological integration conditions.[244] A shift in contact angle (increase) is related to the contamination of the surface by hydrophobic contaminants and decreases the surface tension parameters. Because a spontaneously deposited, host-dependent conditioning film is a prerequisite to the adhesion of any biological element, it is suggested that the wetting of the surface by blood at the time of placement can be a good indication of the high surface energy of the implant.[244]

Passivation and Chemical Cleaning

The ASTM (ASTM B600, ASTM F-86) specifications for final surface treatment of surgical titanium implants require pickling and descaling with molten alkaline base salts. This is often followed by treatment with a solution of nitric or hydrofluoric acid to decrease and eliminate contaminants such as iron. Iron or other elements may contaminate the implant surface as a result of the machining process. This type of debris can have an effect of demineralizing the bone matrix.[246,247] However, these finishing requirements remain very general. Studies of fibroblast attachment on implant surfaces showed great variations, depending on the different processes of surface preparation. Inoue et al.[144] showed fibroblasts developed a capsule or oriented fibrous attachment following the grooves in titanium disks. Contact angles are also greatly modified by acid treatment or water rinsing.[248] Machining operations, polishing, texturing process, residual chemical deposits, and alloy microstructure all inadvertently affect the surface composition. In addition, many ways exist to intentionally modify the surface of the implant. They include conventional mechanical treatment (sand blasting), wet or gas chemical reaction treatment, electroplating or vapor plating, and ion beam processing, which leaves bulk properties intact and has been newly adapted to dentistry from thin film technology. Preliminary studies by Schmidt[249] and Grabowski et al.[250] showed modified fibroblast adhesion on nitrogen and carbon-ion implanted titanium. A general rule has been that cleaner is better.

Sterilization

Manipulation with bare fingers or powdered gloves, tap water, and residual vapor-carried debris from autoclaving can all contaminate implant surfaces. Bauhammers,[240] in an SEM study of dental implants, showed contamination of the surface with acrylic materials, powder for latex gloves, and bacteria. Today, in most cases, the manufacturer guarantees precleaned and presterilized implants with high-technology procedures, with the implants ready to be inserted. If an implant needs to be resterilized, then conventional sterilization techniques are not normally satisfactory. It appears at the present time that no sterilization medium is totally satisfactory for all biomaterials and designs. Metal or alloy constituents, inorganic and organic particles, corrosion products, polymers, and precipitates can be absorbed at the surface throughout the manufacturing, polishing, cleaning, sterilization, packaging, and storage processes. Baier et al.[12] correlated the usual type of contaminant found in relation to the sterilization technique used. Baier et al.[244] showed that steam sterilization can cause deposits of organic substances resulting in poor tissue adhesion. Doundoulakis[132] submitted titanium samples to different sterilization techniques, concluded to the adverse effect of steam sterilization and degradative effect of endodontic glass bead sterilizers, found that dry heat sterilization leaves organic deposits on the surface, and suggested that ultraviolet (UV) light sterilization may become a good alternative after further evaluation. In addition, accelerated oxide growth on titanium may occur with impurity contamination leading to surface discoloration.[32,122,250] In a study by Keller et al.,[252] corrosion products and films from autoclaving, chemicals, and cytotoxic residues from solutions were identified at the surface of implants submitted to sterilization. They suggested that alteration of the titanium surface by sterilization methods may in turn affect the host response and adhesive properties of the implant. On the other hand, Schneider et al.[253] compared the surface of titanium plasma-sprayed and HA-coated titanium implants after steam or ethylene dioxide sterilization using energy dispersive radiograph analysis and concluded that these techniques do not modify the elemental composition of the surface. Keller et al.[254] studied the growth of fibroblasts on disks of CP titanium sterilized by autoclaving, ethylene oxide, ethyl alcohol, or solely passivated with 30% nitric acid and concluded that sterilization seems to inhibit cell growth, whereas passivation does not.

Presently, proteinaceous deposits and their action as films can be best eliminated by radio frequency glow discharge technique (RFGDT), which seems to

WWW.HIGHDENT.IR

be a suitable final cleaning procedure. The implants are treated within a controlled noble gas discharge at very low pressure. The gas ions bombard the surface and remove surface atoms and molecules, which are absorbed onto it or are constituents of it. However, the quality of the surface treated depends on the gas purity. Baier et al.[255] showed that RFGDT is good for cleaning and, at the same time, for granting a high-energy state to the implant, which is related to improved cell adhesion capabilities. Thinner, more stable oxide films and cleaner surfaces have been reported with RFGDT plus improved wettability and tissue adhesion.[255-257] The principal oxide at the surface is unchanged by the RFGDT process.[258] A decrease in bacteria contamination on HA-coated implant surfaces was reported after RFGDT,[259] and studies suggest that RFGDT may enhance calcium and/or phosphate affinity because of an increase in elemental zone at the surface resulting in the formation of amorphous $CaPO_4$ compounds.[257]

Recently, a modified UV light sterilization protocol was shown to enhanced bioreactivity, which was also effective for eliminating some biological contaminants. Singh and Schaaf[260] assessed the quality of UV light sterilization and its effects on irregularly shaped objects, and they established its effectiveness on spores and its ability to safely and rapidly clean the surface and to grant high surface energy. Hartman et al.[261] submitted implants to various pretreatment protocols (RFGDT, UV light, or steam sterilization) and inserted them in miniature swine. Although RFGDT and UV-sterilized implants showed rapid bone ingrowth and maturation, steam-sterilized implants seemed to favor thick collagen fibers at the surface. On the other hand, Carlsson et al.[262] inserted implants in rabbits and compared the performances of conventionally treated implants with implants treated with RFGDT, found similar healing responses, and further cautioned that the RFGDT process produces a much thinner oxide layer at the surface of the implant and may deposit silica oxide from the glass envelope.

Adequate sterilization of clean, prepackaged dental implants and related surgical components has resulted in an ever-expanding use of gamma radiation procedures. Because gamma radiation sterilization of surgical implants is a well-established methodology within the industry, facilities, procedures, and standards are well known. Most metallic systems are exposed to radiation doses exceeding 2.5 Mrad where the packaging and all internal parts of the assembly are sterilized. This is an advantage in that components remain protected, clean, and sterile until the inner containers are opened within the sterile field of the surgical procedure. The healing screws, transfer elements, wrenches, and implants are all exposed to the gamma sterilization, which reduces opportunities for contamination.

Some ceramics can be discolored and some polymers degraded by gamma radiation exposures. The limits are known for classes of biomaterials, and all types of biomaterials can be adequately sterilized within the industry. Systems control, including prepackaging and sterilization, has been an important part of the success of dental implantology.

SUMMARY

In the 1960s dental implantology as a clinical discipline was judged by some to be rather disorganized, and treatments provided were often said to be not as successful as hospital-based orthopedic and cardiovascular surgery procedures. One part of this opinion related to the use of standard intraoral dental materials for implants plus general dental operatories for surgical activities (e.g., no gloves, high-speed drills, tap water). The biomaterials discipline evolved rapidly in the 1970s. Successful uses of synthetic biomaterials have been based on experience within the field of dental implantology. The basis for many of the newer and more clinically successful surgical reconstructions evolved within dentistry, with some now recognized as the most successful types of musculoskeletal reconstructive surgery. The biomaterials discipline therefore has evolved significantly over the past decades, and synthetic biomaterials are now constituted, fabricated, and provided to health care professionals as mechanically and chemically clean devices that have a high predictability of success when used appropriately within the surgical disciplines. This chapter on biomaterials has been separated into sections related to bulk and surface properties of biomaterials, and emphasis has been placed on the published literature on how these biomaterial properties relate to interactions at the tissue interface.

Surface characterization and working knowledge about how surface and bulk biomaterial properties interrelate to dental implant biocompatibility profiles represent an important area in implant-based reconstructive surgery. This chapter has provided summary information on surface and bulk properties for metallic, ceramic, and surface-modified biomaterials. The authors strongly recommend the reference material listed, in addition to a desire to have investigators always provide biomaterial surface and bulk property information as a component of any research studies on tissue response (biocompatibility) profiles.

ACKNOWLEDGMENT

In 1970, Jack E. Lemons was attending his first American and International Associations for Dental Research (AADR/IADR) meeting and was introduced to Ralph Phillips within a group discussion on dental materials. He quickly determined that Lemons knew little about the "dental" and some about the "materials." Phillips included Lemons in the interactions with carefully

WWW.HIGHDENT.IR

placed and directed questions and comments so that he was not excluded. This happened repeatedly over the years, until Lemons had the opportunity to reverse the exchange after making a presentation on behalf of the AADR/IADR Dental Materials Group on basic biocompatibility testing, with Phillips as the overview discussant. This opportunity was to coordinate and help direct some of the emerging exchanges among those experienced in the material and biological sciences. Subsequently, ongoing interactions with Phillips fostered many wonderful times with colleagues, students, and friends throughout the world.

Contents of this chapter represent a later stage, in which Lemons provided written comments and opinions about implant biomaterials as one extension of dental biomaterials. This chapter is dedicated in part to his memory, and most especially to the long-term friendship. The dental implant field, in the author's opinion, will benefit from a continuation of a multidisciplinary approach to the science, technology, and applications. We wish that Ralph could have continued, and we certainly will miss his counsel.

References

1. Williams DF, editor: *Biocompatibility of clinical implant materials,* vol 1, Boca Raton, Fla, 1981, CRC Press.
2. Zierold AA: Reaction of bone to various metals, *Arch Surg* 9:365, 1924.
3. Menegaux GA: Action cytotoxique de quelques metaux sur le tissu osseux cultive en vie ralentie, *Presse Med* 42:1, 1935.
4. Venable CS, Stuck WG, Beach A: The effects on bone of the presence of metals based upon electrolysis, an experimental study, *Ann Surg* 105:917-938, 1939.
5. Ludwigson DC: Today's prosthetic metals, *J Metals* 16, 1964.
6. Williams DF, Roaf R: *Implants in surgery,* London, 1973, WB Saunders.
7. Weissman SL: Models for systemic effects of implants, *Nat Spec Pub* 472:28, 1997.
8. Natiella J, Armitage JJ, Meenaghan M et al: Current evaluation of dental implants, *J Am Dent Assoc* 84:1358, 1972.
9. Rizzo AA, editor: Proceedings of the 1988 Consensus Development Conference on Dental Implants, *J Dent Educ* 52:678-827, 1988.
10. Schnitman PA, Shulman LB, editors: Dental implants: benefit and risk, PHS No 81-1531. In *Proceedings of the Harvard-National Institute of Dental Research Conference,* Boston, 1980.
11. VonRecuum A, editor: *Handbook of biomaterials evaluation,* New York, 1986, Macmillan.
12. Baier R, Meyer A, Natiella J et al: Surface properties determine bioadhesive outcomes, *J Biomed Mater Res* 18:337-355, 1984.
13. Baier R, Shafrin E, Zisman WA: Adhesion: mechanisms that assist or impede it, *Science* 162:1360, 1968.
14. Davies JE, editor: *The bone-biomaterial interface,* Toronto, 1991, University of Toronto Press.
15. Plenk H, Zitter H: Material considerations. In Watzek G, editor: *Endosseous implants: scientific and clinical aspects,* Chicago, 1996, Quintessence.
16. Zitter H, Plenk H: The electrochemical behavior of metallic implant materials as an indicator of their biocompatibility, *J Biomed Mater Res* 21:881-896, 1987.
17. Zwicker U, Breme J, Etzold U: *Titanwerkstoffe mit niedrigem elastizitatsmodul aus gesinterten titanlegierungspulvern, Vortage der 7. Sitzung d. DVM-Arbeitskreises Implantate am 18.11 1986,* Bundesanstalt fur Material prufung, 47-58, Berlin, 1986.
18. Zitter H, Maurer KL, Gather T et al: Implantatwerkstoffe. *Berg und Hüttenmänn Monatshefte* 135: 171-181, 1990.
19. American Society for Testing and Materials: *Surgical and medical devices,* vol 14.01, Philadelphia, 1996, American Society for Testing and Materials.
20. *International Standards Organization, standard references,* Philadelphia, 1996, ANSI-USA.
21. American Dental Association: *Standards,* Chicago, 1996.
22. Williams DF: *Biocompatibility of orthopaedic implants,* vol 1, Boca Raton, Fla, 1982, CRC Press.
23. Ducheyne P, Hastings GW, editors: *Functional behavior of orthopaedic materials,* 2 vols, Boca Raton, Fla, 1984, CRC Press.
24. Till T, Wagner G: Uber elektrochemische untersuchungen an verschiedenen metallischen Zahnreparaturmaterialien, *ZWR* 80:334-339, 1971.
25. Ferguson AB, Laing PG, Hodge ES: The ionization of metal implants in living tissues, *J Bone Joint Surg Am* 42A:77-90, 1960.
26. Mears DC: Electron probe microanalysis of tissues and cells from implant areas, *J Bone Joint Surg* 48B:567, 1966.
27. Geis-Gerstorfer J, Weber H, Sauer KH: In vitro substance loss due to galvanic corrosion in Ti implant/Ni-Cr superconstruction systems, *Int J Oral Maxillofac Implants* 4:119-123, 1989.
28. Jarcho M: Retrospective analysis of hydroxyapatite development for oral implant applications, *Dent Clin North Am* 36:19-36, 1992.
29. Ogus WI: Research report on implantation of metals, *Dent Dig* 57:58, 1951.
30. Laing P: The significance of metallic transfer in the corrosion of orthopaedic screws, *J Bone Joint Surg Am* 40:853-869, 1958.
31. Willert H, Buchhorn G, Semlitsch M: Particle disease due to wear of metallic alloys. In Morrey B, editor: *Biological, material and mechanical considerations of joint replacement,* New York, 1993, Raven Press.
32. Lemons JE: Dental implant retrieval analyses, *J Dent Educ* 52:748-756, 1988.
33. Lemons JE: Metals and alloys for devices in musculoskeletal surgery. In Morrey BF editor: *Joint replacement arthroplasty,* Edinburgh, 1991, Churchill Livingstone.
34. Steinemann S: Tissue compatibility of metals from physico-chemical principles. In Kovaks P, Istephanous N, editors: *Compatibility of biomechanical implants, Electrochem Society, vol 94-15, Conference Proceedings,* San Francisco, 1-14, 1994.
35. Fontana M, Greene N: *Corrosion engineering,* New York, 1967, McGraw-Hill.
36. Williams DF: Titanium as a metal for implantation, *J Med Eng Technol* 1:195-202, 266-270, 1977.
37. Lemons JE: Biomaterial considerations for dental implants. I. Metals and alloys, Alabama Academy of General Dentistry Sponsored Symposium on Dental Implants, *J Oral Implantol* 4:503-515, 1975.
38. Van Orden A, Fraker A, Ruff et al: Surface preparation and corrosion of titanium alloys for surgical implants. In Luckey H, Kublic F, editors: *ASTM STP 796,* Philadelphia, 1981, American Society for Testing and Materials.

WWW.HIGHDENT.IR

39. Geis Gerstorfer J, Weber H: Corrosion resistance of the implant materials contimet 35, memory and Vitallium in artificial physiological fluids, *Int J Oral Maxillofac Implants* 3:135-139, 1988.
40. Skalak R: Biomechanical considerations in osseointegrated prostheses, *J Prosthet Dent* 49:843-848, 1983.
41. Spector M: Biocompatibility in orthopaedic implants. In Williams DF, editor: *Biocompatibility of materials,* Boca Raton, Fla, 1984, CRC Press.
42. Lemons JE, Lucas LC, Johansson B: Intraoral corrosion resulting from coupling dental implants and restorative metallic systems, *Implant Dent* 1:107-112, 1992.
43. Zitter H, Plenk H Jr: The electrochemical behavior of metallic implant materials as an indicator of their biocompatibility, *J Biomed Mater Res* 21:881-896, 1987.
44. Newesely H: Der Stand Der Normung bei Dentalimplantaten, Vortrage des Arbeitskreises Implantate, Berichsband d. 5 Sitzung d. Dtsch Verb f. Materialforschung u prufung eV, 53-55, 1984.
45. *International Standardization Organization technical report 10451: Dental implants—state of the art—survey of materials,* Geneva, 1991.
46. Lautenschlager EP, Sarker NK, Acharaya A et al: Anodic polarization of porous metal fibers, *J Biomed Mater Res* 8:189-191, 1974.
47. Rae T: The biological response to titanium and titanium aluminum vanadium alloy particles, *Biomaterials* 7:3036, 1986.
48. Solar RJ, Pellack SR, Korostoff E: In vitro corrosion testing of titanium surgical implant alloys, *J Biomed Mater Res* 13:217-250, 1979.
49. Hoar TP, Mears DC: Corrosion-resistant alloys in chlorine solution: materials for surgical implants, *Proc R Soc Lond B Biol Sci* A294:486, 1966.
50. Bothe RE, Beaton LE, Davenport HA: Reaction of bone to multiple metallic implants, *Surg Gynecol Obstet* 71: 598-602, 1940.
51. Leventhal GS: Titanium, a metal for surgery, *J Bone Joint Surg* 33:473-474, 1951.
52. Beder OE, Eade G: An investigation on tissue tolerance to titanium metal implants in dogs, *Surgery* 39:470, 1956.
53. Gross PP, Gold L: The compatibility of Vitallium and austanium in completely buried implants in dogs, *Oral Surg* 10:769, 1957.
54. Clarke EG, Hickman J: An investigation into the correlation between the electrical potential of metals and their behavior in biological fluids, *J Bone Joint Surg* 35B:467, 1963.
55. Brettle JA: Survey of the literature on metallic surgical implants, *Injury* 2:26, 1976.
56. Lemons JE, Niemann KMW, Weiss AB: Biocompatibility studies on surgical grade Ti, Co and Fe base alloys, *J Biomed Mater Res* 10:549, 1976.
57. Lemons JE, editor: *Quantitative characterization and performance of porous implants for hard tissue application, ASTM STP 953,* Philadelphia, 1987, American Society for Testing and Materials.
58. Lucas LC, Lemons JE, Lee J et al: *In vitro corrosion characteristics of Co-Cr-Mo/Ti-6Al-4V/Ti alloys.* Paper presented at the American Society for Testing Materials, Symposium on Quantitative Characteristics of Porous Materials for Host Tissues, 1978.
59. Lucas LC, Bearden LF, Lemons JE: Ultrastructural examinations of in vitro and in vivo cells exposed to solutions of 316L stainless steel. In Fraker A, Griffin C, editors: *ASTM STP 859,* Philadelphia, 1985, American Society for Testing and Materials.
60. Van Orden AC: Corrosive response of the interface tissue to 316L stainless steel, titanium base alloys and cobalt base alloys. In McKinney RV, Lemons JE, editors: *The dental implant,* San Diego, 1985, PSG Co.
61. Lang B, Mossie H, Razzoog M: *International workshop: biocompatibility, toxicity and hypersensitivity to alloy systems used in dentistry,* Ann Arbor, Mich, 1985, University of Michigan Press.
62. Steinemann S: Corrosion of surgical implants—in vivo and in vitro tests. In Winter GD, Jeray JL, deGroot K, editors: *Evaluation of biomaterials,* Chichester, England, 1980, Wiley.
63. Steinemann SG, Perren SM, Muller ME: Titanium alloys as metallic biomaterials. In Lutjering G, Zwicker U, Bunk W, editors: Proceedings of the 5th International Conference on Titanium, *Dtsch Gesf Materialkunde eV* 2:1327-1334, 1985.
64. Steinemann SG: Corrosion of titanium and titanium alloys for surgical implants. In Lutering G, Zwicker U, Bunk W, editors: Proceedings of the 5th International Conference on Titanium, *Dtsch Gesf Materialkunde eV* 2:1373-1379, 1985.
65. Hillmann G, Donath K: Licht und elektronemikroskopische untersuchung zur biostabilitat dentaler titanimplantate, *Z Zahnarztl Implantol* 7:170-177, 1991.
66. Schliephake H, Neukam FW, Urban R: Titanbelastung parenchymatoser organe nach insertion von titanschraubenimplantaten, *Z Zahnarztl Implantol* 5:180184, 1989.
67. Schliephake H, Reiss G, Urban R et al: Freisetzung von titan aus schraubenimplantaten, *Z Zahnarztl Implantol* 6:6-10, 1991.
68. Woodman JL, Jacobs JJ, Galante JO et al: Metal ion release from titanium-based prosthetic segmental replacement of long bones in baboons. A long-term study, *J Orthop Res* 1:421-430, 1984.
69. Ferguson AB, Akahoshi Y, Laing PG et al: Characteristics of trace ion release from embedded metal implants in the rabbit, *J Bone Joint Surg* 44:317-336, 1962.
70. Osborn JF, Willich P, Meenen N: The release of titanium into human bone from a titanium implant coated with plasma-sprayed titanium. In Heimke G, Solesz U, Lee AJC, editors: *Advances in biomaterials,* Amsterdam, 1990, Elsevier.
71. Semlitsch M, Staub F, Weber H: Development for biocompatible high strength titanium-aluminum-niobium alloy surgical implants, *Biomed Tech (Berl)* 30:334-339, 1985.
72. Newman JR, Eylon D, Thorne JK: Titanium and titanium alloys. In Stefanescu D, Kurz W, editors: *Metals handbook,* vol 15, ed 9, Metals Park, Ohio, 1988, ASM International.
73. Ott D: Giessen von titan im dentallabor, *Metall* 44: 366-369, 1990.
74. Soom U: Reines titan in der zahnmedizin und zahntechnik: anwendungsbereiche in der implantologie und der prothetik, *Swiss Dent* 8:27-32, 1987.
75. Lemons JE: Surface conditions for surgical implants and biocompatibility, *J Oral Implantol* 7:362-374, 1977.
76. Lucas LC, Buchanan RA, Lemons JE: Investigations on the galvanic corrosion of multialloy total hip prostheses, *J Biomed Mater Res* 15:753-747, 1981.
77. Lucas LC, Buchanan RA, Lemons JE et al: Susceptibility of surgical cobalt-base alloy to pitting corrosion, *J Biomed Mater Res* 16:799-810, 1982.

WWW.HIGHDENT.IR

78. Proceedings of the Third World Biomaterials Congress 11, Society for Biomaterials, Kyoto, Japan, 1988.
79. Brown SA, Lemons JE, editors: *Medical applications of titanium and its alloys: the material and biological issues,* STP 1272, Ann Arbor, Mich, 1996, American Society for Testing and Materials.
80. Bosker H, Kijk L: Het transmandibulaire implantaat, *Ned Tijdschr Tandheelkd* 90:381-389, 1983.
81. Hench LL, Ethridge EC: *Biomaterials, an interfacial approach,* New York, 1982, Academic Press.
82. Vincenzini P, editor: *Ceramics in surgery,* Amsterdam, 1983, Elsevier.
83. Sandhaus S: Nouveaux aspects de l'implantologie, *L'Implant CBS Suisse*, Lausanne, Switzerland, 1969, Sandhaus.
84. Heimke G, Schulte W, d'Hoedt B et al: The influence of fine surface structures on the osseo-integration of implants, *Int J Artif Organs* 5:207-212, 1982.
85. McKinney RV Jr, Lemons JE: *The dental implant*, Littleton, Mass, 1985, PSG Publishing.
86. Steflic D, Sisk A, Parr G et al: HVEM and conventional electron microscopy of interface between bone and endosteal dental implants, *J Biomed Mater Res* 26:529-245, 1992.
87. McKinney R, Steflic D, Koth D et al: The scientific basis for dental implant therapy, *J Dent Educ* 52:696-705, 1988.
88. Koth DL, McKinney RV Jr: The single crystal sapphire endosteal dental implant. In Hardin JF, editor: *Clark's clinical dentistry,* Philadelphia, 1981, JB Lippincott.
89. Horowitz F, Parr J, editors: *Characterization and performance of calcium phosphate coatings for implants,* ASTM STP 1196, Philadelphia, 1994, American Society for Testing and Materials.
90. Brose M et al: Six year evaluation of submerged alumina dental root implants in humans (IADR abstract 56), *J Dent Res* 66:113, 1987.
91. Driskell TS: Development and application of ceramics and ceramic composites for implant dentistry. In Young FA, Hulbert DF, editors: *Materials for implant dentistry,* New York, 1970, Gordon & Breach.
92. deGroot K, editor: *Bioceramics of calcium phosphate,* Boca Raton, Fla, 1983, CRC Press.
93. Ducheyne P, Lemons JE, editors: *Bioceramics: material characteristics versus in vivo behavior,* New York, 1988, New York Academy of Science.
94. Koeneman J: Workshop on characterization of calcium phosphate materials, *J Appl Biomater* 1:79, 1990.
95. LeGeros RZ: Calcium phosphate materials in restorative dentistry: a review, *J Dent Res* 68:164-180, 1988.
96. Yamamuro T, Hench L, Wilson J, editors: *Handbooks of bioactive ceramics,* vols 1 and 2, Boca Raton, Fla, 1990, CRC Press.
97. Kent J et al: Augmentation of deficient edentulous alveolar ridges with dense polycrystalline hydroxylapatite (abstract 3.8.2). In *Final Program and Book of Abstracts*, Vienna, 1980, First World Biomaterials Congress, Society for Biomaterials.
98. dePutter C, deGroot K, Sillevis-Smitt P: Transmucosal apatite implants in dogs, *Trans Soc Biomater* 9:115, 1981.
99. English C: Cylindrical implants, *J Calif Dent Assoc* 16:17-40, 1988.
100. Hench LL, Clark AE: Adhesion to bone. In Williams DF, editor: *Biocompatibility of orthopaedic implants,* vol 2, Boca Raton, Fla, 1982, CRC Press.
101. Ducheyne P, Healy K, Black J et al: *The effects of HA coatings on the metal ion release from porous titanium and Cr-Co alloys.* Paper presented at the *Thirteenth Annual Meeting of the Society for Biomaterials,* San Francisco, Jan 19-22, 1987.
102. Ducheyne P, Martens M: Apatite materials. Clinical and morphological evaluation of custom made bioreactive glass coated canine hip prosthesis, *J Biomed Mater Res* 18:1017-1030, 1984.
103. Ducheyne P, Hench LL, Kagan A et al: Effect of hydroxyapatite impregnation on skeletal bonding of porous coated implants, *J Biomed Mater Res* 14:225-237, 1987.
104. Lemons JE: Hydroxylapatite coatings, *Clin Orthop Relat Res* 235:220-223, 1988.
105. Driessens F: Formation and stability of calcium phosphates in relation to phase composition of the mineral in calcified tissues. In deGroot K, editor: *Bioceramics of calcium phosphates,* Boca Raton, Fla, 1983, CRC Press.
106. Jarcho M: Calcium phosphate ceramics as hard tissue prostheses, *Clin Orthop Relat Res* 157:259-278, 1981.
107. Hjorting-Hansen E, Worsaae N, Lemons LE: Histological response after implantation of porous hydroxylapatite ceramics in humans, *Int J Oral Maxillofac Implants* 5:255, 1990.
108. Lacefield WC: *The coating of hydroxylapatite onto metallic and ceramic implants.* In Proceedings of the Twelfth Annual Meeting of the Society for Biomaterials, St Paul, Minn, 1986.
109. Cook SD et al: Variables affecting the interface strength and histology of hydroxylapatite coated implant surfaces, *Trans Soc Biomater* 9:14, 1986.
110. Lee DR, Lemons J, LeGeros RZ: Dissolution characterization of commercially available hydroxylapatite particulate, *Trans Soc Biomater* 12:161, 1989.
111. Tofe AJ, Watson BA, Bowerman MA: Solution and cell mediated resorption of grafting materials, *J Oral Implantol* 17:345 1991 (abstract).
112. Jarcho M, Bolen CH, Thomas MB et al: Hydroxyapatite synthesis and characterization in dense polycrystalline form, *J Mater Sci Mater Med* 11:2027-2035, 1976.
113. Wang S, Lacefield WR, Lemons JE: Interfacial shear strength and histology of plasma sprayed and sintered hydroxyapatite implants in vivo, *Biomaterials* 17:1965-1970, 1996.
114. Gross U et al: Biomechanically optimized surface profiles by coupled bone development and resorption at hydroxylapatite surfaces, *Trans Soc Biomater* 13:83, 1990.
115. Lemons JE: Ceramics: past, present and future, *Bone* 19(suppl):121S-128S, 1996.
116. Oonishi H, Aoki H, Sawai K: *Bioceramics,* St Louis, 1989, EuroAmerica.
117. Hollinger JO, Battistone GC: Biodegradable bone repair materials, *Clin Orthop Relat Res* 20:290-305, 1986.
118. Lemons JE: Phase boundary interactions for surgical implants. In Rubin LE, editor: *Biomaterials in reconstructive surgery,* St Louis, 1983, Mosby.
119. Andrade JD: The interface between physics, materials, science and biology, *Trans Soc Biomater* 12:6, 1989.
120. Kirsch A: The two phase implantation method using IMZ intramobile cylinder implant, *J Oral Implantol* 11:197-210, 1983.
121. Albrektsson T, Isidor F: Consensus report of session V. In Lang NP, Karring T, editors: *Proceedings of the 1st European Workshop on Periodontology,* London, 1993, Quintessence.
122. Kasemo B: Biocompatibility of titanium implants: surface science aspects, *J Prosthet Dent* 49:832-837, 1983.

WWW.HIGHDENT.IR
مرجع تخصصی دندانسازان و دندانپزشکان

123. Kasemo B, Lausmaa J: Metal selection and surface characteristics. In Brånemark PI, editor: *Tissue integrated prostheses, osseointegration in clinical dentistry,* Chicago, 1985, Quintessence.
124. Lausmaa J, Kasemo B, Mattson H: Surface spectroscopic characterization of titanium implant materials, *Appl Surf Sci* 44:133-146, 1990.
125. Samsonov GV: *The oxide handbook,* New York, 1973, IFI Plenum.
126. Ong JL, Lucas LC, Connatser RW et al: Spectroscopic characterization of passivated titanium in a physiological solution, *J Mater Sci Mater Med* 6:113-119, 1995.
127. Radegran G, Lausmaa J, Rolander U et al: Preparation of ultra-thin oxide windows on titanium for TEM analysis, *J Electron Miscrosc Tech* 19:99-106, 1991.
128. Smith DC, Piliar RM, Chernecky R: Dental implant materials. I. Some effects of preparative procedures on surface topography, *J Biomed Mater Res* 25:1045-1068, 1991.
129. Ask M, Lausmaa J, Kasemo G: Preparation and surface spectroscopic characterization of oxide films Ti6A14V, *Appl Surf Sci* 35:283-301, 1988.
130. Ask M, Rolander U, Lausmaa J et al: Microstructure and morphology of surface oxide films Ti6A14V, *J Mater Res* 5:1662-1667, 1990.
131. Mausli PA, Block PR, Geret V et al: Surface characteristics of titanium and titanium alloys. In Christel P, Meunier A, Lee AJC, editors: *Biological and biochemical performance of biochemicals,* Amsterdam, 1986, Elsevier.
132. Doundoulakis JH: Surface analysis of titanium after sterilization: role in implant-tissue interface and bioadhesion, *J Prosthet Dent* 58:471-478, 1987.
133. Binon P, Weir D, Marshall S: Surface analysis of an original Brånemark implant and three related clones, *Int J Oral Maxillofac Implants* 7:168-175, 1992.
134. Kilpadi DV, Lemons JE: Surface energy characterization of unalloyed titanium implants, *J Biomed Mater Res* 29:1469, 1995.
135. Parr GR, Gardner LK, Toth RW: Titanium: the mystery metal of implant dentistry, dental material aspects, *J Prosthet Dent* 54:410-414, 1985.
136. Anderson G, Gaechter G, Rostoker W: Segmental replacement of long bones in baboons using a fiber implant, *J Bone Joint Surg* 60:31, 1978.
137. Kasemo B, Lausmaa J: Biomaterials and implant surface: on the role of cleanliness, contamination and preparation procedures, *J Biomed Mater Res B Appl Biomater* 22A2:145-158, 1988.
138. Mausli PA, Simpson JP, Burri G et al: Constitution of oxides or titanium alloys for surgical implants. In de Putter C, editor: *Implant materials in biofunction,* Amsterdam, 1988, Elsevier.
139. Smith DC, Pilliar KM, Mattson JB et al: Dental implants materials. II. Preparative procedures and spectroscopic studies, *J Biomed Mater Res* 25:1069-1084, 1991.
140. Albrektsson T: Bone tissue response. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue integrated prostheses—osseointegration in clinical dentistry,* Chicago, 1985, Quintessence.
141. Johansson CB, Albrektsson T, Ericson LE et al: A qualitative comparison of the cell response to commercially pure titanium and Ti6A14V implants in the abdominal wall of rats, *J Mater Sci Mater Med* 3:126-136, 1992.
142. Johansson C, Hansson HA, Albrektsson T: Qualitative interfacial study of bone and tantalum, niobium or commercially pure titanium, *Biomaterials* 11:277, 1990.
143. Johansson C, Lausmaa J, Ask M et al: Ultrastructural differences of the interface zone between bone and Ti-6Al-4V or commercially pure titanium, *J Biomed Eng* 11:3, 1989.
144. Inoue T, Box JE, Pilliar RM et al: The effect of the surface geometry of smooth and porous coated titanium alloy on the orientation of fibroblasts in vitro, *J Biomed Mater Res* 21:107-126, 1987.
145. Brunette DM: The effects of implant surface topography on the behavior of cells, *Int J Oral Maxillofac Implants* 3:231, 1988.
146. Lum L, Beirne O, Dillinges M et al: Osseointegration of two types of implants in non human primates, *J Prosthet Dent* 60:700-705, 1988.
147. Small IA, Helfrick IF, Stines AV: The fixed mandibular implant. In Fonseca RJ, Davis WH, editors: *Reconstructive preposthetic oral and maxillofacial surgery,* ed 2, Philadelphia, 1995, WB Saunders.
148. Smith DC, Piliar RM, McIntyre NS: Surface characteristics of dental implant materials. In Kawahara H, editor: *Oral implantology and biomaterials,* Amsterdam, 1989, Elsevier.
149. Small IA, Misiek D: A sixteen year evaluation of the mandibular stable bone plate, *J Oral Maxillofac Surg* 44:60-66, 1986.
150. Walker C, Aufdemorte TB, McAnear JT et al: The mandibular staple bone plate, a $5^1/_2$ year follow-up, *J Am Dent Assoc* 114:189-192, 1987.
151. Morris HF, Manz MC, Tarolli JH: Success of multiple endosseous dental implant designs to second stage surgery across study sites, *J Oral Maxillofac Surg* 55:76-82, 1997.
152. McQueen D, Sundgren JE, Ivarson B et al: Auger electron spectroscopy studies of titanium implants. In Lee AJC, Albrektsson T, Brånemark PI, editors: *Clinical applications of biomaterials,* New York, 1982, John Wiley & Sons.
153. Sundgren JE, Bodo P, Lundstrom I et al: Auger electron spectroscopic studies of stainless-steel implants, *J Biomed Mater Res* 19:663-671, 1985.
154. Sundgren JE, Bodo P, Lundstrom I: Auger electron spectroscopic studies of the interface between human tissue and implants of titanium and stainless steel, *J Colloid Interface Sci* 110:9-20, 1986.
155. Tengvall P, Elwing H, Sjoqvist L et al: Interaction between hydrogen peroxide and titanium: a possible role in the biocompatibility of titanium, *Biomaterials* 10:118-120, 1989.
156. Tengvall P: *Titanium-hydrogen peroxide interaction with reference to biomaterial applications* [dissertation], Linkoping, Sweden, 1990, University of Linkoping.
157. Tengvall P, Lindstrom I: Physicochemical considerations of titanium as biomaterial, *Clin Mater* 9:115-134, 1992.
158. Hanawa T: Titanium and its oxide film: a substratum for formation of apatite. In Davies JE, editor: *The bone-biomaterial interface,* Toronto, 1991, Toronto University Press.
159. Hanawa T, Ota M: Calcium phosphate naturally formed on titanium in electrolyte solution, *Biomaterials* 12: 767-774, 1991.
160. Jacobs JJ, Skipor AK, Black J et al: Release and excretion of metal in patients who have total hip-replacement component made of titanium-base alloy, *J Bone Joint Surg* 73A:1475-1486, 1991.
161. Lugowski SJ, Smith DC, McHugh AD et al: Release of metal ions from dental implant materials in vivo:

WWW.HIGHDENT.IR

determinations of Al, Co, Cr, Mo, Ni, V, and Ti in organ tissue, *J Biomed Mater Res* 25:1443-1458, 1991.
162. Bruneel N, Helsen JA: In vitro stimulation of biocompatibility of Ti-6A1-4V, *J Biomed Mater Res* 22:203-214, 1988.
163. Ducheyne P, Williams G, Martens M: In vivo metal-ion release from porous titanium fiber material, *J Biomed Mater Res* 18:293-308, 1984.
164. Healy KE, Ducheyne P: The mechanisms of passive dissolution of titanium in a model physiological environment, *J Biomed Mater Res* 26:319-338, 1992.
165. Blumenthal NC, Cosma V: Inhibition of apatite formation by titanium and vanadium ions, *J Biomed Mater Res* 23:13-22, 1989.
166. Solar RJ, Pollack SR, Korostoff E: In vitro corrosion testing of titanium surgical implant alloys, an approach to understanding titanium release from implants, *J Biomed Mater Res* 13:217, 1979.
167. Bundy KJ, Marck M, Hochman RF: In vivo and in vitro studies of stress-corrosion cracking behavior of surgical implant alloys, *J Biomed Mater Res* 17:467, 1993.
168. Jones RL, Wing SS, Syrett BC: Stress corrosion cracking and corrosion fatigue of some surgical implant materials in a physiological saline environment, *Corrosion* 36: 226-236, 1978.
169. Meachim ZG, Williams DF: Changes in non osseous tissue adjacent to titanium implants, *J Biomed Mater Res* 7:555-572, 1973.
170. Rostoker W, Pretzel CW: Coupled corrosion among alloys for skeletal prostheses, *J Biomed Mater Res* 8: 407-419, 1974.
171. Rostoker W, Galante JO, Lereim P: Evaluation of couple crevice corrosion by prosthetic alloys under in vivo conditions, *J Biomed Mater Res* 12:823, 1979.
172. Thompson NG, Buchanan RA, Lemons JE: In vitro corrosion of Ti-6A1-4V and 316 stainless steel when galvanically coupled with carbon, *J Biomed Mater Res* 13:35, 1979.
173. Marshak BL, Ismail Y, Blachere J et al: Corrosion between titanium alloy implant components and substructure alloy, *J Dent Res* 71:723, 1992 (abstract).
174. Marshak BL: *An in vitro study of corrosion at the implant abutment interface* [master's thesis], Pittsburgh, 1994, University of Pittsburgh.
175. Cohen F, Burdairon G: Corrosive properties of odontologic fluoride containing gels against titanium, *J Dent Res* 71:525, 1992 (abstract).
176. Wig P, Ellingsen JE, Videm K: Corrosion of titanium by fluoride, *J Dent Res* 72:195, 1993 (abstract).
177. Rozenbaijer N, Probster L: Titanium surface corrosion caused by topical fluorides, *J Dent Res* 72:227, 1993 (abstract).
178. Liles A, Salkend S, Sarkar N: Galvanic corrosion between titanium and selected crown and bridge alloys, J Dent Res 72:195, 1993 (abstract).
179. Luthy H, Strub JR, Scharer P: Analysis of plasma flame-sprayed coatings on endosseous oral titanium implants exfoliated in man: preliminary results, *Int J Oral Maxillofac Implants* 2:197-202, 1987.
180. Morita M, Hayashi H, Sasada T et al: The corrosion fatigue properties of surgical implants in a living body. In de Putter C et al, editors: *Implant materials in biofunction*, vol 8, Amsterdam, 1988, Elsevier.
181. Sella C, Martin JC, Lecoeur J et al: Biocompatibility and corrosion resistance in biological media of hard ceramic coatings sputter deposited on metal implants, *Mater Sci Eng A Struct Mater* 139:49-57, 1991.
182. Toni A, Lewis CG, Sudanese A et al: Bone demineralization induced by cementless alumina coated femoral stems, *J Arthrop* 9:435-441, 1994.
183. Baro AM, Garcia N, Miranda A et al: Characterization of surface roughness in titanium dental implants measured with scanning tunneling microscopy at atmospheric pressure, *Biomaterials* 17:463-467, 1986.
184. Olin H, Aronssoid BO, Kasemo B et al: Scanning tunneling microscopy of oxidized titanium surfaces in air, *Ultramicroscopy* 42-44:567-571, 1992.
185. Niznick GA: The core-vent implant system, *J Oral Implantol* 10:379, 1982.
186. Niznick GA: Comparative surface analysis of Brånemark and core-vent implants, *J Oral Maxillofac Surg* 45, 1987.
187. Cochran DL, Nummikoski PV, Higginbottom FL et al: Evaluation of an endosseous titanium implant with a sandblasted and acid etched surface in the canine mandible: radiographic results, *Clin Oral Implants Res* 7:240-252, 1996.
188. Clarke A: Particulate debris from medical implants. In St. John KR editor: *ASTM STP 1144*, Philadelphia, 1992, American Society for Testing and Materials.
189. Ricci JL, Kummer FJ, Alexander H et al: Embedded particulate contaminants in textured metal implant surfaces, *J Appl Biomater* 3:225-230, 1992.
190. *Technical data on the RBM Surface roughening treatment*, Southfield, Mich, 1996, Bio-Coat Inc.
191. Hermann H: Plasma spray deposition processes, *MRS Bull* 60-67, 1988.
192. Hahn H, Palich W: Preliminary evaluation of porous metal surfaced titanium for orthopaedic implants, *J Biomed Mater Res* 4:571-577, 1970.
193. Karagianes MT, Westerman RE: Development and evaluation of porous ceramic and titanium alloy dental anchors implanted in miniature swine, *J Biomed Mater Res Symp* 8:391-399, 1974.
194. Schroeder A, Van der Zypen E, Stich H et al: The reactions of bone, connective tissue and epithelium to endosteal implants with titanium sprayed surfaces, *J Maxillofac Surg* 9:15-25, 1981.
195. Young FA, Spector M, Kresch CH: Porous titanium endosseous dental implants in rhesus monkeys: microradiography and histological evaluation, *J Biomed Mater Res* 13:843-856, 1979.
196. Deporter DA, Watson PA, Pilliar RM et al: A histological assessment of the initial healing response adjacent to porous surfaced titanium alloy dental implants in dogs, *J Dent Res* 5:1064-1070, 1986.
197. Deporter DA, Watson PA, Pilliar RM et al: A histological evaluation of a functional endosseous, porous-surfaced, titanium alloy dental implant in the dog, *J Dent Res* 67:1990-1995, 1988.
198. Deporter DA, Watson PA, Pilliar RM et al: A histological comparison in the dog of porous-coated versus threaded dental implant, *J Dent Res* 69:1138-1145, 1990.
199. Pilliar RM, Deporter DA, Watson PA et al: Dental implant design effect on bone remodeling, *J Biomed Mater Res* 25:467-483, 1991.
200. Deporter DA, Watson PA, Pilliar RM et al: A prospective clinical study in humans of an endosseous dental implant partially covered with a powder-sintered porous coating: 3 to 4 year results, *Int J Oral Maxillofac Implants* 11:87-95, 1996.

WWW.HIGHDENT.IR

201. Lowenberg BF, Pilliar RM, Aubin JE et al: Migration, attachment and orientation of human gingival fibroblasts to root slices, naked and porous surfaces titanium alloy discs and zircalloy discs in vitro, *J Dent Res* 66:1000-1005, 1987.
202. Clemow AJT, Weinstein AM, Klawitter JJ et al: Interface mechanics of porous titanium implants, *J Biomed Mater Res* 1:73-82, 1981.
203. Hulbert SF, Morrison S, Klawitter JJ: Tissue reactions to three ceramics of porous and nonporous structures, *J Biomed Mater Res* 6:347-374, 1972.
204. Hulbert SF, Cooke FW, Klawitter JJ et al: Attachment of prostheses to the musculoskeletal system by tissue ingrowth, *J Biomed Mater Res* 7:1-23, 1973.
205. Bobyn JD, Pilliar RM, Cameron HU et al: The optimum pore size for the fixation of porous surfaced metal implants by the ingrowth of bone, *Clin Ortop Relat Res* 150:263-270, 1980.
206. Predecki P, Auslaender BA, Stephan JE et al: Attachment of bone to threaded implants by ingrowth and mechanical interlocking, *J Biomed Mater Res* 6:401-412, 1972.
207. Claes L, Hutzschenreutet O, Pholer V et al: Lose-momente von corticaliszugschrauben in abhangigkeit von implantationszeit und oberflachebeschaffenheit, *Arch Orthop Unfallchir* 85:155-159, 1976.
208. Proceedings of an International Congress, Brussels. Tissue integration. In Van Steenberghe D, editor: *Oral and maxillofacial reconstruction*, Amsterdam, 1985, Excerpta Medica.
209. Maniatopoulos C, Pilliar RM, Smith DC: Threaded vs porous surface designs for implant stabilization in bone-endodontic implant model, *J Biomed Mater Res* 20: 1309-1333, 1986.
210. Pilliar RM, Lee J, Maniatopoulos C: Observations on the effect of movement on bone in growth into porous surface implants, *Clin Orthop* 208:108-113, 1986.
211. Moser W, Nentwig GH: Zur problematik von titan-spritzbeschich-tungen, *Z Zahnarztl Implantol* 3:282-285, 1987.
212. Watzek G, Danhel-Mayhauser M, Matejka M et al: *Experimental comparison of Brånemark and TPS dental implants in a sheep model, abstract 50.* Paper presented at the UCLA Symposium on Implants in the Partially Edentulous Patient, Palm Springs, Calif, 1990.
213. American Society for Testing and Materials: *Standard specification for titanium and titanium 6Al4V alloy powders for coating of surgical implants*, ASTM F 1580, Philadelphia, 1995, American Society for Testing and Materials.
214. Kay JF, Jarcho M, Logan G et al: *The structure and properties of HA coatings on metal.* In Proceedings of the Twelfth Annual Meeting of the Society for Biomaterials, 1986.
215. Block MS, Kent JN, Kay JF: Evaluation of hydroxylapatite-coated titanium dental implants in dogs, *J Oral Maxillofac Surg* 45:601-607, 1987.
216. Thomas KA, Kay JF, Cook SD et al: Effect of surface microtexture and hydroxylapatite coating on the mechanical strengths and histologic profiles of titanium implant materials, *J Biomed Mater Res* 21:1395-1414, 1987.
217. Griffin CD, Kay JF, Smith CL: *The effect of hydroxylapatite coatings on corrosion of Co-Cr alloy.* Proceedings of the Thirteenth Annual Meeting of the Society for Biomaterials, San Francisco, 1987.
218. Cook SD, Kay JF, Thomas KA et al: Interface mechanics and histology of titanium and HA coated titanium for dental implant applications, *Int J Oral Maxillofac Implants* 2:15-22, 1987.
219. Geesink RGT, deGroot K, Klein CPAT: Bonding of bone to apatite coated implants, *J Bone Joint Surg* 70B:17-22, 1988.
220. Thomas KA, Jay JF, Cook SD et al: The effect of surface macrotexture and hydroxylapatite coating on the mechanical strengths and histologic profiles of titanium implant materials, *J Biomed Mater Res* 21:1395-1414, 1987.
221. Meffert RM, Block MS, Kent JN: What is osseointegration? *Int J Periodontics Restorative Dent* 4:9-21, 1987.
222. Block MS, Finger IM, Fontenot MG et al: Loaded hydroxylapatite coated and grit blasted titanium implants in dogs, *Int J Oral Maxillofac Implants* 4:219-224, 1989.
223. deGroot K, Geesink R, Klien C et al: Plasma sprayed coatings of hydroxyapatite, *J Biomed Mater Res* 21:1375-1381, 1987.
224. Kent JN, Block MS, Finger IM et al: Biointegrated hydroxylapatite coated dental implants. 5 year observations, *J Am Dent* 121:138-144, 1990.
225. Jensen R, Jensen J, Krauser JT et al: Hydroxylapatite coated dental implants, *New Dent* 68:14-25, 1989.
226. Kaufmann J, Ricci JL, Jaffe W et al: *Bone attachment to chemically textured titanium alloy with and without HA coating.* Proceedings of the Twenty-Third Annual Meeting of the Society for Biomaterials, New Orleans, La, 1997.
227. Wolke JGC, deGroot K, Jansen JA: *Initial wound healing around subperiosteal RF magnetron sputtered Ca-P implants.* Proceedings of the Twenty-Third Annual Meeting of the Society for Biomaterials, New Orleans, La, 1997.
228. Kim YK, Kim S, Lee JH: *Interfacial shear strength of laser treated hydroxylapatite layer on titanium alloy.* Proceedings of the twenty-third annual meeting of the Society for Biomaterials, New Orleans, La, 1997.
229. Gerner BT, Barth E, Albrektsson T et al: Comparison of bone reactions to coated tricalcium phosphate and pure titanium dental implants in the canine iliac crest, *Scand J Dent Res* 96:143-148, 1988.
230. Kay JF: Calcium phosphate coatings for dental implants current status and future potential, *Dent Clin North Am* 36:1-18, 1992.
231. Klien CPAT, deGroot K: Histology of hydroxylapatite coatings, *J Dent Res* 68:863, 1989.
232. Meffert RM: How to treat ailing and failing implants, *Implant Dent* 1:25-33, 1992.
233. Caulier H, Vercaigne S, Naert I et al: The effect of Ca-P plasma sprayed coatings on the initial bone healing of oral implants and experimental study in the goat, *Biomed Mater Res* 34:121-128, 1997.
234. Ducheyne P, Healy KE: The effect of plasma sprayed calcium phosphate ceramic coatings on the metal ion release from porous titanium and cobalt chrome alloys, *J Biomed Mater Res* 22:1127-1163, 1988.
235. Filiggi MJ, Coombs NA, Pilliar RM: Characterization of the interface in the plasma sprayed HA coating/Ti6A14V implant system, *J Biomed Mater Res* 25:1211-1229, 1991.
236. Tufecki E, Brantley WA, Mitchell JC et al: Microstructures of plasma sprayed HA coated Ti6A14V dental implants, *Int J Oral Maxillofac Implants* 12:25-31, 1997.
237. Lewandrowski JA, Johnson CM: Structural failure of osseointegrated implants at the time of restoration, a clinical report, *J Prosthet Dent* 62:127-129, 1989.
238. Krauser J, Berthold P, Tamary I et al: A scanning electron microscopy study of failed endosseous root formed dental implants, *J Dent Res* 70:274, 1991 (abstract).
239. Bordji K, Jouzeau JY, Mainand D et al: Evaluation of the effect of three surface treatments on the biocompatibility

WWW.HIGHDENT.IR

of 316 stainless steel using human differentiated cells, *Biomaterials* 17:491-500, 1996.
240. Baumhammers A: Scanning electron microscopy of contaminated titanium implants following various cleaning techniques, *J Oral Implantol* 6:202-209, 1975.
241. Baier RE, Meyer AE, Akers CK et al: Degradative effects of conventional steam sterilization on biomaterial surfaces, *Biomaterials* 3:241-244, 1982.
242. Grobe GL, Baier RE, Gardella J et al: *Relation of metal interface properties to broad adhesive success.* Paper presented at The Third International Conference on Environment Degradation of Engineering Materials, Pennsylvania State University, 1987.
243. Smith DC: Surface characterization of implant materials. Biological implication. In Davies JB, editor: *The bone biomaterial interface,* Toronto, 1991, Toronto University Press.
244. Baier RE, Meyer AE: Implant surface preparation, *Int J Oral Maxillofac Implants* 3:3-19, 1988.
245. Baier RE, Meenaghan MA, Hartman LC et al: Implant surface characteristics and tissue interaction, *J Oral Implantol* 13:594-605, 1988.
246. American Society for Testing and Materials: *B 600-74, standard recommended practice for descaling and cleaning titanium and titanium alloy surfaces,* Philadelphia, 1985, American Society for Testing and Materials.
247. American Society for Testing and Materials: *F-86-84: standard practice for surface preparation and marking of metallic surgical implants,* Philadelphia, 1985, American Society for Testing and Materials.
248. Keller JC, Stanford CM, Wightman JP et al: Characterization of commercially pure titanium surfaces, *J Biomed Mater Res* 28:939-946, 1994.
249. Schmidt FA: Surface modification by ion implantation, *Naval Res Lab Rev* 69-79, 1985.
250. Grabowski KS, Gossett CR, Young FA et al: *Cell adhesion to ion implanted surfaces.* Proceedings of the Materials Research Society Symposium, 1987.
251. Lausmaa J, Kasemo B, Hannson S: Accelerated oxide growth on titanium implants during autoclaving caused by fluorine contamination, *Biomaterials* 6:23-27, 1985.
252. Keller JC, Draughn RA, Wightman JP et al: Characterization of sterilized commercially pure titanium surfaces, *Int J Oral Maxillofac Implants* 5:360-366, 1990.
253. Schneider R, Olson RA, Krizan KB: Sterilization effects on the surface of coated implants [abstract], *J Dent Res* 68, 1989.
254. Stanford CM, Keller JC, Solvrsh M: Bone cell expression on titanium surfaces is altered by sterilization treatments, *J Dent Res* 73:1061-1071, 1994.
255. Baier RE, Glantz PO: Characterization of oral in vivo films formed on different types of solid surfaces, *Acta Odontol Scand* 36:289-301, 1988.
256. Baier RE: *Improved passivation of implantable biomaterials by glow discharge process.* Paper presented at the Surfaces in Biomaterials Symposium, Minneapolis, Minn, Oct 9-11, 1991.
257. Walivaraza B, Aronsson BO, Rodahl M et al: Titanium with different oxides in vitro studies of protein adsorption and contact activation, *Biomaterials* 15:827-834, 1994.
258. Kawahara D, Ong JL, Raikar GN et al: Surface characterization of radio frequency glow discharged and autoclaved titanium surfaces, *Int J Oral Maxillofac Implants* 11:435-442, 1996.
259. Moreira M: *Radio frequency glow discharge treatment as an alternative for cleaning and sterilizing dental implants* [master's thesis], Birmingham, Ala, 1993, University of Alabama.
260. Singh S, Schaaf NG: Dynamic sterilization of titanium implants with UV light, *Int J Oral Maxillofac Implants* 4:139-146, 1989.
261. Hartman LC, Meenaghan MA, Schaaf MG et al: Effects of pretreatment sterilization and cleaning methods on materials properties and osseoinductivity of a threaded implant, *Int J Oral Maxillofac Implants* 4:11-18, 1989.
262. Carlsson LV, Albrektsson T, Berman C: Bone response to plasma cleaned titanium implants, *Int J Oral Maxillofac Implants* 4:199-204, 1989.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 25

Clinical Biomechanics in Implant Dentistry

Martha Warren Bidez, Carl E. Misch

The discipline of biomedical engineering, which applies engineering principles to living systems, has unfolded a new era in diagnosis, treatment planning, and rehabilitation in patient care. One aspect of this field, biomechanics, concerns the response of biological tissues to applied loads. Biomechanics uses the tools and methods of applied engineering mechanics to search for structure-function relationships in living materials.[1] Advancements in prosthetic, implant, and instrumentation design have been realized because of mechanical design optimization theory and practice.[2] This chapter provides fundamental concepts and principles of dental biomechanics as they relate to long-term success of dental implants and restorative procedures.

LOADS APPLIED TO DENTAL IMPLANTS

Dental implants are subjected to occlusal loads when placed in function. Such loads may vary dramatically in magnitude, frequency, and duration, depending on the patient's parafunctional habits. Passive mechanical loads also may be applied to dental implants during the healing stage because of mandibular flexure, contact with the first-stage cover screw, and second-stage permucosal extension.

Perioral forces of the tongue and circumoral musculature may generate low but frequent horizontal loads on implant abutments. These loads may be of greater magnitude with parafunctional oral habits or tongue thrust. Finally, application of nonpassive prostheses to implant bodies may result in mechanical loads applied to the abutment, even in the absence of occlusal loads.

So many variables exist in implant treatment that it becomes almost impossible to compare one treatment philosophy with another. However, basic units of mechanics may be used to provide the tools for the consistent description and understanding of such physiologic (and nonphysiologic) loads. Two different approaches may render a similar short-term result; however, a biomechanical approach still can determine which treatment renders more risk over the long term.

MASS, FORCE, AND WEIGHT

Mass, a property of matter, is the degree of gravitational attraction the body of matter experiences. As an example, consider two cubes composed of hydroxyapatite (HA) and commercially pure titanium, respectively. If the two cubes are restrained by identical springs, then each spring will deflect by a certain amount relative to the attraction of gravity for the two cubes. The two spring deflections in this example can be made equal by removing part of the material from the titanium cube. Even though the cubes are of completely different composition and size, they can be made equivalent with respect to their response to the pull of gravity. This innate property of each cube that is related to the amount of matter in physical objects is referred to as *mass*. The unit of mass in the metric (International System of Units) system is the kilogram (kg); in the English system, it is the pound mass (lbm).[3]

In 1687, Sir Isaac Newton described a force in what is now referred to as *Newton's laws of motion*.[3] In his second law, Newton stated that the acceleration of a body is inversely proportional to its mass and directly proportional to the force that caused the acceleration. The familiar relation expresses this law:

$$F = ma$$

where F is force (newtons [N]), m is mass (kilograms), and a is acceleration (meters per second squared [m/sec^2]). In the dental implant literature, force commonly is expressed as kilograms of force. The gravitational constant (a = 9.8 m/sec^2) is approximately the same at every location on Earth; therefore mass (kilograms) is the determining factor in establishing the magnitude of a static load.

Weight is simply a term for the gravitational force acting on an object at a specified location. Weight and force can be expressed by the same units, newtons or pound force (lbf). If a titanium cube is considered as though placed on the moon, then its weight (force caused by gravity) is different from its weight on the Earth. The mass in the cube has not changed, but the

WWW.HIGHDENT.IR

acceleration caused by gravity has changed. Recalling Sir Isaac Newton's work, an apple weighs approximately 1 N (0.225 lbf). The reader will find the conversion factors in Box 25-1 useful.[4]

Box 25-1 Useful Conversion Factors

Mass

1 kg = 2.205 lbm
1 lbm = 0.45 kg

Force

1 N = 1 kg(m/s^2) = 0.225 lbf
1 lbf = 4.448 N

Area

1 m^2 = 10.764 sq ft
1 sq ft = 0.093 m^2
1 sq in = 6.452 × 10^{-4} m^2

Pressure*

1 lbf/sq in (psi) = 144 lbf/sq ft = 6894.8 Pa = 6.89 kPa = 0.0069 MPa
1 Pa = 1 N/m^2 = 1.450 x 10^{-4} psi = 0.021 lbf/sq ft

*Stress uses these same units of measurement.

FORCES

Forces may be described by magnitude, duration, direction, type, and magnification factors. Forces acting on dental implants are referred to as *vector quantities*; that is, they possess magnitude and direction. Restated, to state simply that "a force of 75 lb exists on the distal abutment" is not sufficient. The more correct statement is "a force of 75 lb exists on the distal abutment directed axially along the long axis of the implant body." The dramatic influence of load direction on implant longevity and bone maintenance is discussed later in this chapter and others. Typical maximum bite force magnitudes exhibited by adults are affected by age, sex, degree of edentulism, bite location, and especially parafunction[5-9] (Table 25-1).

A force applied to a dental implant rarely is directed absolutely longitudinally along a single axis. In fact, three dominant clinical loading axes exist in implant dentistry: (1) mesiodistal, (2) faciolingual, and (3) occlusoapical (Figure 25-1). A single occlusal contact most commonly results in a three-dimensional occlusal force. Importantly, this three-dimensional force may be described in terms of its component parts (fractions) of the total force that are directed along the other axes. For example, if an occlusal scheme on an implant restoration is used that results in a large magnitude of force component directed along the faciolingual axis (lateral loading), then the implant is at extreme risk for fatigue failure (described later in this chapter). The process by which three-dimensional forces are broken down into their component parts is referred to as *vector resolution* and may be used routinely in clinical practice for enhanced implant longevity.

Table 25-1 Maximum Bite Force

REFERENCE	AGE (YR)	NUMBER	INCISOR	CANINE	PREMOLAR	MOLAR	COMMENTS
Braun et al.*	26-41	142				710 N	Between premolar and molar; male subjects 789 N; female subjects 596 N
van Eijden†	31.1 (±4.9)	7		323-485 N	424-583 N	475-749 N	Second premolar and second molar, left and right (male subjects only)
Dean et al.‡	Adult	57	150 N			450 N	Converted from figures
Bakke et al.§	21-30	20				572 N	Measured in left and right first molar
	31-40	20				481 N	
	41-50	20				564 N	
	51-60	17				485 N	
	61-70	8				374 N	
Braun et al.‖	18-20					176 N	First molar or first premolar

* Braun S, Bantleon H-P, Hnat WP et al: A study of bite force. 1. Relationship to various physical characteristics, *Angle Orthod* 65:367-372, 1995.
† van Eijden TMGJ: Three-dimensional analyses of human bite-force magnitude and moment, *Arch Oral Biol* 36:535-539, 1991.
‡ Dean JS, Throckmorton GS, Ellis EE et al: A preliminary study of maximum voluntary bite force and jaw muscle efficiency in preorthognathic surgery patients, *J Oral Maxillofac Surg* 50: 1282-1288, 1992.
§ Bakke M, Holm B, Jensen L et al: Unilateral, isometric bite force in eight- to eighty-eight year old women and men related to occlusal factors, *Second J Dent Res* 98: 149-158, 1990.
‖ Braun S, Hnat WP, Freudenthaler JW et al: A study of maximum bite force during growth and development, *Angle Orthod* 66: 261-264, 1996.

WWW.HIGHDENT.IR

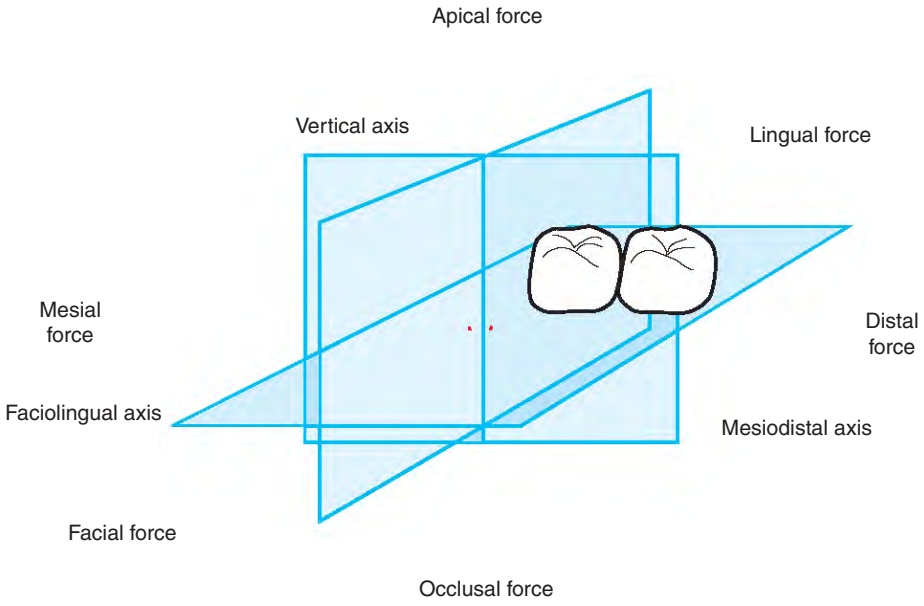


Figure 25-1 Forces are three-dimensional, with components directed along one or more clinical coordinate axes: mesiodistal, faciolingual, and occlusoapical (vertical).

TYPE OF FORCE APPLIED	STRENGTH (MPa)*	LOAD DIRECTION/ COMMENTS
Compressive	193.0 (13.9)	Longitudinal
	173.0 (13.8)	30 degrees off axis
	133.0 (15.0)	60 degrees off axis
	133.0 (10.0)	Transverse
Tensile	133.0 (11.7)	Longitudinal
	100.0 (8.6)	30 degrees off axis
	60.5 (4.8)	60 degrees off axis
	51.0 (4.4)	Transverse
Shear	68.0 (3.7)	Torsion

From Reilly DT, Burstein AH: The elastic and ultimate properties of compact bone tissue, *J Biomech* 8:393, 1975.
*Standard deviations are listed in parentheses.

Components of Forces (Vector Resolution)

Occlusion serves as the primary determinant in establishing load direction. The position of occlusal contacts on the prosthesis directly influences the type of force components distributed throughout the implant system.

The dentist should visualize each occlusal contact on an implant restoration in its component parts. Consider the example of a restored dental implant subjected to a premature contact during occlusion. When the contact is broken down into its component parts directed along the three clinical loading axes, a large, potentially dangerous lateral component is observed. Occlusal adjustments consistent with implant protective occlusion to eliminate the premature contact minimize the development of such dangerous load components.

Angled abutments also result in development of dangerous transverse force components under occlusal loads in the direction of the angled abutment. Implants should be placed surgically to provide for mechanical loading down the long axis of the implant body to the maximum extent possible. Angled abutments are used to improve esthetics or the path of insertion of a restoration, not to determine the direction of load.

Three Types of Forces

Forces may be described as compressive, tensile, or shear. Compressive forces attempt to push masses toward each other. Tensile forces pull objects apart. Shear forces on implants cause sliding. Compressive forces tend to maintain the integrity of a bone-implant interface, whereas tensile and shear forces tend to distract or disrupt such an interface. Shear forces are most destructive to implants and bone compared with other load modalities. In general, compressive forces are accommodated best by the complete implant-prosthesis system. Cortical bone is strongest in compression and weakest in shear[10] (Table 25-2). Additionally, cements and retention screws, implant components, and bone-implant interfaces all accommodate greater compressive forces than tensile or shear. For example, whereas the compressive strength of an average zinc-phosphate dental cement is 83 to 103 MPa (12,000 to 15,000 psi), the resistance to tension and shear is significantly less (500 psi) (Figure 25-2).

The implant body design transmits the occlusal load to the bone. Threaded or finned dental implants impart a combination of all three force types at

WWW.HIGHDENT.IR

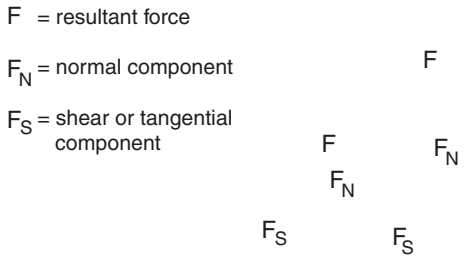


Figure 25-2 Force can be resolved into a combination of normal and shear force components in a given plane. Depending on the direction of load application, the same magnitude of force has different effects.

the interface under the action of a single occlusal load. This "conversion" of a single force into three different types of forces is controlled completely by the implant geometry. The prevalence of potentially dangerous tensile and shear forces in threaded or finned implants may be controlled optimally through careful engineering design. Cylinder implants in particular are at highest risk for harmful shear loads at the implant-tissue interface under an occlusal load directed along the long axis of the implant body. As a consequence, cylinder implants require a coating to manage the shear stress at the interface through a more uniform bone attachment along the implant length. Bone loss adjacent to cylindrical implants and coating degradation result in a mechanically compromised implant.

Offset loading on single-tooth or multiple-abutment restorations results in moment (bending) loads (described later under Force Delivery and Failure Mechanisms). As a result, an increase in tensile and shear force components is often found. Compressive forces typically should be dominant in implant prosthetic occlusion.

Multiple abutment restorations, particularly with distal cantilevers, produce a remarkably complex load profile in the prosthesis and in the bone-implant interface. These clinical realities underscore the need for optimizing dental implant design to provide the maximum functional surface area to dissipate such forces.

Stress

The manner in which a force is distributed over a surface is referred to as *mechanical stress*. Thus the familiar relation defines stress:

$$\sigma = F/A$$

where σ is stress (pounds per square inch; pascals), F is force (newtons; pound force), and A is area (square inches; square meters). The internal stresses that develop in an implant system and surrounding biological tissues under an imposed load may have a significant influence on the long-term longevity of the implants in vivo. As a general rule, a goal of treatment planning should be to minimize and evenly distribute mechanical stress in the implant system and the contiguous bone.

The magnitude of stress depends on two variables: (1) force magnitude and (2) cross-sectional area over which the force is dissipated. It is rare that a dentist can control

	Biomaterial				
				Co ALLOY (WROUGHT)	
PROPERTY	Ti (WROUGHT)	Ti-Al-V (WROUGHT)	Co-Cr-Mo (CAST)	ANNEALED	COLD WORKED
Density (g/mL)	—	4.5	8.3	9.2	9.2
Hardness (Vickers)	R_b100	—	300	240	450
Yield strength					
MPa	170-480	795-827	490	450	1050
	(25-70)	(115-120)	71	(62)	(152)
Ultimate tensile strength					
MPa	240-550	860-896	690	950	1540
	(35-80)	(125-130)	(100)	(138)	(223)
Elastic modulus					
GPa	96	105-117	200	230	230
(psi × 10^3)	(14)	(15-17)	(29)	(34)	(34)
Endurance limit (fatigue)					
Mpa	—	170-240	300	—	240-490
(psi × 10^3)		(24.6-35)	(43)		(35-71)
Elongation %	15-24	10-15	8	30-45	9

WWW.HIGHDENT.IR

the force magnitude completely. The magnitude of the force may be decreased by reducing these significant magnifiers of force: cantilever length, offset loads, and crown height. Night guards to decrease nocturnal parafunction; occlusal materials that decrease impact force; and overdentures, rather than fixed prostheses, that can be removed at night are further examples of force reduction strategies. The functional surface area over which the force is distributed, however, is controlled completely through careful treatment planning.

A *functional cross-sectional* area is defined as that surface that participates significantly in load bearing and stress dissipation. This area may be optimized by (1) increasing the number of implants for a given edentulous site and (2) selecting an implant geometry that has been designed carefully to maximize functional cross-sectional area. An increase in functional surface area serves to decrease the magnitude of mechanical stress imposed on the prosthesis, implant, and biological tissues.

Stress components are described as normal (perpendicular to the surface and given the symbol σ) and shear (parallel to the surface and given the symbol τ). One normal stress and two shear stresses act on each plane (x, y, z); therefore $\tau_{xy} = \tau_{yx}$, $\tau_{yz} = \tau_{zy}$, and $\tau_{xz} = \tau_{zx}$. Thus any three-dimensional element may have its stress state completely described by three normal stress components and three shear components.

The question arises as to what are the peak stresses or maximum stresses that an implant and the surrounding interfacial tissues experience. Peak stresses occur when the stress element is positioned in a particular orientation (or geometric configuration) in which all shear stress components are zero. When an element is in this configuration, the normal stresses are given a particular name, *principal stresses,* and are indicated as σ_1, σ_2, and σ_3. By convention, maximum principal (σ_1) stresses represent the most positive stresses (typically peak tensile stresses) in an implant or tissue region and minimum principal (σ_3) stresses, the most negative stresses (typically peak compressive stresses). Sigma 2 (σ_2) represents a value intermediate between σ_1 and σ_3. Determination of these peak normal stresses in a dental implant system and tissues may give valuable insights regarding sites of potential implant fracture and bone atrophy.

Deformation and Strain

A load applied to a dental implant may induce deformation of the implant and surrounding tissues. Biological tissues may be able to interpret deformation or a manifestation thereof and respond with the initiation of remodeling activity.

The deformation and stiffness characteristics of the materials used in implant dentistry, particularly the implant materials, may influence interfacial tissues, ease of implant manufacture, and clinical longevities. Elongation (deformation) of biomaterials used for surgical dental implants ranges from 0 for aluminum oxide ceramics to up to 55 for annealed 316L stainless steel[11] (Table 25-3). Related to deformation is the concept of strain, a parameter believed to be a key mediator of bone activity.

Under the action of a tensile force (F), the straight bar (of original gauge length, l_0) undergoes elongation

Biomaterial							
Fe-Cr-Ni 316-L			Al_2O_3				
ANNEALED	COLD WORKED	C-Si	SAPPHIRE	ALUMINA	UHMW POLYETHYLENE	PMMA	PTFE
7.9	7.9	1.5-2.0	3.99	3.9	0.94	1.2	2.2
170-200	300-350	—	—	HV23.000	D65	M60-100	D50-65
240-300 (35-44)	700-800 (102-116)	—	—	—	—	—	—
600-700 (87-102)	1000 (145)	350-517 (51-75)	480 (70)	400 (58)	21-44 (3.0-6.4)	55-85 (8.0-12.3)	14-34 (2-5)
200 (29)	200 (29)	28-34 (4.0-4.9)	414 (60)	380 (55.1)	1 (0.145)	2.4–3.3 (0.3480-0.479)	0.4 (0.058)
300 (43)	230-280 (33.3-40.6)	—	—	—	—	—	—
35-55	7-22	0	0	0	400	2-7	200-400

From Lemons JE, Bidez MW: Biomaterials and biomechanics in implant dentistry. In McKinney RV, editor: *Endosteal dental implants,* St Louis, 1991, Mosby.
UHMW, Ultrahigh molecular weight polyethylene; *PMMA,* polymethylmethacrylate; *PTFE,* polytetrafluoroethylene.

WWW.HIGHDENT.IR

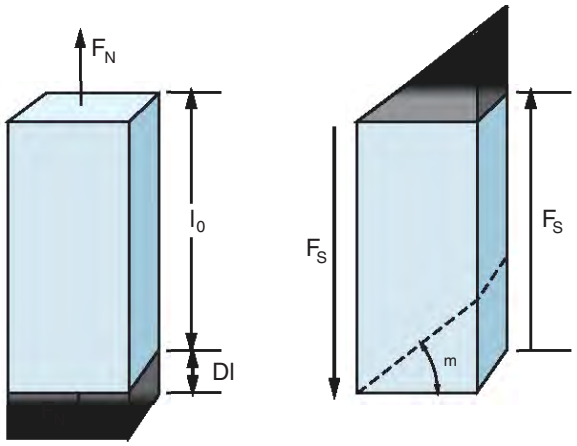


Figure 25-3 Under action of tensile force (F_N), the straight bar originally l_0 is elongated by an amount Δl. Engineering strain ε is the deformation per unit length. Shear strain γ is the change in a right angle of a body or stress element under action of a pure shearing load (F_S).

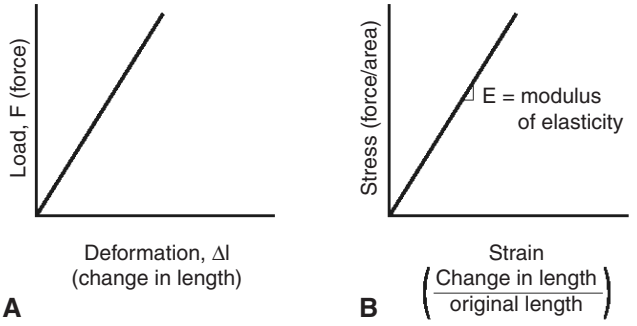


Figure 25-4 **A,** Load-versus-deformation curve may be generated for any elastic body experimentally subjected to a load. **B,** Dividing load values by the surface area and the deformation of the original gauge length of the specimen produces a stress-strain curve.

to a final length ($l_0+\Delta 1$) (Figure 25-3). Engineering strain, which is unitless, is defined as elongation per unit length and is described as:

$$\varepsilon = \frac{1\text{-}1_0}{1_0} = \frac{\Delta 1}{1_0}$$

where Δl is elongation, l_0 is original gauge length, and l is final length after elongation, Δl. Shear strain, γ, describes the change in a right angle of a body or stress element under the action of a pure shearing load. All materials (biological and nonbiological) are characterized by a maximum elongation possible before permanent deformation or fracture results. Furthermore, biological materials exhibit strain rate dependence in that their material properties (e.g., modulus of elasticity, ultimate tensile strength) are altered as a function of the rate of loading (and subsequent deformation rate).

Experimental observation also has demonstrated that lateral strain also accompanies axial strain under the action of an axial load. Within an elastic range (defined later in this section), these two strains are proportional to one another as described by Poisson's ratio, μ. For tensile loading:

$$\mu = \frac{\text{Lateral strain}}{\text{Axial strain}}$$

The material and mechanical properties described provide for the determination of implant-tissue stress-strain behavior according to established relationships in solid mechanics theory.[12]

Stress-Strain Relationship

A relationship is needed between the applied force (and stress) that is imposed on the implant and surrounding tissues and the subsequent deformation (and strain) experienced throughout the system. If any elastic body is subjected experimentally to an applied load, then a load-versus-deformation curve can be generated (Figure 25-4, *A*). Dividing the load (force) values by the surface area over which they act and the change in the length by the original length produces a classic engineering stress-strain curve (Figure 25-4, *B*). Such a curve provides for the prediction of how much strain will be experienced in a given material under the action of an applied load. The slope of the linear (elastic) portion of this curve is referred to as the *modulus of elasticity (E)*, and its value indicates the stiffness of the material under study.

The closer the modulus of elasticity of the implant resembles that of the contiguous biological tissues, the less the likelihood of relative motion at the tissue-implant interface. The cortical bone is at least five times more flexible than titanium. As the stress magnitude increases, the relative stiffness difference between bone and titanium increases. As the stress magnitude decreases, the stiffness difference becomes much less. Restated, the viscoelastic bone can stay in contact with more rigid titanium more predictably when the stress is low. In terms of full-arch kinematics, the practitioner should consider that the mandible flexes toward the midline on opening. A prosthesis and implant support system that is splinted from molar to molar must provide similar movement if the interface is to remain intact.

Once a particular implant system (i.e., a specific biomaterial) is selected, the only way for an operator to control the strain experienced by the tissues is to control the applied stress or change the density of bone around the implant (Figure 25-5). Such stress (force/area) may be influenced by the implant design, size, implant number, implant angulation, and restoration. The macrogeometry of the implant (i.e., the amount and orientation of functional surface area available to dissipate loads) has a strong influence on the nature of the force transfer at the tissue-implant interface. Surgical grafting procedures may increase the quantity and quality of bone and allow placement of a larger implant with more bone contiguous to the interface implant. The applied stress also is influenced by the restoration, including the size of occlusal tables, stress breakers, use of overdenture versus fixed prosthesis, and occlusal contact design. Generally,

WWW.HIGHDENT.IR

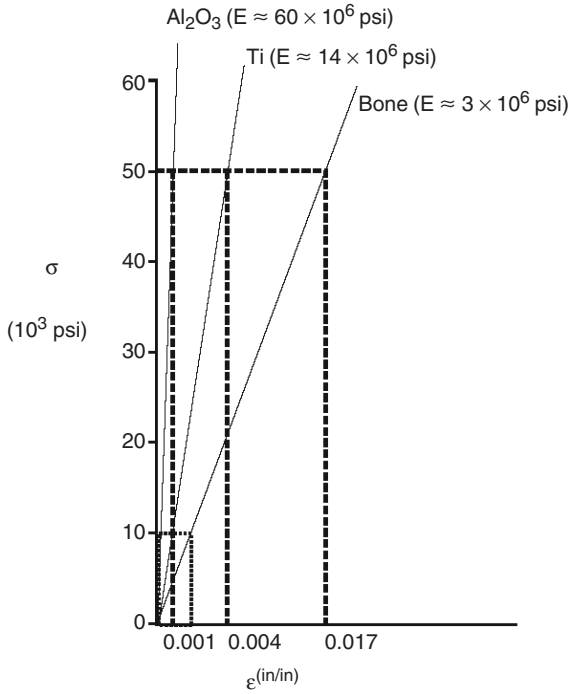


Figure 25-5 Once a particular implant system is selected, the only way to control strain (ε) on tissues is to control applied stress (σ). The greater the magnitude of stress applied to the system, the greater the difference in strain between implant material and bone. *E,* Modulus of elasticity.

the greater the magnitude of stress applied to a dental implant system, the greater the difference in strain between the implant material and bone. In such cases, the implant is less likely to stay attached to the bone, and the probability of fibrous tissue ingrowth into the interfacial region to accommodate the range of difference becomes greater. The density of bone is related not only to the bone strength but also to the modulus of elasticity (stiffness). The stiffer the bone, the more rigid it is; the softer the bone, the more flexible the bone. Therefore the difference in stiffness is less for commercially pure titanium (or its alloy) and Division 1 dense bone compared with titanium and Division 4 soft bone. Decreasing stress in softer bone is more important for two primary reasons: (1) to reduce the resultant tissue strains resulting from the elasticity difference and (2) because softer bone exhibits a lower ultimate strength.

Hooke's law is the name given to the relationship between stress and stain; in its simplest form, the law is described mathematically as the following:

$$\sigma = E\varepsilon$$

where σ is normal stress (pascal or pounds per square inch), *E* is the modulus of elasticity (pascal or pounds per square inch), and ε is normal strain (unitless). A similar relationship exists for shear stress and shear strain, where the constant of proportionality is the modulus of rigidity *(G)* expressed by the following:

$$\tau = G\gamma$$

where τ is shear stress (pascal or pounds per square inch), *G* is the modulus of rigidity (pascal or pounds per square inch), and γ is shear strain (unitless).

Impact Loads

When two bodies collide in a small interval of time (fractions of a second), large reaction forces develop. Such a collision is described as *impact*. In dental implant systems subjected to occlusal implant loads, deformation may occur in the prosthodontic restoration, in the implant itself, and in the contiguous interfacial tissues. The nature of the relative stiffness of these components in the overall implant system largely controls the response of the system to impact load. The higher the impact load, the greater the risk of implant and bridge failure and bone fracture.

Rigidly fixed implants generate a higher interfacial impact force with occlusion compared with natural teeth, which possess a periodontal ligament. Soft tissue–borne prostheses have the least impact force because the gingival tissues are resilient. Occlusal material fracture is a significant complication of fixed prostheses on natural teeth. The incidence of occlusal material fracture is greater on implants and may approach rates as high as 30%.

Various methods have been proposed to address the issue of reducing implant loads. Skalak[13] has suggested the need for using acrylic teeth along with osteointegrated fixtures partially to mitigate high-impact loads that might damage bony tissues adjacent to the implant. Weiss[14] has proposed that a fibrous tissue-implant interface provides for physiologic shock absorption in a fashion similar to that exhibited by a functioning periodontal ligament. At least one implant design has attempted to incorporate shock absorption capability in the design itself by the use of an "intramobile element" of lower stiffness compared with the rest of the implant.[15] Misch[16] advocates an acrylic provisional restoration with a progressive occlusal loading to improve the bone-implant interface before the final restoration, occlusal design, and masticatory loads are distributed to the system. Only limited data exist concerning impact forces on natural dentition and tooth-supported bridgework.[17,18]

FORCE DELIVERY AND FAILURE MECHANISMS

The manner in which forces are applied to implant restorations within the oral environment dictates the likelihood of system failure. The duration of a force may affect the ultimate outcome of an implant system. Relatively low-magnitude forces, applied repetitively over a long time, may result in fatigue failure of an implant or prosthesis. Stress concentrations and, ultimately, failure may develop if insufficient cross-sectional area is

WWW.HIGHDENT.IR

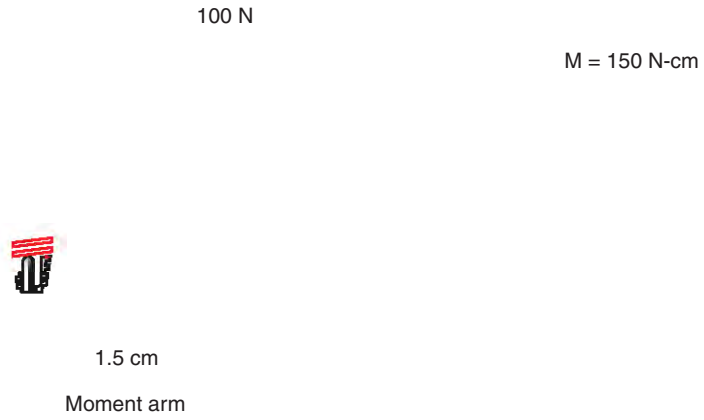


Figure 25-6 The moment of a force is defined as a vector *(M)*, the magnitude of which equals the product of the force magnitude multiplied by the perpendicular distance (moment arm) from the point of interest to the line of action of the force.

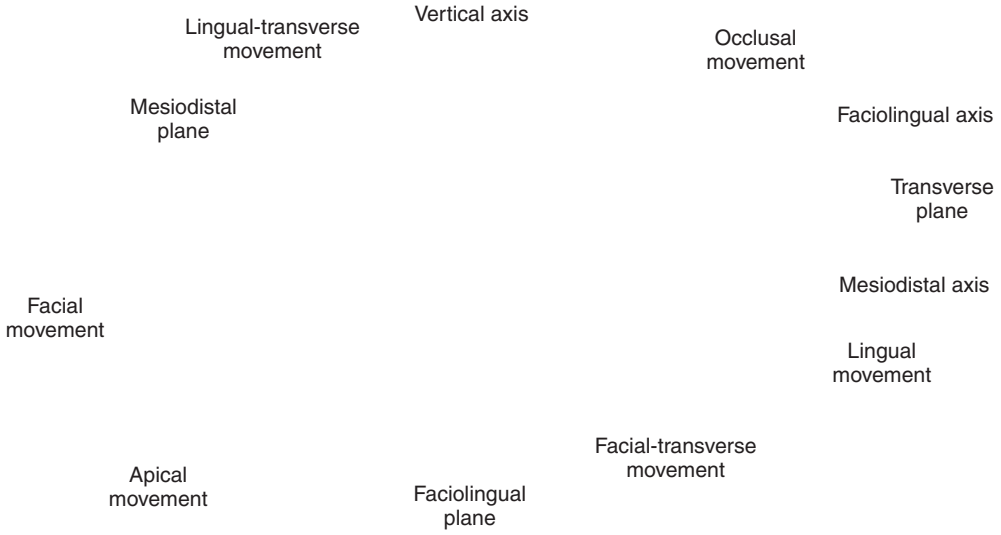


Figure 25-7 Moment loads tend to induce rotations in three planes. Clockwise and counterclockwise rotations in these three planes result in six moments: lingual-transverse, facial-transverse, occlusal, apical, facial, and lingual.

present to dissipate high-magnitude forces adequately. If a force is applied some distance away from a weak link in an implant or prosthesis, then bending or torsional failure may result from moment loads. An understanding of force delivery and failure mechanisms is critically important to the implant practitioner to avoid costly and painful complications.

Moment Loads

The moment of a force about a point tends to produce rotation or bending about that point. In Figure 25-6, the moment is defined as a vector *(M)* (vectors are described in terms of magnitude and direction), the magnitude of which equals the product of the force magnitude multiplied by the perpendicular distance (also called the *moment arm*) from the point of interest to the line of action of the force. This imposed moment load also is referred to as a *torque* or *torsional load* and may be destructive to implant systems. Torques or bending moments imposed on implants because of, for example, excessively long cantilever bridge or bar sections may result in interface breakdown, bone resorption, prosthetic screw loosening, or bar or bridge fracture. The negative effect of cantilevers has been reported for more than 30 years.[19,20] Proper restorative design must include consideration of forces and the moment loads caused by those forces.

Clinical Moment Arms

A total of six moments (rotations) may develop about the three clinical coordinate axes previously described (occlusoapical, faciolingual, and mesiodistal axes) (Figure 25-7). Such moment loads induce microrotations and stress concentrations at the crest of the alveolar ridge

WWW.HIGHDENT.IR

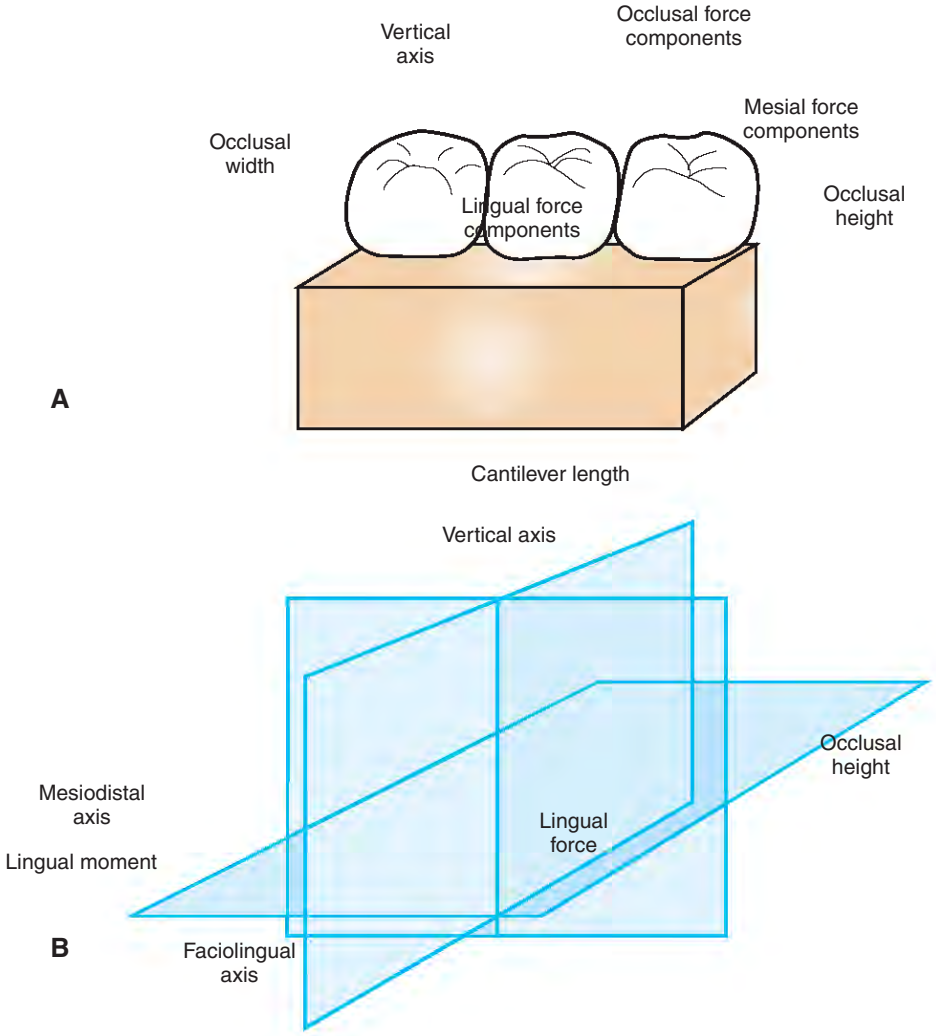


Figure 25-8 **A,** Three clinical moment arms contribute to torsional (moment) loads on dental implants: occlusal height, occlusal width, and cantilever length. **B,** Occlusal height serves as moment arm for force components directed along faciolingual axis and force components directed along mesiodistal axis **(C).** *Continued*

at the implant-tissue interface, which lead inevitably to crestal bone loss.

Three clinical moment arms exist in implant dentistry: (1) occlusal height, (2) cantilever length, and (3) occlusal width. Minimization of each of these moment arms is necessary to prevent unretained restorations, fracture of components, crestal bone loss, or complete implant system failure.

Occlusal Height

Figure 25-8 shows that the occlusal height serves as the moment arm for force components directed along the faciolingual axis—working or balancing occlusal contacts, tongue thrusts, or in passive loading by cheek and oral musculature (Figure 25-8, *B*), as well as force components directed along the mesiodistal axis (Figure 25-8, *C*).

In Division A bone, initial moment load at the crest is less than in Division C or D bone because the crown height is greater in C and D bone. Treatment planning must take into account this initially compromised bio-mechanical environment (Table 25-4). The moment contribution of a force component directed along the vertical axis is not affected by the occlusal height because no effective moment arm exists. Offset occlusal contacts or lateral loads, however, introduce significant moment arms (see Figure 25-8, *E*).

Cantilever Length

Large moments may develop from vertical axis force components in prosthetic environments designed with cantilever extensions or offset loads from rigidly fixed implants. A lingual force component also may induce a twisting moment about the implant neck axis if applied through a cantilever length (Figure 25-8, *D*).

An implant with a cantilevered mesobar extending 1, 2, and 3 cm has significant ranges of moment loads. A 100-N force applied directly over the implant does not induce a moment load or torque because no rotational forces are applied through an offset distance. This same 100-N force applied 1 cm from the implant results

WWW.HIGHDENT.IR

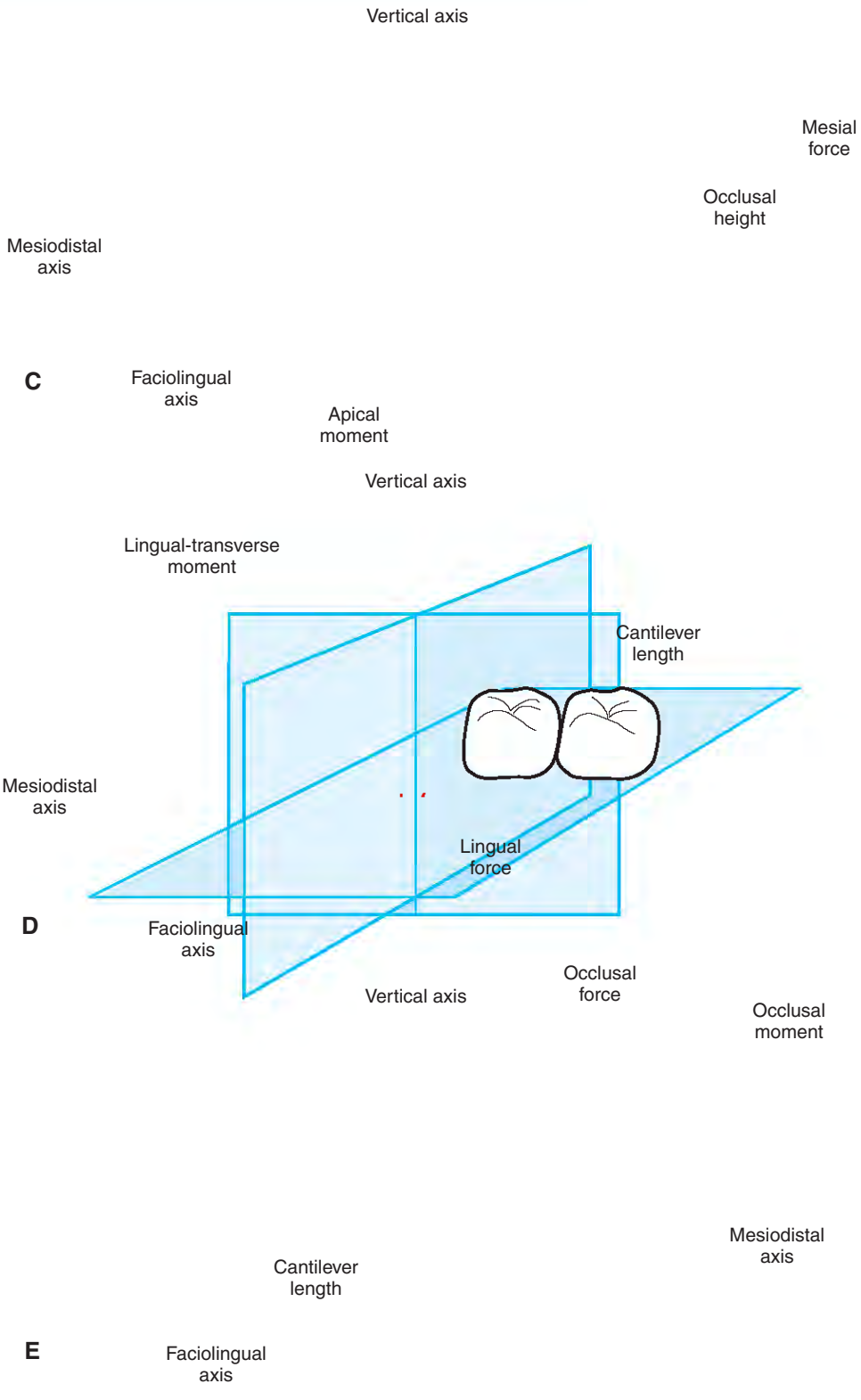


Figure 25-8, cont'd **D,** Lingual force component also may induce twisting moment about the implant neck if applied through the cantilever length. **E,** Moment of force along the vertical axis is not affected by occlusal height because its effective moment arm is zero if positioned centrically.

WWW.HIGHDENT.IR

Table 25-4 Moment Load at Crest, Division A Bone When Subjected to Forces Shown in Figure 25-9

Influences on Moment		Imposed Moments (N/mm) at Implant Crown-Crest Interface					
OCCLUSAL HEIGHT (mm)	CANTILEVER LENGTH (mm)	LINGUAL	FACIAL	APICAL	OCCLUSAL	FACIAL-TRANSVERSE	LINGUAL-TRANSVERSE
10	10	100	0	50	200	0	100
10	20	100	0	50	400	0	200
10	30	100	0	50	600	0	300
20	10	200	0	100	200	0	100
20	20	200	0	100	400	0	200
20	30	200	0	100	600	0	300

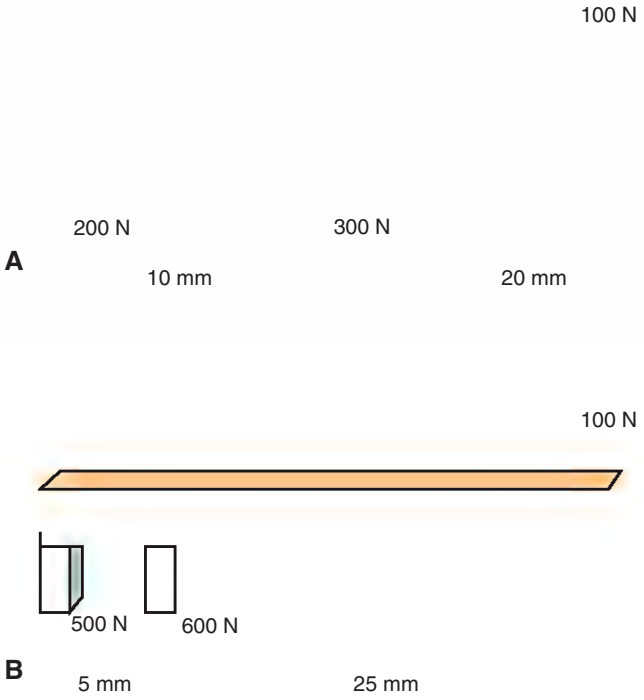


Figure 25-9 **A,** If two implants are designed 10 mm apart and splinted together with a 20-mm distal cantilever, then a 100-N load is resisted by a 200-N force by the mesial implant, and the distal implant acts as a fulcrum with a 300-N load. **B,** If implants are 5 mm apart, then the anterior implant must resist a 500-N force and the distal fulcrum implant receives a 600-N force.

in a 100 N-cm moment load. Similarly, if the load is applied 2 cm from the implant, then a 200 N-cm torque is applied to the implant-bone region, and at 3 cm a 300 N-cm moment load results. For comparison, recall that implant abutments typically are tightened with 30 N-cm of torque.

Cantilever prostheses attached to splinted implants result in a complex load reaction. In its simplest form, a Class 1 lever action may be expressed. If two implants 10 mm apart are splinted together, and a 20-mm distal cantilever is designed with a 100-N load, then the following forces result. The 100-N load is resisted with a 200-N tensile force by the mesial implant, and the distal implant acts as a fulcrum with a 300-N compressive load (Figure 25-9, *A*). If the position and amount of distal load remain the same, but the distal implant is positioned 5 mm anterior, then the resultant loads on the implants change (Figure 25-9, *B*). The anterior implant must resist a 500-N tensile force and the distal, fulcrum implant receives a 600-N compressive force. Therefore the tensile force is increased 2.5 times on the anterior implant, whereas the compressive force is increased twofold. Because bone and screws are weaker under the action of tensile forces, both implants become more at risk for complications.

Similar principles regarding Class 1 lever forces apply to cantilever loads with anterior splinted implants placed on a curve with distal extended prostheses. The Nobel Biocare prosthetic protocol uses four to six anterior implants placed in front of the mental foramen or maxillary sinuses and uses a full-arch fixed prosthesis with cantilevered segments.[21-23] Specific cantilever lengths are not stated, although two to three premolars are recommended. The cantilever length is suggested to be reduced when four rather than six implants are used to support the restoration[24] or when implants are in the softer bone of the maxilla.[25] A line is drawn from the distal of each posterior implant. The distance to the center of the most anterior implant is called the *anteroposterior distance* (A-P spread).[26] The greater the A-P spread is between the center of the most anterior implant or implants and the most distal aspect of the posterior implants, the smaller is the resultant loads on the implant system from cantilevered forces because of the stabilizing effect of the A-P distance. According to Misch,[25] the amount of stress applied to the system determines the length of this distal cantilever. Because stress equals force divided by area, both aspects must be considered. The magnitude and direction of force are determined by parafunction, crown height, masticatory dynamics, gender, age, and arch location. The functional surface area is determined by the number of implants, width, length, design, and bone density, which determines

WWW.HIGHDENT.IR

the area of contact and bone strength. Clinical experiences suggest that the distal cantilever should not extend 2.5 times the A-P spread under ideal conditions (e.g., parafunction absent or five Division A implants). One of the greatest determinants for the length of the cantilever is the magnitude of the force. Patients with severe bruxism should not be restored with any cantilevers, regardless of other factors.

A square arch form involves smaller A-P spreads between splinted implants and should have shorter-length cantilevers. A tapered arch form has the largest distance between anterior and posterior implants and may have the longest cantilever design. The maxilla has less dense bone than the mandible and more often has an anterior cantilever with the prosthesis. As a result, more distal implants may be required in the maxilla to increase the A-P spread for the anterior or posterior cantilever than in the mandible, and sinus augmentation may be required to permit posterior placement of the implant.

Occlusal Width

Wide occlusal tables increase the moment arm for any offset occlusal loads. Faciolingual tipping (rotation) can be reduced significantly by narrowing the occlusal tables or adjusting the occlusion to provide more centric contacts.

In summary, a vicious, destructive cycle can develop with moment loads and result in crestal bone loss. As crestal bone loss develops, occlusal height automatically increases. With an increased occlusal height moment arm, the faciolingual microrotation and rocking increases and causes even more crestal bone loss. Unless the bone increases in density and strength, the cycle continues to spiral toward implant failure if the biomechanical environment is not corrected.

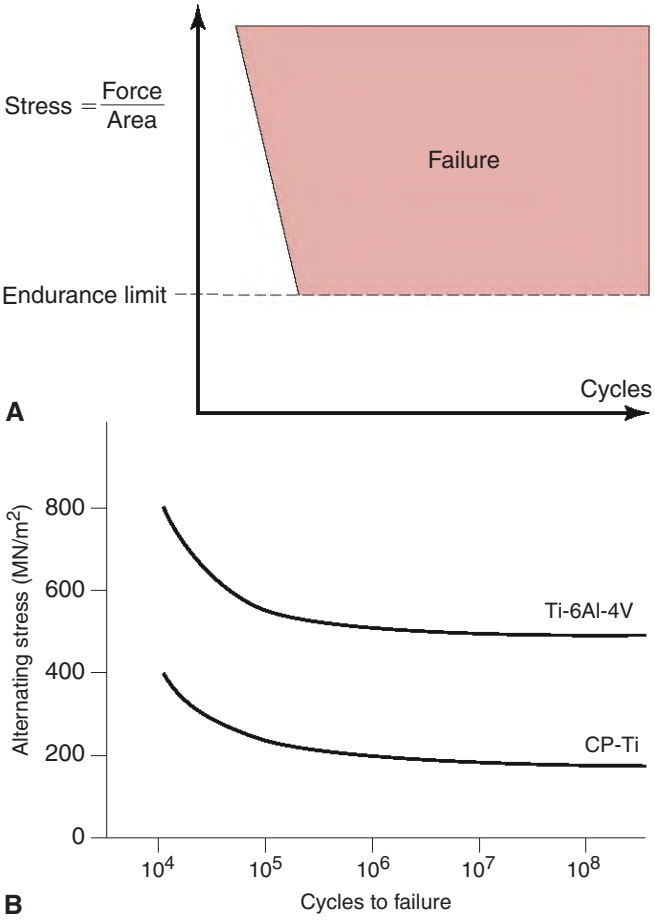


Figure 25-10 **A,** Fatigue behavior of biomaterials is characterized by a plot of applied stress versus number of loading cycles, an S-N curve. **B,** Endurance limit defines the stress level below which an implant biomaterial may be loaded indefinitely without failure. Titanium alloy is two to four times stronger in fatigue conditions compared with commercially pure titanium.

Fatigue Failure

Fatigue failure is characterized by dynamic, cyclic loading conditions. Four fatigue factors significantly influence the likelihood of fatigue failure in implant dentistry: (1) biomaterial, (2) macrogeometry, (3) force magnitude, and (4) number of cycles.

Fatigue behavior of biomaterials is characterized graphically in what is referred to as an *S-N curve* (a plot of applied stress versus number of loading cycles) (Figure 25-10, *A*). If an implant is subjected to an extremely high stress, then only a few cycles of loading can be tolerated before fracture occurs. Alternatively, an infinite number of loading cycles can be maintained at low stress levels. The stress level below which an implant biomaterial can be loaded indefinitely is referred to as its *endurance limit*. Titanium alloy exhibits a higher endurance limit compared with commercially pure titanium (Figure 25-10, *B*).

The geometry of an implant influences the degree to which it can resist bending and torsional loads and ultimately fatigue fracture. Implants rarely, if ever, display fatigue fracture under axial compressive loads. Morgan et al.[27] reported fatigue fractures of Brånemark dental implants caused by cyclical buccolingual loads (lateral loading) in an area of weak bending strength within the fixture (i.e., reduced moment of inertia [defined later]). The fracture of the implant body occurred in three of the patients studied, and fracture of the abutment screws for the Brånemark implant occurred in less than three patients. Fifteen acrylic or composite tooth fractures occurred on 10 to 20 of the fixed prostheses supported by implants over a 1- to 5-year period.[28-31]

The geometry also includes the thickness of the metal or implant. The fatigue fracture is related to the fourth power of the thickness difference. A material two times thicker in wall thickness is approximately 16 times stronger. Even small changes in thickness can result in significant differences. Often the weak link in an implant body design is affected by the difference in the inner and outer diameter of the screw and the abutment screw space in the implant.[32]

WWW.HIGHDENT.IR

To the extent that an applied load (stress) can be reduced, the likelihood of fatigue failure is reduced. As described previously, the magnitude of loads on dental implants can be reduced by careful consideration of arch position (i.e., higher loads in the posterior compared with anterior mandible and maxilla), elimination of moment loads, and increase in surface area available to resist an applied load (i.e., optimize geometry for functional area, or increase the number of implants used).

Finally, fatigue failure is reduced to the extent that the number of loading cycles is reduced. Thus aggressive strategies to eliminate parafunctional habits and reduce occlusal contacts serve to protect against fatigue failure.

MOMENT OF INERTIA

Moment of inertia is an important property of cylindrical implant design because of its importance in the analysis of bending and torsion. The bending stress in a cylinder is given by the following equation:

$$\sigma = \frac{My}{I}$$

where M is moment (newton-centimeters), y is the distance from the neutral axis of bending (centimeters), and I is the moment of inertia (centimeters to the fourth power).

Root form implants have varying cross-sectional geometries. The root form implant may be modeled as a hollow circle, because a channel exists in the implant body to allow for abutment screw engagement. In the distal (apical) region of a root form implant, the cross-sectional geometry may more closely represent a solid circle. In some designs, vents that penetrate transversely through the cross-sectional geometry may interrupt the apical geometry.

The bending stress (and likelihood of bending fracture) decreases with increasing moment of inertia. Consider the mathematical formulations for the solid versus hollow cylindrical cross-sectional geometry:

Solid circle (cylinder in the middle region): $4\ I = \pi R^4$

Hollow circle (cylinder in apical region): $4\ I = \pi(R^4 - R_i 4)$

where R is the outer radius (centimeters) and R_i is the inner wall radius.

SUMMARY

The most common complications in implant-related reconstruction are related to biomechanical conditions. Implant healing failures may result from micromovement of the implant from too much stress. Early crestal bone loss may be related to occlusal overload conditions. Prostheses or abutment screws may become loose from bending or moment forces. Implant or component fracture may occur from fatigue conditions. Prosthesis failure may result from all of the foregoing or bending fracture resistance. In addition, the manifestation of biomechanical loads on dental implants (moments, stress, and strain) controls the long-term health of the bone-implant interface. Knowledge of basic biomechanical principles is thus required for the dentist.

References

1. Schmid-Schonbein GW, Woo SL-Y, Zweifack BW, editors: *Frontiers in biomechanics,* New York, 1986, Springer-Verlag.
2. National Institutes of Health Consensus Development Conference Statement on Dental Implants, *J Dent Educ* 52:824-827, 13-15, June 1988.
3. Higdon A: *Engineering mechanics, vol 2, Dynamics,* Englewood Cliffs, NJ, 1976, Prentice-Hall.
4. Baumeister T, Avallone EA, Baumeister T, editors: *Marks' standard handbook for mechanical engineers,* ed 8, New York, 1978, McGraw-Hill.
5. Braun S, Bantleon H-P, Hnat WP et al: A study of bite force. I. Relationship to various physical characteristics, *Angle Orthod* 65:367-372, 1995.
6. van Eijden TMGJ: Three-dimensional analyses of human bite-force magnitude and moment, *Arch Oral Biol* 36: 535-539, 1991.
7. Dean JS, Throckmorton GS, Ellis EE et al: A preliminary study of maximum voluntary bite force and jaw muscle efficiency in preorthognathic surgery patients, *J Oral Maxillofac Surg* 50:1284-1288, 1992.
8. Bakke M, Holm B, Jensen L et al: Unilateral, isometric bite force in eight- to eighty-eight year old women and men related to occlusal factors, *Scand J Dent Res* 98:149-158, 1990.
9. Braun S, Hnat WP, Freudenthaler JW et al: A study of maximum bite force during growth and development, *Angle Orthod* 66:261-264, 1996.
10. Reilly DT, Burstein AH: The elastic and ultimate properties of compact bone tissue, *J Biomech* 8:393, 1975.
11. Lemons JE, Bidez MW: Biomaterials and biomechanics in implant dentistry. In McKinney RV, editor: *Endosteal dental implants,* St Louis, 1991, Mosby.
12. Timoshenko SP, Goodier JN: *Theory of elasticity,* ed 3, New York, 1970, McGraw-Hill.
13. Skalak R: Biomedical considerations in osseointegrated prostheses, *J Prosthet Dent* 49:843-848, 1983.
14. Weiss CW: Fibro-osteal and osteal integration: a comparative analysis of blade and fixture type dental implants supported by clinical trials, *J Dent Educ* 52:706-711, 1988.
15. Kirsch A: The two-phase implantation method using IMZ intramobile cylinder implants, *J Oral Implantol* 11:197-210, 1983.
16. Misch CE: Progressive bone loading, *Pract Periodontics Esthetic Dent* 2:27-30, 1990.
17. Salis SG, Hood JA, Stokes AN et al: Impact-fracture energy of human premolar teeth, *J Prosthet Dent* 58:43-48, 1987.
18. Saunders WP: The effects of fatigue impact forces upon the retention of various designs of resin-retained bridgework, *Dent Mater* 3:85-89, 1986.
19. Schweitzer JM, Schweitzer RD, Schweitzer J: Free end pontics used on fixed partial dentures, *J Prosthet Dent* 20:120-138, 1968.

WWW.HIGHDENT.IR

20. Crabb HSM: A reappraisal of cantilever bridgework, *J Oral Rehabil* 1:3-17, 1974.
21. Brånemark PI, Zarb GA, Albrektsson T: *Tissue integrated prostheses,* Chicago, 1985, Quintessence.
22. Zarb GA, Schmitt A: The longitudental clinical effectiveness of osseointegrated dental implants, the Toronto study. III. Problems and complications encountered, *J Prosthet Dent* 64:185-194, 1990.
23. Taylor R, Bergman G: *Laboratory techniques for the Brånemark system,* Chicago, 1990, Quintessence.
24. Rangert B, Jemt T, Jorneus L: Forces and moments on Brånemark implants, *Int J Oral Maxillofac Implants* 4: 241-247, 1989.
25. Misch CE: Cantilever length and its relationship to bio-mechanical stress. In *Misch Implant Institute Manual,* Dearborn, Mich, 1990, Misch International Implant Institute.
26. English C: The critical A-P spread, *Implant Soc* 1:2-3, 1990.
27. Morgan MJ, James DF, Pilliar RM: Fractures of the fixture component of an osseointegrated implant, *Int J Oral Maxillofac Implants* 8:409-413, 1993.
28. Lekholm U, Adell R, Brånemark PI: Complications. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue integrated prostheses,* Chicago, 1985, Quintessence.
29. Lekholm U, van Steenberghe D, Herman D: Osseointegrated implants in the treatment of partially edentulous jaws: a prospective 5-year multicenter study, *Int J Oral Maxillofac Implants* 9:627-635, 1994.
30. Quirynen M, Naert I, van Steenberghe D et al: The cumulative failure rate of the Brånemark system in the overdenture, the fixed partial, and the fixed full prosthesis design: a prospective study in 1,273 fixtures, *J Head Neck Pathol* 10:43-53, 1991.
31. Naert I, Quirynen M, van Steenberghe D et al: A six-year prosthodontic study of 509 consecutively inserted implants for the treatment of partial edentulism, *J Prosthet Dent* 67:236-245, 1992.
32. Boggan RS, Strong JT, Misch CE et al: Influences of hex geometry and geometric table width on static and fatigue strength of dental implants, *J Prosthet Dent* 82:436-440, 1999.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 26

Bone Physiology, Metabolism, and Biomechanics

W. Eugene Roberts

Consistent success with implant-supported prostheses requires a thorough knowledge of the physiology, metabolism and biomechanics of bone as a tissue, and bones as musculoskeletal organs. Bone is a vital mineralized tissue and bones are unique morphologic organs, composed of calcified and soft tissues that provide structural and metabolic support for a wide variety of interactive functions (Figure 26-1).

Understanding the clinical manipulation of bone begins with an appreciation of the fundamental genetic and environmental mechanisms of osseous development and adaptation. The genome codes for growth factors, ischemic agents, vascular induction/invasion mechanisms, and mechanically induced inflammation. These biological mechanisms interact with the physical factors of diffusion limitation and mechanical loading to produce bone morphology (Figure 26-2). Fundamental principles control the quality and quantity of bone that directly and indirectly supports stomatognathic function. A firm grasp of the modern concepts of bone physiology, metabolism, and biomechanics is an essential prerequisite for innovative clinical practice. These principles are an objective basis for designing a realistic treatment plan that has a high probability of meeting the esthetic and functional expectations of the patient.

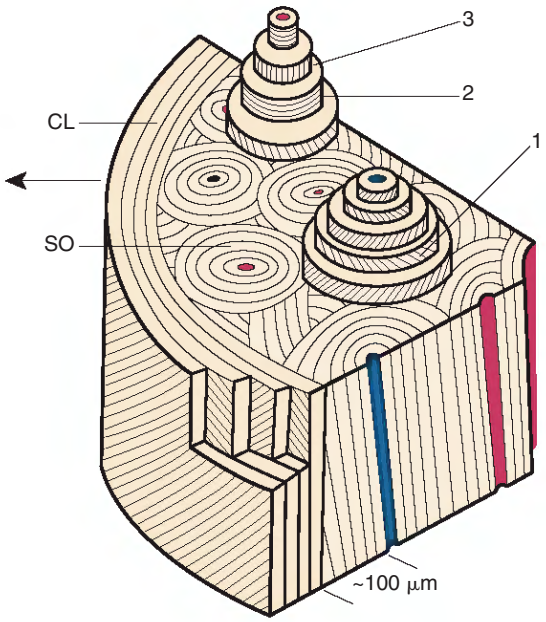


Figure 26-1 A schematic drawing of a wedge of a cortical bone that is growing to the left demonstrates the morphology of circumferential lamellae *(CL)* and secondary osteons *(SO)*. Depending on the mechanical loading at the time the matrix is formed, bone lamellae may have a collagen orientation that is an alternating bias *(1)* or alternating horizontal *(2)* and vertical *(3)* orientations. *(From Roberts WE, Hartsfield JK Jr: Bone development and function: genetic and environmental mechanisms,* Semin Orthod *10:100-122, 2004.)*

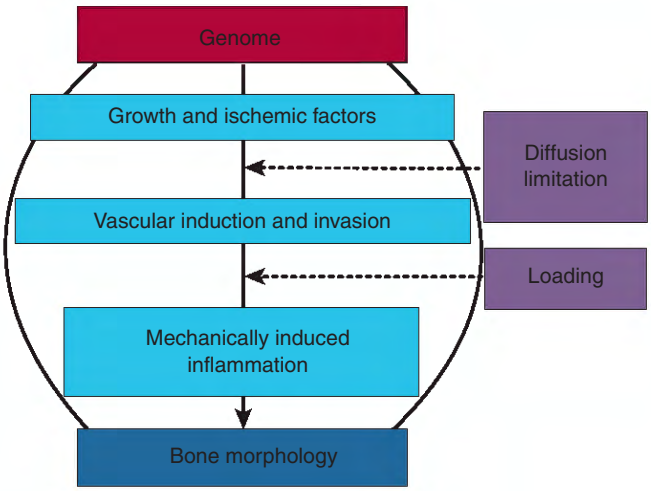


Figure 26-2 The genome dictates bone morphology by a sequence of three genetic mechanisms: (1) growth and ischemic factors, (2) vascular induction and invasion, and (3) mechanically induced inflammation. The latter two are influenced by two major physical influences: (1) diffusion limitation for maintaining viable osteocytes and (2) mechanical loading history. *(From Roberts WE, Hartsfield JK Jr: Bone development and function: genetic and environmental mechanisms,* Sem Orthod *10:100-122, 2004.)*

WWW.HIGHDENT.IR

Bone is a dynamic structure that is adapting constantly to its environment. Because the skeleton is the principal reservoir of calcium, bone remodeling (physiologic turnover) performs a critical life support role in mineral metabolism (Figure 26-3). Collectively, bones are essential elements for locomotion, antigravity support, and life-sustaining functions such as mastication. Mechanical adaptation of bone is the physiologic basis of stomatognathic reconstruction with implant-supported prostheses. A detailed knowledge of the dynamic nature of bone physiology and biomechanics is essential to enlightened clinical practice.

OSTEOLOGY

In defining the physiologic basis of orthodontics, the initial consideration is bone morphology (osteology) of the craniofacial complex. Via the systematic study of a personal collection of more than 1000 human skulls, Spencer Atkinson[1] provided the modern basis of craniofacial osseous morphology as it relates to the biomechanics of stomatognathic function. A frontal section of an adult skull shows the bilateral symmetry of bone morphology and functional loading (Figures 26-4 and 26-5). Because the human genome contains genes to pattern the structure of only half of the body, the contralateral side is a mirror image. Consequently, normal development of the head is symmetrical. Thus unilateral structures are on the midline, and bilateral structures are equidistant from it. As shown in Figure 26-5, the vertical components of the cranium tend to be loaded in compression (negative stress), and the horizontal components are loaded in tension (positive stress). From an engineering perspective, the internal skeletal structure of the midface is similar to that of a ladder: vertical rails loaded in compression connected by rungs loaded in tension. This is one of the most efficient structures for achieving maximal compressive strength with minimal mass in a composite material.

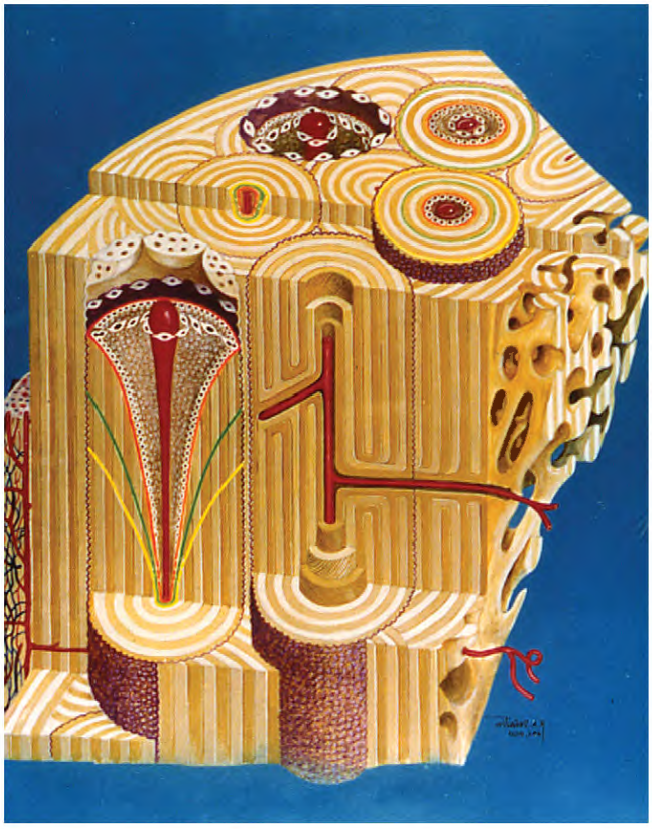


Figure 26-3 This artist's rendition of the dynamic principles of cortical bone remodeling was produced by the renowned dental illustrator Rolando De Castro. Remodeling is a vascularly mediated process of bone turnover that maintains the integrity of structural support and is a source of metabolic calcium. Osteoblasts are derived from preosteoblasts circulating in the blood, and perivascular mesenchymal cells give rise to osteoblasts. Note the three colored chevrons (yellow, green, and orange) progressively marking the mineralization front of the evolving second osteon that is moving superiorly on the left. *(From Roberts WE, Arbuckle GR, Simmons KE: What are the risk factors of osteoporosis? Assessing bone health,* J Am Dent Assoc *122:59-61, 1991.)*

Differential Osteology of the Maxilla and Mandible

Although equal and opposite functional loads are delivered to the maxilla and mandible, the maxilla transfers stress to the entire cranium, whereas the mandible must absorb the entire load. Consequently, the mandible is much stronger and stiffer than the maxilla.

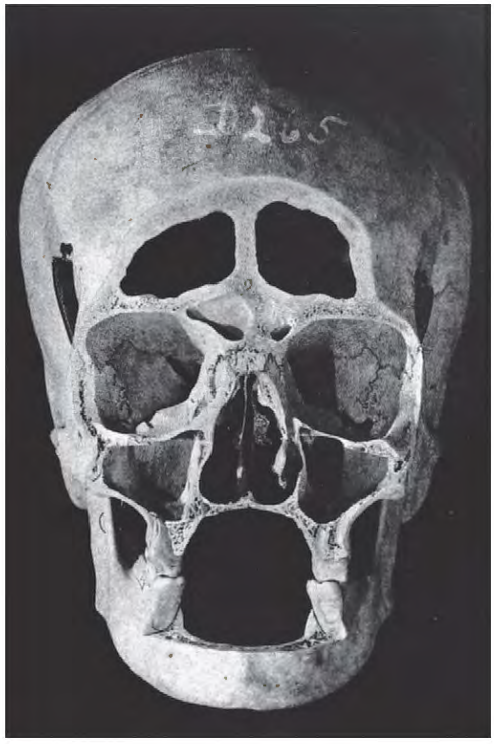


Figure 26-4 Frontal section of a human skull in the plane of the first molars. *(From Atkinson SR: Balance: the magic word,* Am J Orthod *50:189, 1964.)*

WWW.HIGHDENT.IR

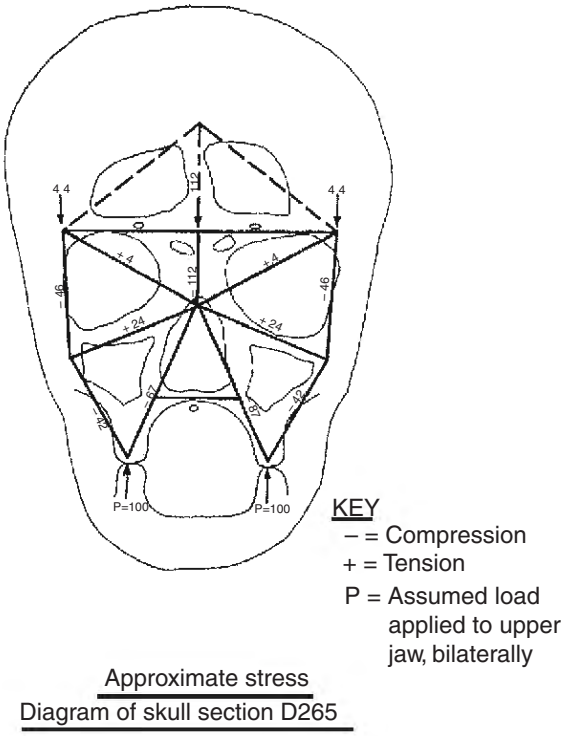


Figure 26-5 Two-dimensional vector analysis of stress in the frontal section of the human skull depicted in Figure 26-4. Relative to a bilateral biting force of 100 arbitrary units, the load is distributed to the vertical components of the midface as compressive (negative) stress. The horizontal structural components are loaded in tension. In a nongrowing individual the stress across the midpalatal suture is 0. When masticating, loads increase and the midpalatal suture is subjected to a tensile load, resulting in an increase in maxillary width. *(From Atkinson SR: Balance: the magic word,* Am J Orthod *50:189, 1964.)*

Figure 26-6 Midsagittal section of a human skull shows that the maxilla primarily is composed of trabecular (spongy) bone. The opposing mandible has thick cortices connected by relatively coarse trabeculae. *(From Atkinson SR: Balance: the magic word,* Am J Orthod *50:189, 1964.)*

A midsagittal section through the incisors (Figure 26-6) and a frontal section through the molar region (Figure 26-7) show the distinct differences in the osseous morphology of the maxilla and mandible. The maxilla has relatively thin cortices that are interconnected by a network of trabeculae (see Figures 26-4, 26-6, and 26-7).

Figure 26-7 Frontal section of the maxilla and mandible in the plane of the first molars. Because it transmits masticatory loads to the entire cranium, the maxilla has thin cortices connected by relatively fine trabeculae. The mandible, however, is loaded in bending and torsion; it therefore is composed of thick cortical bone connected by coarse, oriented trabeculae. *(From Atkinson SR: Balance: the magic word,* Am J Orthod *50:189, 1964.)*

Because it is loaded primarily in compression, the maxilla is structurally similar to the body of a vertebra.

The mandible, however, has thick cortices and more radially oriented trabeculae (see Figures 26-6 and 26-7). The structural array is similar to the shaft of a long bone and indicates that the mandible is loaded predominantly in bending and torsion. This biomechanical impression based on osteology is confirmed by in vivo strain gauge studies in monkeys. Hylander[2,3] demonstrated substantial bending and torsion in the body of the mandible associated with normal masticatory function (Figure 26-8). A clinical correlation consistent with this pattern of surface strain is the tendency of some humans to form tori in the areas of maximal bending and torsion (Figure 26-9). The largest tori are on the side on which the individual habitually chews (preferential working side).

Temporomandibular Articulation

The temporomandibular joint (TMJ) is the principal adaptive center for determining the intermaxillary relationship in all three planes of space. Figure 26-10 shows optimal skeletal development consistent with normal morphology of the TMJ. Figure 26-11 shows aberrant skeletal and dental relationships consistent with degeneration of the fossa and mandibular condyle (i.e., the enlarged mushroom shape of the condylar process, the roughened topography of the articulating

WWW.HIGHDENT.IR

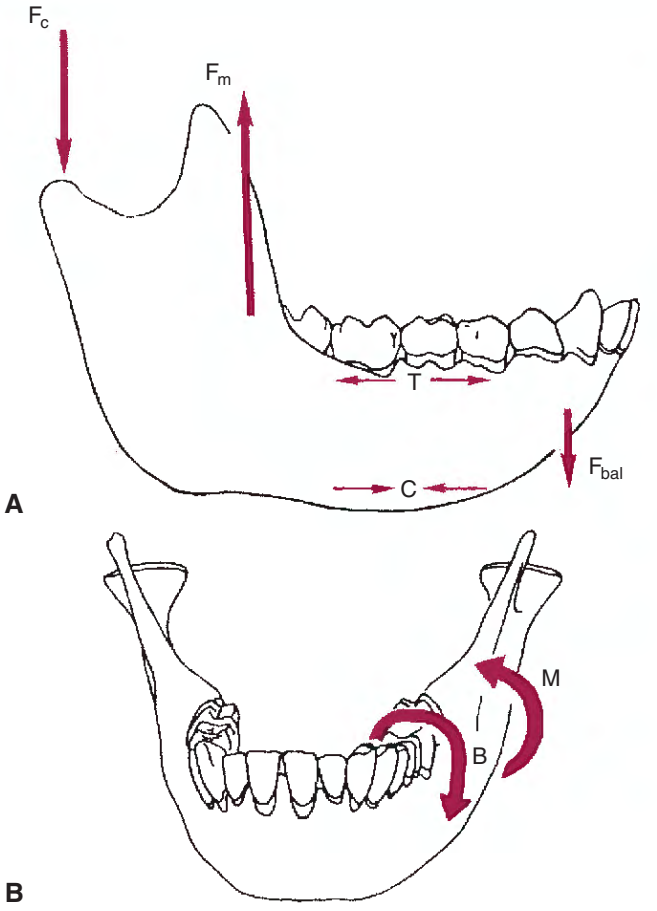


Figure 26-8 Stress patterns in the primate mandible during unilateral mastication. F_c and F_m are the condyla reaction and the resultant muscle forces on the balancing side, respectively. F_{bal} is the force transmitted through the symphysis from the balancing to the working side. T and C indicate the location of tensile stress and compressive stress, respectively. **A,** During the power stroke, the mandibular corpus on the balancing side is bent primarily in the sagittal plane, resulting in tensile stress along the alveolar process and compressive stress along the lower border of the mandible. **B,** On the working side, the corpus is twisted primarily about its long axis (it also experiences direct shear and is slightly bent). The muscle force on this side tends to evert the lower border of the mandible and invert the alveolar process *(curved arrow M).* The twisting movement associated with the bite force has the opposite effect *(curved arrow B).* The portion of the corpus between these two twisting movements experiences maximal twisting stress. *(From Hylander WL: Patterns of stress and strain in the macaque mandible. In Carlson DS, editor:* Craniofacial biology, *Ann Arbor, Mich, 1981, Center for Human Growth and Development.)*

Figure 26-9 Occlusal view of the mandibular dentition of a male patient with extensive buccal and lingual tori. Note that the exostoses are most extensive in the area of the second premolar and first molar, the area of maximal torsion in the posterior segment of the mandible.

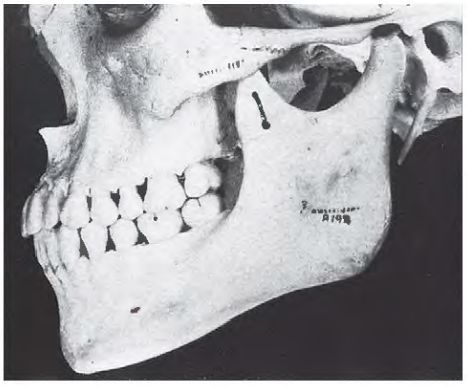


Figure 26-10 Adult human skull with ideal occlusion and osseous form of the maxilla and mandible. Note the ideal anatomical form of the condyle and articular fossa of the temporomandibular joint. *(From Atkinson SR: Balance: the magic word,* Am J Orthod *50:189, 1964.)*

Figure 26-11 Adult human skull with a severe Class II malocclusion. Note the degeneration of the temporomandibular joint (i.e., the large, mushroom-shaped condyle and the enlarged, roughened articular fossa). *(From Atkinson SR: Balance: the magic word,* Am J Orthod *50:189, 1964.)*

surfaces, the loss of articular cartilage and subchondral plate). Progressive degeneration or hyperplasia of one or both mandibular condyles may result in substantial intermaxillary discrepancies in the sagittal, vertical, and frontal dimensions. Adaptation of the TMJ allows for substantial growth change to occur without disturbing the intermaxillary relationship of the dentition (e.g., Class I occlusion remains Class I). In the adult years the intermaxillary relationship continues to change but at a slower rate. The face lengthens and may rotate anteriorly as much as 10 mm over the adult lifetime.[4] The mandible adapts to this change by lengthening and maintaining the intermaxillary dental relationship (Figure 26-12). However, if the TMJs of an adult undergo bilateral degenerative change, whether

WWW.HIGHDENT.IR

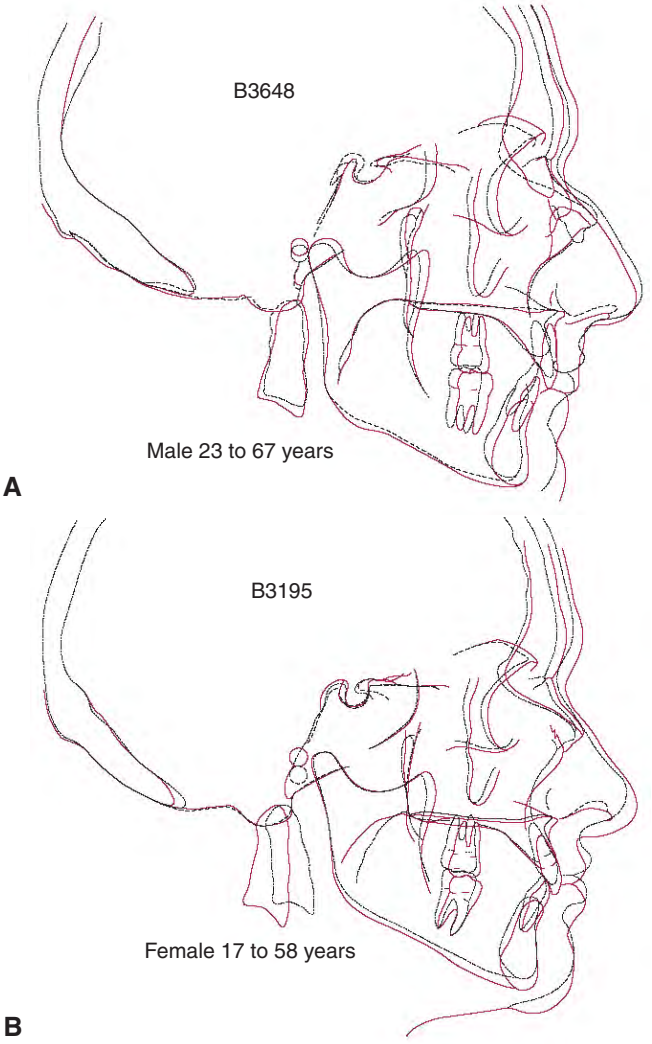


Figure 26-12 **A,** Superimposed cephalometric tracings of a male patient at ages 23 and 67 years (black and red tracings, respectively). Note the downward and forward growth of the mandible and the substantial increase in the length of the face. The nasal form was altered by a rhinoplasty in the intervening years. **B,** Superimposed cephalometric tracings of an adult female at ages 17 and 58 years (black and red tracings, respectively). Note the downward growth of the mandible and the substantial increase in the length of the face. *(From Behrents RG: Adult facial growth. In Enlow DH, editor:* Facial growth, *ed 3, Philadelphia, 1990, WB Saunders.)*

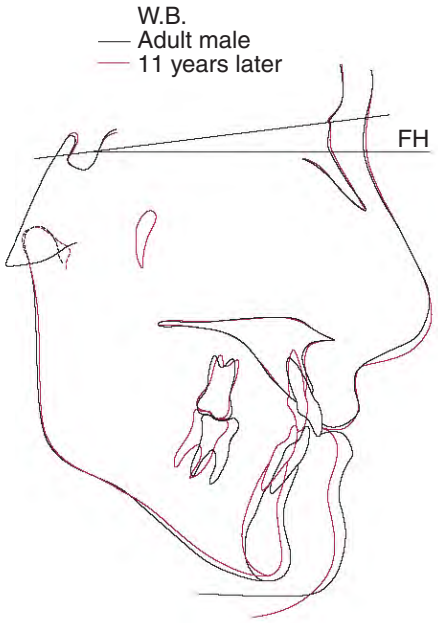


Figure 26-13 Superimposed cephalometric tracings of a middle-aged man over an 11-year period. Although the soft tissue changes are similar to those seen in other adult males (see Figure 26-12, *A*), the less prominent mandible and more convex skeletal profile are consistent with a shortened mandible. This man had a history of bilateral temporomandibular internal derangement that progressed to crepitus. However, despite the substantial change in facial form and occlusal compensation, no appreciable pain was reported.

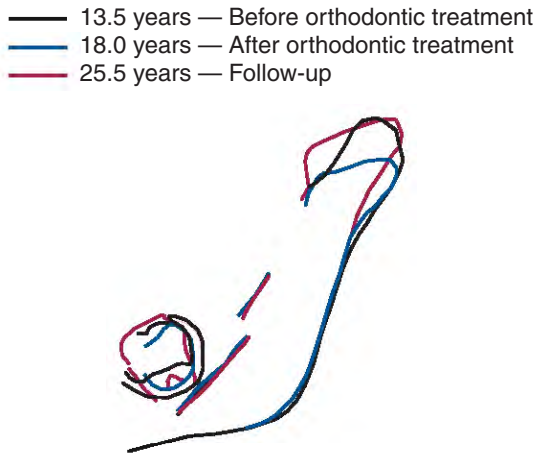


Figure 26-14 Superimposed tracings of a series of panoramic radiographs documenting the degeneration of a mandibular condyle between 13.5 and 18 years of age. Between the ages of 18 and 25.5 years, the degenerative condyle "grew," restoring the original length of the mandible. *(From Peltola JS, Kononen M, Nystrom M: A follow-up study of radiographic findings in the mandibular condyles of orthodontically treated patients and associations with TMD,* J Dent Res *74:1571, 1995.)*

symptomatic or not, the mandible can decrease in length, resulting in a shorter, more convex face (Figure 26-13).

Within physiologic limits, the TMJ has remarkable regenerative and adaptive capabilities allowing for spontaneous recovery from degenerative episodes (Figure 26-14). Unlike other joints in the body, the TMJ has the ability to adapt to altered jaw structure and function. After a subcondylar fracture, the condylar head is pulled medially by the superior pterygoid muscle and resorbs. If the interocclusal relationship is maintained, a new condyle forms from the medial aspect of the ramus and assumes normal function. Unilateral subcondylar fractures usually result in regeneration of a new functional condyle with no significant deviation of the mandible.[5] However, about one fourth of subcondylar fractures result in a mandibular deviation toward the injured side, resulting in an asymmetrical Class II malocclusion with a midline deviation. Another sequela of mandibular trauma is internal derangement such as a unilateral closed lock (a condyle distally

WWW.HIGHDENT.IR

displaced relative to the disk). If the range of motion is reduced in a growing patient, the compromised function may inhibit mandibular growth, resulting in a cant of the occlusal plane. Progressive dysfunction and pain may ensue, particularly when associated with occlusal trauma. Reestablishing normal bilateral function allows the compromised condyle or condyles to adapt favorably.

BONE PHYSIOLOGY

The morphology of bone has been well described, but its physiology is elusive because of the technical limitations inherent in the study of mineralized tissues. Accurate assessment of the orthodontic or orthopedic response to applied loads requires time markers (bone labels) and physiologic indexes (DNA labels, histochemistry, and in situ hybridization) of bone cell function. Systematic investigation with these advanced methods has defined new concepts of clinically relevant bone physiology.

Specific Assessment Methodology

Physiologic interpretation of the response to applied loads requires the use of specially adapted methods, as follows:

- Mineralized sections are an effective means of accurately preserving structure and function relationships.[6]
- Polarized light birefringence detects the preferential orientation of collagen fibers in the bone matrix.[7]
- Fluorescent labels (e.g., tetracycline) permanently mark all sites of bone mineralization at a specific point in time (anabolic markers).[7]
- Microradiography assesses mineral density patterns in the same sections.[8]
- Autoradiography detects radioactively tagged precursors (e.g., nucleotides, amino acids) used to mark physiologic activity.[9-11]
- Nuclear volume morphometry differentially assesses osteoblast precursors in a variety of osteogenic tissues.[12]
- Cell kinetics is a quantitative analysis of cell physiology based on morphologically distinguishable events in the cell cycle (i.e., DNA synthesis [S] phase, mitosis, and differentiation-specific change in nuclear volume).[12,13]
- Finite element modeling is an engineering method of calculating stresses and strains in all materials, including living tissue.[14-17]
- Microelectrodes inserted in living tissue such as the periodontal ligament (PDL) can detectelectrical potential changes associated with mechanical loading.[13,18]
- Backscatter emission is a variation of electron microscopy that assesses relative mineral density at the microscopic level in a block specimen.[19]
- Microcomputed tomography is an in vitro imaging method for determining the relative mineral density of osseous tissue down to a resolution of approximately 5 µm (about the size of an osteoblast nucleus).[20]
- Microindentation testing is a method for determining the mechanical properties of bone at the microscopic level.[21]

Mineralized Sections

Fully mineralized specimens are superior to routine demineralized histologic sections for most critical analyses of teeth, periodontium, and supporting bone because fully mineralized specimens experience less processing distortion. Furthermore, the inorganic mineral and organic matrix can be studied simultaneously.[6-8,22] For tissue-level studies, sections 100 µm thick are appropriate because they can be studied by means of several analytic methods. Even without bone labels, microradiographic images of polished mineralized sections provide substantial information about the strength, maturation, and turnover rate of cortical bone (Figure 26-15, *A*). Reducing the thickness of the section to less than 25 µm considerably enhances cellular detail and resolution of bone labels. Specific stains are useful for enhancing the contrast of cellular and extracellular structures. The disadvantages of thin mineralized sections are (1) bone labels quench more rapidly and (2) tissue density is inadequate for microradiographic analysis.

Polarized Light

Birefringence of polarized light (see Figure 26-15, *B*) has particular biomechanical significance. The lamellar fringe patterns revealed with polarized light indicate the preferential collagen orientation within the matrix.[23] Most lamellar bone has alternating layers of collagen fibers at right angles. However, two specialized collagen configurations can be seen in the same or adjacent osteons: (1) longitudinally aligned collagen fibers efficiently resist tension and (2) transverse or circumferential collagen fibers are preferential supports for compression.[24] Loading conditions at the time of bone formation appear to dictate the orientation of the collagen fibers to best resist the loads to which the bone is exposed. The important point is that bone formation can adapt to different loading conditions by changing the internal lamellar organization of mineralized tissue.

Fluorescent Labels

Administered in vivo, calcium-binding labels are anabolic time markers of bone formation. Histomorphometric analysis of label incidence and inter-label distance is an effective method of determining the mechanisms of bone growth and functional adaptation (see Figure 26-15, *C*). Because they fluoresce at different wavelengths (colors), six bone labels can be used: (1) tetracycline (10 mg/kg, bright yellow); (2) calcein green

WWW.HIGHDENT.IR

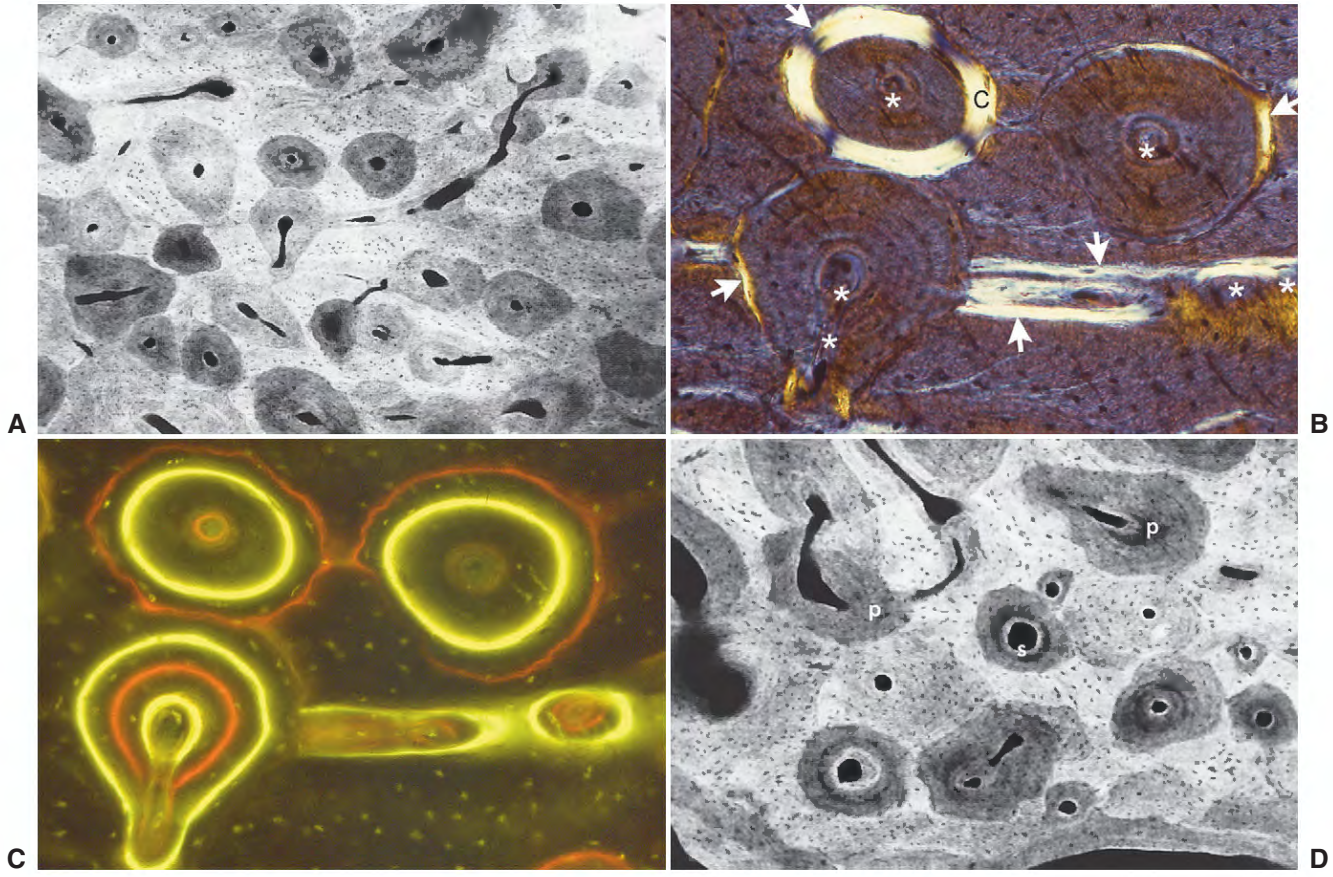


Figure 26-15 **A,** Microradiography provides a physiologic index of bone turnover and relative stiffness. The more radiolucent (dark) osteons are the youngest, the least mineralized, and the most compliant. Radiodense (white) areas are the oldest, most mineralized, and rigid portions of the bone. **B,** Polarized light microscopy shows the collagen fiber orientation in bone matrix. Lamellae with a longitudinally oriented matrix *(C)* are particularly strong in tension, whereas a horizontally oriented matrix (dark) has preferential strength in compression (arrows mark resorption arrest lines, and asterisks mark vascular channels). **C,** Multiple fluorochrome labels administered at 2-week intervals demonstrate the incidence and rates of bone formation. **D,** This microradiograph shows an array of concentric secondary osteons (haversian systems) characteristic of rapidly remodeling cortical bone. Primary *(p)* and beginning secondary *(s)* mineralization are more radiolucent and radiodense, respectively. *(From Roberts WE et al:* Bone biodynamics in orthodontic and orthopedic treatment: craniofacial growth series*, vol 27, Ann Arbor, 1991, University of Michigan Press.)*

(5 mg/kg, bright green); (3) xylenol orange (60 mg/kg, orange); (4) alizarin complexone (20 mg/kg, red); (5) demeclocycline (10 mg/kg, gold); and (6) oxytetracycline (10 mg/kg, dull or greenish yellow). The multiple-fluorochrome method (sequential use of a variety of different colored labels) is a powerful method of assessing bone growth, healing, functional adaptation, and response to applied loads.[7,25]

Microradiography

High-resolution images require polished sections approximately 100 µm thick. Differential radiographic attenuation shows that new bone is less mineralized than mature bone. Newly formed bone matrix is osteoid and requires about 1 week of maturation to become mineralized bone matrix. Depending on the collagen configuration of the bone matrix, osteoblasts deposit 70% to 85% of the eventual mineral complement by a process called *primary mineralization.*[7,24] Secondary mineralization (mineral maturation) completes the maturation process in about 8 months by a crystal growth process (see Figure 26-15, *D*). Because the strength of bone tissue is related directly to mineral content, the stiffness and strength of an entire bone depends on the distribution and relative degree of mineralization of its osseous tissue.[26] The initial strength of new bone is the result of the cell-mediated process of primary mineralization, but its ultimate strength is dictated by secondary mineralization, which is the physiochemical process of crystal growth. This concept has important clinical value in orthodontics. Fully mineralized lamellar bone (i.e., bone in steady state with respect to modeling and remodeling) is expected to be less susceptible to relapse tendencies than its woven and composite bone predecessors.[6,27] After active orthodontic therapy, retaining dental corrections for at least 6 to 8 months is important to allow for mineral maturation of the newly formed bone (Figure 26-16).

Endosseous implants can be placed in the nasal bones of rabbits to serve as anchorage units to load the nasal suture. Slow sutural expansion (Figure 26-17) produces high-quality lamellar bone along the osseous

WWW.HIGHDENT.IR

Figure 26-16 Periapical radiographs comparing bone maturation at the end of active orthodontic treatment and 2 years later. **A,** At the end of treatment, large amorphous areas of relatively immature bone can be seen. **B,** After retention and restorative treatment, including endodontics, distinct definition of cortices and trabeculae is evident. *(From Roberts WE et al:* Bone biodynamics in orthodontic and orthopedic treatment: craniofacial growth series*, vol 27, Ann Arbor, Mich, 1991, University of Michigan Press.)*

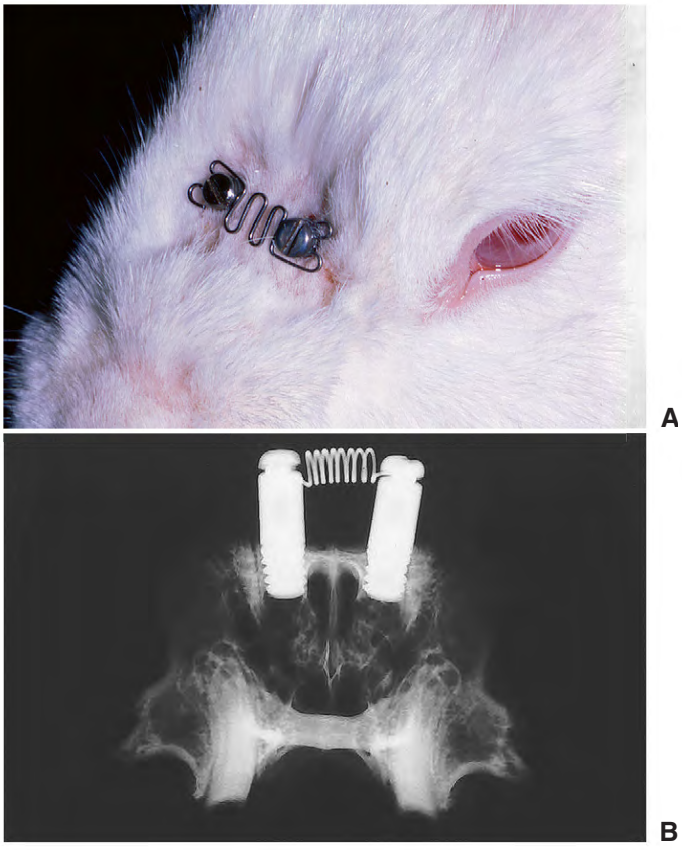


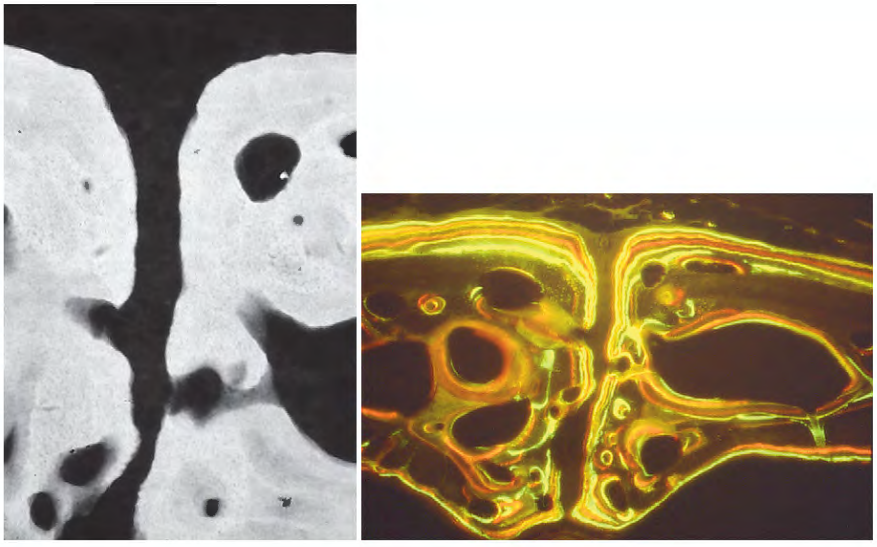
Figure 26-17 **A,** Endosseous implants in rabbit nasal bones are loaded in tension to expand the suture between the nasal bones. **B,** Radiograph of a postmortem specimen of rabbit maxilla and nasal bones shows two bilateral endosseous implants used as abutments for a coil spring that delivers a compressive load across the internasal suture. *(From Don MT:* Orthopedic anchorage with endosseous implants in rabbit nasal bones *[master's thesis], San Francisco, 1988, University of the Pacific.)*

margins of the suture and the periosteal (superior) surface of the nasal bones (Figure 26-18). Compression of the nasal bones (see Figure 26-17, *A*) is manifested by resorption along the margins of the suture (Figure 26-19). Compressive and tensile loading of the suture are associated with extensive bone modeling and remodeling of the adjacent bones. Sutural adaptation to physiologic and therapeutic loads is associated with a regional acceleration of modeling and remodeling in the adjacent bones.

The PDL is the adaptive connective tissue interface between a tooth and its supporting bone. The overall quality and relative maturation of alveolar bone supporting a rat maxillary molar is shown by a microradiographic image of a histologic section (Figure 26-20, *A*). If the same section is viewed with fluorescent light, the pattern of osteogenic activity in the bone directly supporting the PDL is visible. Sharp labels mark lamellar bone, and diffuse labels indicate woven bone. Extensive turnover (remodeling) of the alveolar process is shown by uptake of internal labels (Figure 26-20, *B*). These data reveal that the entire alveolar process responds to tooth movement. Uncoupled anabolic and catabolic modeling occurs along bone surfaces that border the periosteum and PDL. Remodeling (coupled foci of bone resorption and formation) is the process of internal turnover and adaptation.

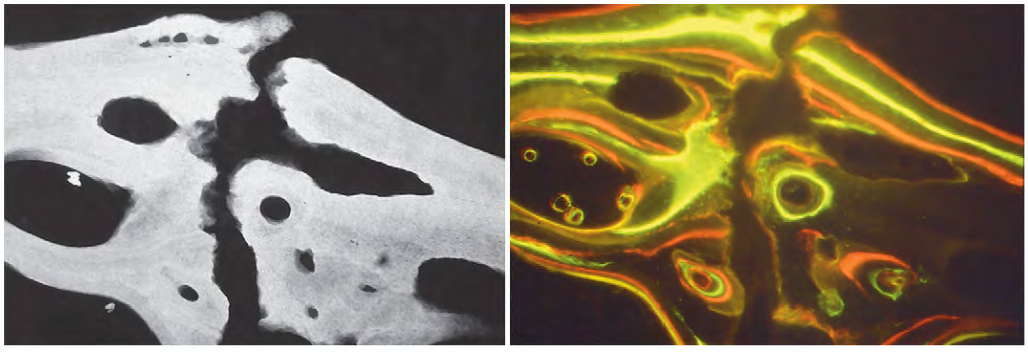
To a limited extent the temporal fossa can adapt to growth and functional loading, primarily in an anteroposterior direction, but the principal site of skeletal growth and adaptation is the mandibular condyle. In one study, multifluorochrome labeling and microradiography were used to compare bone in growing adolescent rabbits (Figure 26-21) with that in adult female rabbits who had completed growth (Figure 26-22). The adolescent primary spongiosa, the layer of endochondral bone immediately beneath the articular cartilage, is predominantly woven bone (marked by the diffuse labels in Figure 26-22). More inferiorly, the primary spongiosa is remodeled to secondary spongiosa (broad, distinct labels). Progressing deeper into the secondary spongiosa (trabecular bone), continuing remodeling of lamellar bone is shown by the sharp labels (see Figure 26-21). This progressive pattern

WWW.HIGHDENT.IR



A B

Figure 26-18 **A,** Microradiograph of the superficial portion of an internasal suture loaded in tension shows smooth bone surfaces consistent with bone apposition. **B,** Fluorescent light photomicrograph of rabbit nasal bones loaded in tension shows bone modeling and remodeling patterns associated with mechanical expansion of the suture. Bone-forming surfaces were labeled with fluorescent dyes at weekly intervals. The superior portion of the labeled suture corresponds to the microradiograph in **A.** *(From Don MT:* Orthopedic anchorage with endosseous implants in rabbit nasal bones *[master's thesis], San Francisco, 1988, University of the Pacific.)*



A B

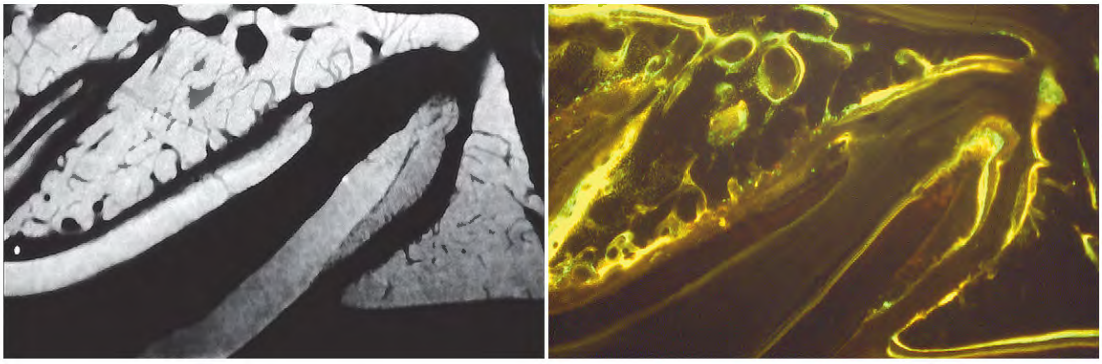
Figure 26-19 **A,** Microradiograph of an internasal suture loaded in compression shows scalloped bone surfaces consistent with bone resorption. **B,** Fluorescent light photomicrograph of rabbit nasal bones loaded in compression shows bone modeling and remodeling patterns associated with mechanical contraction of the suture. Bone-forming surfaces were labeled with fluorescent dyes at weekly intervals. *(From Don MT:* Orthopedic anchorage with endosseous implants in rabbit nasal bones *[master's thesis], San Francisco, 1988, University of the Pacific.)*

of bone modeling and remodeling is characteristic of the skeletal mechanism of long bone growth.

In contrast, the nongrowing condyles of adult animals have a much thinner subchondral plate composed primarily of woven bone (see Figure 26-22). The supporting metaphysis is composed entirely of secondary spongiosa. Bone label uptake documents a high rate of remodeling of lamellar bone.

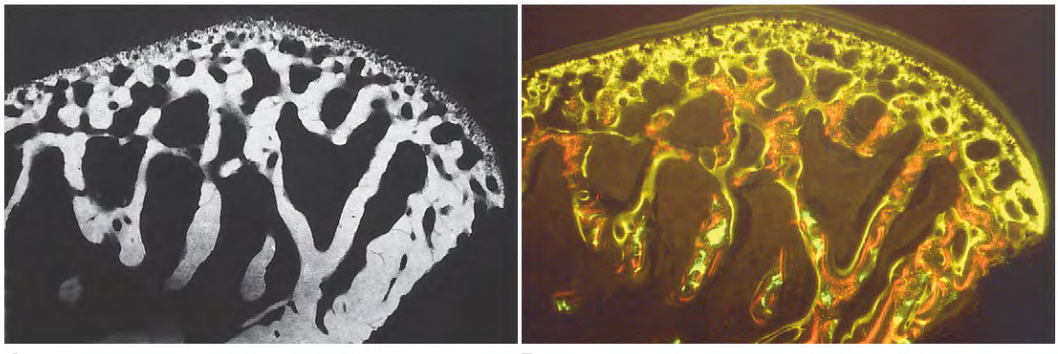
These data suggest that the mandibular condyle has a high rate of remodeling consistent with heavy functional loading. All things considered, the substantial histologic variance of functioning condyles in adolescent and adult animals indicates that the TMJ is highly adaptable. However, the presence of woven bone and diffuse labels in the thin subchondral plate of adults suggests that the mandibular condyle may be fragile

WWW.HIGHDENT.IR



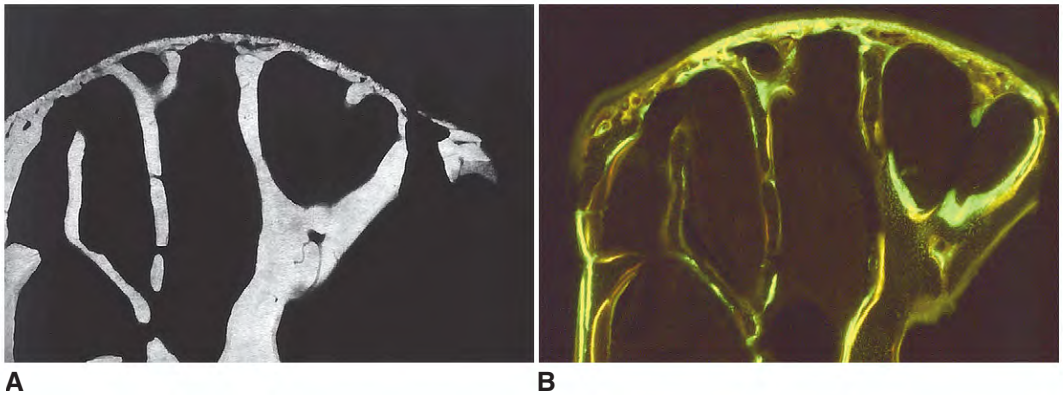
A B

Figure 26-20 **A,** Microradiograph of a midsagittal section through the mesial root of a rat maxillary first molar shows the varying degrees of mineralization of the alveolar bone and the tooth root. **B,** Fluorescent light photomicrograph of the corresponding section shows the bone modeling and remodeling patterns associated with extrusion and distal (left) tipping of the root. *(From Shimizu KA:* The effects of hypofunction and hyperfunction on the supporting structures of rat molar teeth *[master's thesis], San Francisco, 1987, University of the Pacific.)*



A B

Figure 26-21 **A,** Microradiograph of a frontal section through the mandibular condyle of a young, growing rabbit reveals that the superior cortical plate (primary spongiosa) is composed of relatively porous, primary cortical bone that is supported by a secondary spongiosa of lamellar trabeculae. **B,** Fluorescent light photomicrograph of the corresponding section shows that the superior cortical plate is composed primarily of woven bone (indistinct labels). The supporting trabeculae are composed of remodeling trabecular bone (sharp labels). *(From Larsen SJ:* The influence of age on bone modeling and remodeling *[master's thesis], San Francisco, 1986, University of the Pacific.)*



A B

Figure 26-22 **A,** Microradiograph of a frontal section through the mandibular condyle of a mature adult rabbit shows that the superior cortical plate (primary spongiosa) is composed of a thin layer of porous primary bone supported by a secondary spongiosa of lamellar trabeculae. **B,** Fluorescent light photomicrograph of the corresponding section shows that the superior cortical plate is composed primarily of woven bone (indistinct labels). The supporting trabeculae are composed of remodeling trabecular bone (sharp labels). *(From Larsen SJ:* The influence of age on bone modeling and remodeling *[master's thesis], San Francisco, 1986, University of the Pacific.)*

WWW.HIGHDENT.IR

and susceptible to degenerative changes if overloaded. Nevertheless the high rate of physiologic activity in the mandibular condyle of young and old animals may explain the remarkable ability of this joint to heal and even regenerate after injury (see Figure 26-14).

Microindentation, Backscatter Imaging, and Microcomputed Tomography

Huja et al.[21] developed a microindentation method for determining the material properties of bone in a block specimen and demonstrated that the lamellar bone within 1 mm of the surface of an implant is more compliant than the supporting bone of the jaw. Polarized microscopy demonstrates the more irregular collagen pattern of the compliant lamellar bone near the interface (Figure 26-23). Backscatter emission imaging[19] recently has been refined as a high-resolution method for assessing the bone mineral density and surface topography patterns of the osseous interface of dental implants (Figure 26-24). In another important technologic advancement, Yip et al.[20] developed a special tuning sequence for the microcomputed tomography that allows three-dimensional detection of bone mineral density patterns to a resolution of 5 µm (Figure 26-25).

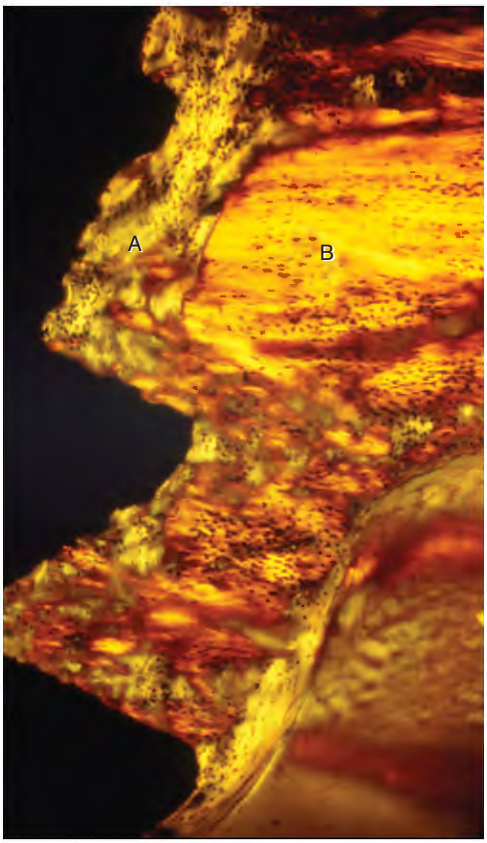


Figure 26-23 A polarized illumination photomicrograph shows the healed interface of a titanium implant in rabbit cortical bone. Note the layer of newly formed lamellar bone *(A)* formed by multidirectional remodeling within about 1 mm of the implant surface. Compare the more compliant layer of primarily mineralized lamellar bone *(A)* with the fully mineralized lamellar bone *(B)* supporting the interfacial layer.

Furthermore, this exciting new method can detect bone remodeling foci within intact specimens (Figure 26-26) and can differentiate between primary and secondary lamellar bone along the metallic surfaces of endosseous implants. Collectively, these new methods have been valuable for assessing the material properties and mineral density of bone integrating endosseous implants that are used for orthodontic and dentofacial orthopedic anchorage. However, these advanced technologies offer the promise of considerably exceeding the capability of previous histologic methods for defining the adaptive

Figure 26-24 Backscatter emission imaging of the bone surface immediately adjacent to an implant (removed) reveals the mineral density of surface topography of the rapidly remodeling interfacial layer. *(From Huja SS, Roberts WE: Mechanism of osseointegration: characterization of supporting bone with indentation testing and backscattered imaging,* Semin Orthod *10:162-173, 2004.)*

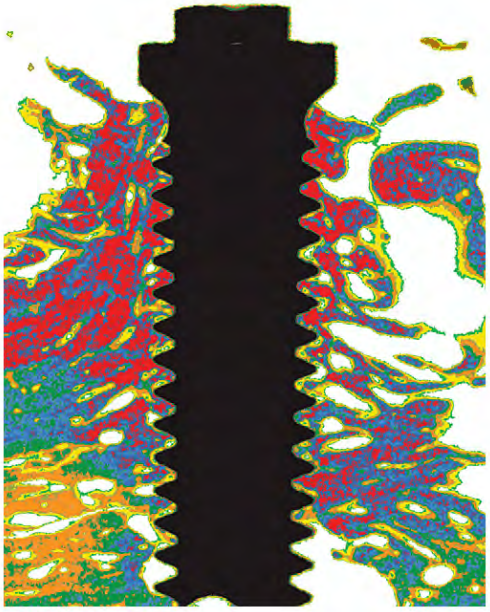


Figure 26-25 Microcomputed tomography of a section through an implant placed in canine cortical bone reveals a broad array of mineralized tissues. The original gray-level distribution has been color coded gold, blue, red, and yellow to demonstrate decreasing levels of mineral density. The method can resolve structures as small as an osteoblast. *(From Yip G, Schneider P, Roberts WE: Mechanism of osseointegration: characterization of supporting bone with indentation testing and backscattered imaging,* Semin Orthod *10:174-187, 2004.)*

WWW.HIGHDENT.IR

response of the oral and craniofacial structures to therapeutic loads.

Autoradiography

Radioactive precursors for structural and metabolic materials can be detected in tissue by coating histologic sections with a nuclear track emulsion. By localizing radioactive disintegrations, one can determine the location of the radioactive precursors (Figure 26-27). Specific radioactive labels for proteins, carbohydrates, and nucleic acids are injected at a known interval before tissue sampling is done. Qualitative and quantitative assessment of label uptake is a physiologic index of cell activity. The autoradiographic labeling procedures most often used in bone research are ^{3}H-thymidine labeling of cells synthesizing DNA (S phase cells) and ^{3}H-proline labeling of newly formed bone matrix. Bromodeoxyuridine immunocytochemistry, a nonradioactive method of labeling S phase cells in vivo (Figure 26-28), shows promise of becoming an important bone cell kinetic method of the future.[14]

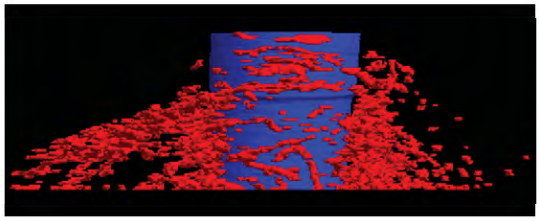


Figure 26-26 A three-dimensional microcomputed tomography view of the peri-implant radiolucent areas reveals an uneven distribution of vascular areas and remodeling foci. This image of the cervical half of a cylindric implant is consistent with the intense bone remodeling pattern focused within the center of the endosseous portion. This image was tuned to demonstrate less mineralized structures: internal vascularity including remodeling foci (cutting/filling cones). *(From Yip G, Schneider P, Roberts WE:* Semin Orthod *10:174-187, 2004.)*

Nuclear Volume Morphometry

Measuring the size of the nucleus is a cytomorphometric procedure for assessing the stage of differentiation of osteoblast precursor cells. This method has been particularly useful for assessing the mechanism of osteogenesis in orthodontically activated PDLs (Figure 26-29). Preosteoblasts (C and D cells) have significantly larger nuclei than committed osteoprogenitor (A′) cells or their less differentiated precursors (A cells). The B cell compartment is a group of fibroblast-like cells that appear to have little or no osteogenic potential.[28] Careful cytomorphometric assessment of the size of the nucleus (Figure 26-30, *A*) has proved to be an effective means of determining the relative differentiation of PDL and other bone-lining cells.

Classification of Bone Tissue

Orthodontic tooth movement involves a cytokine-mediated bone adaptation response similar to wound healing; therefore, tooth movement is a good experimental model for understanding the types of bone formed during the postoperative bone modeling and long-term remodeling response to bone manipulative therapy. The first bone formed is relatively immature woven bone (Figure 26-31). Woven bone is compacted to form composite bone (primary ostons) and subsequently is remodeled to lamellar bone. To appreciate the biologic mechanism of bone healing and adaptation, the practitioner must have knowledge of bone types.

Woven Bone

Woven bone varies considerably in structure; it is realtively weak, disorganized, and poorly mineralized. However, it serves a crucial role in wound healing by (1) rapidly filling osseous defects; (2) providing initial continuity for fractures, osteotomy segments, and endos-

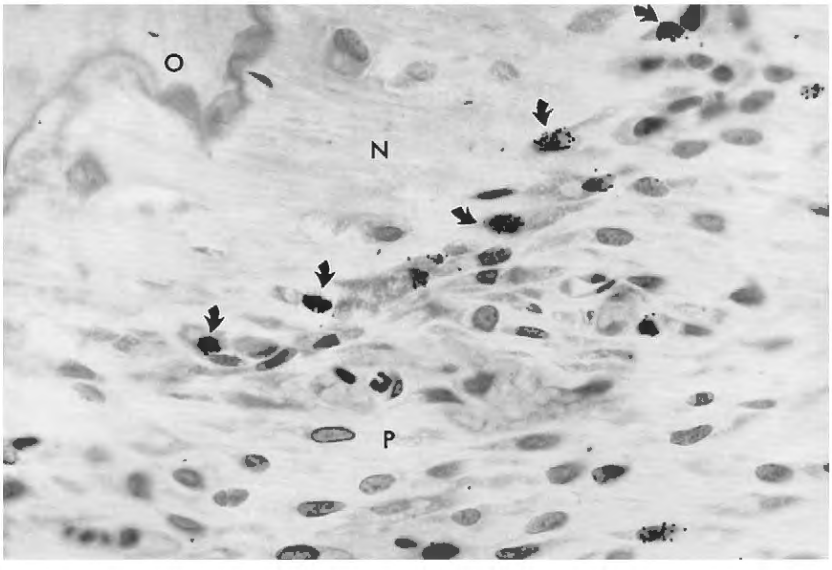


Figure 26-27 At 56 hours after initiation of orthodontic force, new bone *(N)* is forming on the original *(O)* alveolar bone surface. The ^{3}H-thymidine–labeled osteoblasts *(arrows)* are derived from preosteoblasts in the periodontal ligament *(P)* (×450).

WWW.HIGHDENT.IR

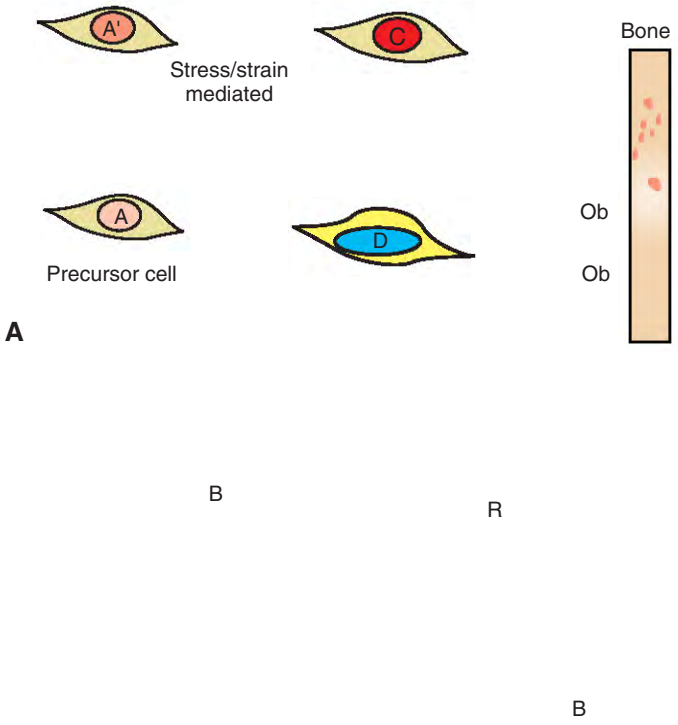


Figure 26-28 **A,** Histogenesis sequence of osteoblasts *(Ob).* Precursor cells, located around blood vessels inthe periodontal ligament (PDL), migrate toward bone as they differentiate through several stages (A→A'→C→D→Ob) to become alveolar bone forming cells (Ob). The A'→C step is believed to be mediated by stress and strain. **B,** The area between the lines is the PDL; *R,* root, *B,* bone. 5bromo-2'-deoxyuridine (BDU) is a thymidine analog taken up by cells in the PDL (dark dots) in which the nuclei are synthesizing DNA. BDU-labeled regions in the PDL are adjacent to areas of eventual bone formation (thick line segments). The BDU immunohistochemically labeled pattern of cells in the PDL of a young control rat is consistent with proliferation supporting tooth eruption. *(Adapted from Katona TR et al: Stress analysis of bone modeling response to rat molar orthodontics,* J Biomech *28:27, 1995.)*

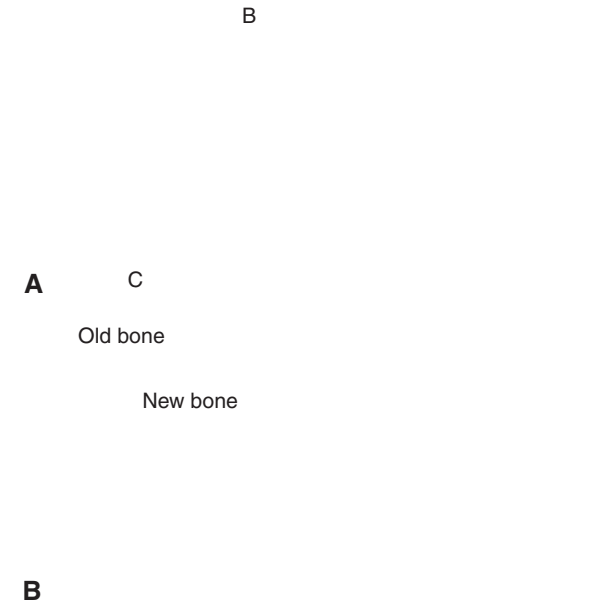


Figure 26-29 **A,** Bone *(B),* periodontal ligament *(P),* and cementum *(C)* in the control periodontium of a young adult rat (6 to 8 weeks of age) (×100). **B,** At 56 hours after application of force, new bone is noted; a ^{3}H-thymidine-labeled preosteoblast is selected from the periodontal ligament *(circle)* and magnified to 1000 times in the upper right-hand corner (×100) (*large arrow* indicates the zoom in magnification to show cellular detail).

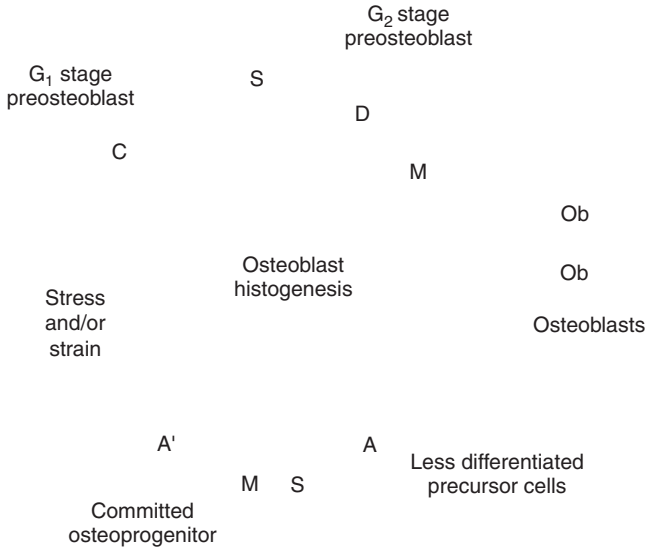


Figure 26-30 Frequency distribution of nuclear volume for fibroblast-like cells in unstimulated rat periodontal ligament. A, A', C, and D cells are a morphologic classification based on peaks in the distribution curve. The osteoblast histogenesis sequence is a progression of five morphologicallyand kinetically distinguishable cells. The process involves two DNA S phases and two mitotic *(M)* events. *(Redrawn from Roberts WE, Morey ER: Proliferation and differentiation sequence of osteoblast histogenesis under physiological conditions in rat periodontal ligament,* Am J Anat *174:105, 1985.)*

seous implants; and (3) strengthening a bone weakened by surgery or trauma. The first bone formed in response to wound healing is the woven type. Woven bone is not found in the adult skeleton under normal, steady-state conditions; rather, it is compacted to form composite bone, remodeled to lamellar bone, or rapidly resorbed if prematurely loaded.[8,29] The functional limitations of woven bone are an important aspect of orthodontic retention (see Figure 26-16), as well as postoperative healing of implants and orthognathic surgery segments.[30]

Lamellar Bone

Lamellar bone, a strong, highly organized, well-mineralized tissue, makes up more than 99% of the adult human skeleton. When new lamellar bone is formed, a portion of the mineral component (hydroxylapatite) is deposited by osteoblasts during primary mineralization (see Figure 26-15, *D*). Secondary mineralization, which completes the mineral component, is a physical process (crystal growth) that requires many months. Within physiologic limits, the strength of bone

WWW.HIGHDENT.IR

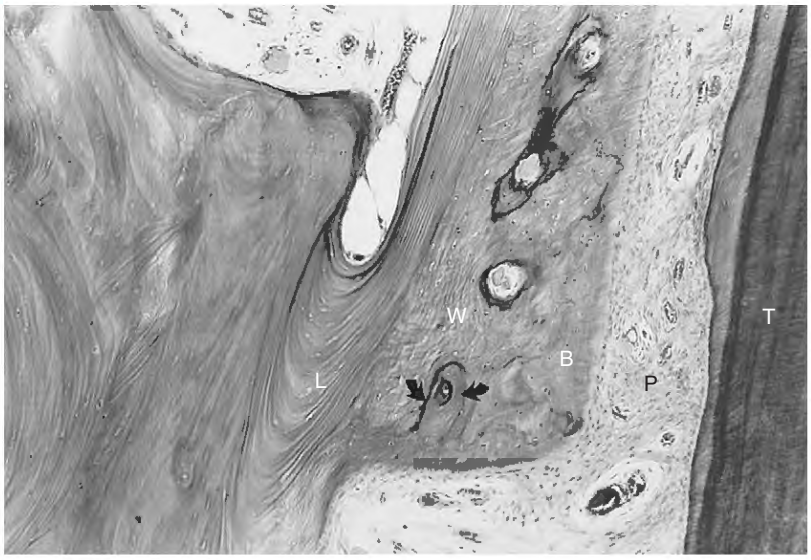


Figure 26-31 A section of human periodontium from the lower first molar region shows a typical histologic response to orthodontic tooth movement. With respect to the mature lamellar bone *(L)* on the left, the tooth *(T)* is being moved to the right. The first bone formed adjacent to the periodontal ligament *(P)* is of the woven type *(W)*. Subsequent lamellar compaction forms primary osteons of composite bone *(arrows)*. Bundle bone *(B)* is formed where ligaments, such as the periodontal ligament, are attached. *(From Roberts WE Turkey PK, Breznia KN et al: Implants: bone physiology and metabolism,* Calif Dent Assoc *J 15:54-61, 1987.)*

is related directly to its mineral content.[24,26] The relative strengths of different histologic types of osseous tissue are such that woven bone is weaker than new lamellar bone, which is weaker than mature lamellar bone.[30] Adult human bone is almost entirely of the remodeled variety: secondary osteons and spongiosa.[7,8,24] The full strength of lamellar bone that supports an endosseous implant is not achieved until about 1 year postoperatively. This is an important consideration in planning the functional loading of an implant-supported prosthesis.

Composite Bone

Composite bone is an osseous tissue formed by the deposition of lamellar bone within a woven bone lattice, a process called *cancellous compaction.*[6,31] This process is a rapid means of producing relatively strong bone in a short period.[26] Composite bone is an important intermediary type of bone in the physiologic response to functional loading (see Figure 26-31), and it usually is the predominant osseous tissue for stabilization during the early process of postoperative healing. When the bone is formed in the fine compaction configuration, the resulting composite of woven and lamellar bone forms structures known as *primary osteons.* Although composite bone may be high-quality, load-bearing osseous tissue, it is eventually remodeled into secondary osteons.[7,30]

Bundle Bone

Bundle bone is a functional adaptation of lamellar structure to allow attachment of tendons and ligaments. Perpendicular striations, called *Sharpey's fibers,* are the major distinguishing characteristics of bundle bone. Distinct layers of bundle bone usually are seen adjacent to the PDL (see Figure 26-31) along physiologic bone-forming surfaces.[32] Bundle bone is the mechanism of ligament and tendon attachment throughout the body. First-generation blade implants were thought to form a ligamentous attachement to bone, which was deemed a *pseudoperiodontium.* However, histologic studies could not demonstrate any bundle bone attaching fibrous conective tissue to bone at the interface. Because the fibrous tissue encapsulation had no physiologic role, it was actually scar tissue, which was equivalent to a nonunion in a failed facture repair.

SKELETAL ADAPTATION: MODELING AND REMODELING

Skeletal adaptation to the mechanical environment is achieved through changes in (1) bone mass, (2) geometric distribution, (3) matrix organization, and (4) collagen orientation of the lamellae. In addition to these adaptive mechanisms that influence bone formation, the mechanical properties of osseous structures change as a result of maturation, function, aging, and pathologic processes. A few physiologic and pathologic examples are (1) secondary mineralization, (2) mean bone age, (3) fatigue damage, and (4) loss of vitality (pathologic hypermineralization).[33]

Trabecular and cortical bone grow, adapt, and turn over by means of two fundamentally distinct mechanisms: modeling and remodeling. In bone modeling, independent sites of resorption and formation change the form (shape, size, or both) of a bone. In bone remodeling, a specific, coupled sequence of resorption and formation occurs to replace previously existing bone (Figure 26-32). The mechanism for internal remodeling (turnover) of dense compact bone involves axially oriented cutting and filling cones (Figure 26-33).[6] From an orthodontic perspective the biomechanical response to tooth movement involves an integrated array of bone modeling and remodeling events (Figure 26-34, *A*).

WWW.HIGHDENT.IR

Bone modeling is the dominant process of facial growth and adaptation to applied loads such as headgear, rapid palatal expansion, and functional appliances. Modeling changes can be seen on cephalometric tracings (see Figure 26-34, *B*), but remodeling events, which usually occur at the same time, are apparent only at the microscopic level. True remodeling usually is not imaged on clinical radiographs,[34] but can be detected with clinical scintillation scans. Constant remodeling (internal turnover) mobilizes and redeposits calcium by means of coupled resorption and formation: bone is resorbed and redeposited at the same site. Osteoblasts, osteoclasts, and possibly their precursors are thought to communicate by chemical messages known as *coupling factors.* Transforming growth factor β is thought to be a coupling factor.[35]

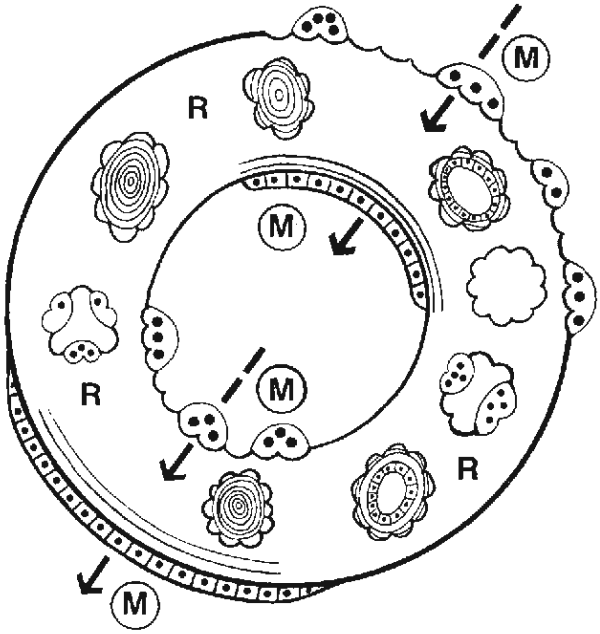


Figure 26-32 A schematic cross section of cortical bone shows surface modeling *(M),* which is the process of uncoupled resorption and formation. Remodeling *(R)* is the turnover of existing bone. *(From Roberts WE et al: Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants,* Indiana Dent Assoc J *68:19, 1989.)*

CORTICAL BONE GROWTH AND MATURATION

Enlow[31] sectioned human skulls and histologically identified areas of surface apposition and resorption. The overall patterns of bone modeling ("external remodeling") helped define the mechanisms of facial growth. Although the method could not distinguish between active and inactive modeling sites, it was adequate for determining the overall direction of regional activity in the maxilla and mandible. This method of osseous topography was a considerable advance in the understanding of surface modeling of facial bones.

Melsen[36] used microradiographic images of mineralized sections to extend the capability of the osseous topography method. Patterns of primary and secondary mineralization (as described in Figure 26-15) identified active appositional sites and provided a crude index of bone formation rates. Through the systematic study of autopsy specimens of 126 normal males and females from birth to 20 years of age, the most stable osseous structures in the anterior cranial base of growing children and adolescents were defined anatomically (Figure 26-35, *A*). This research established that the three most stable osseous landmarks for superimposition of cephalometric radiographs are (1) the anterior curvature of the sella turcica, (2) the cribriform plate, and (3) the internal curvature of the frontal bone (see Figure 26-35, *B*). In effect, this research established the gold standard for reliable superimposition on the anterior cranial base. This information is valuable for implantologists because a superimposed tracing of serial cephalometric radiographs is the most reliable means for determining when postadolescent growth is complete. The latter is essential for treatment planning implant placement during the late adolescent and early adult periods.

Roberts et al.[6,7,25] introduced simultaneous use of multiple fluorochrome labels and microradiography to assess modeling and remodeling patterns over extended periods of time. Noorda[37] applied these methods for a three-dimensional assessment of subcondylar growth of the mandible of adolescent rabbits. Twenty-week-old rabbits (early adolescents) were labeled every 2 weeks

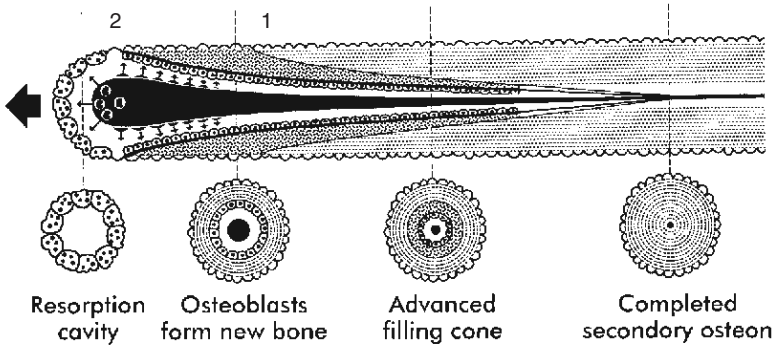


Figure 26-33 The cutting/filling cone has a head of osteoclasts that cut through the bone and a tail of osteoblasts that form a new secondary osteon. The velocity through bone is determined by measuring between two tetracycline labels (1 and 2) administered 1 week apart. *(Adapted from Roberts WE Garetto LP, Arbuckle GR et al: What are the risk factors of osteoporosis? Assessing bone health,* J Am Dent Assoc *122:59-61, 1991.)*

WWW.HIGHDENT.IR

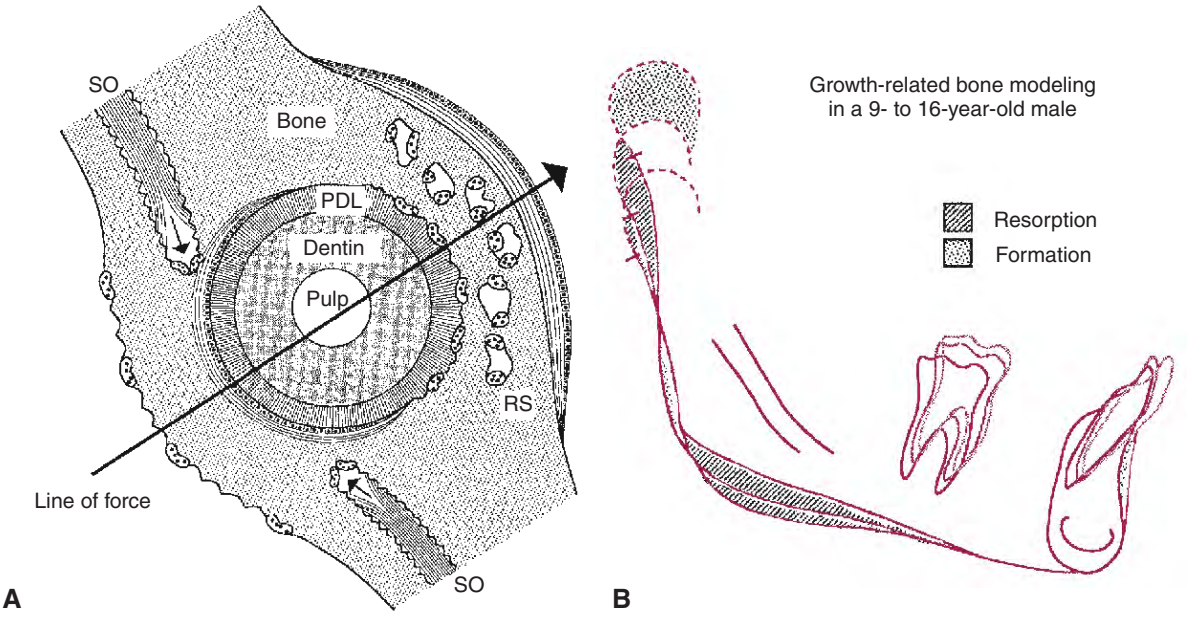


Figure 26-34 **A,** Orthodontic bone modeling, or site-specific formation and resorption, occurs along the periodontal ligament (PDL) and periosteal surfaces. Remodeling, or turnover, occurs withinalveolar bone along the line of force on both sides of the tooth. **B,** Orthopedic bone modeling related to growth in an adolescent male involves several site-specific areas of bone formation and resorption. Although extensive bone remodeling (i.e., internal turnover) also is underway, itis not evident in cephalometric radiographs superimposed on stable mandibular structures.

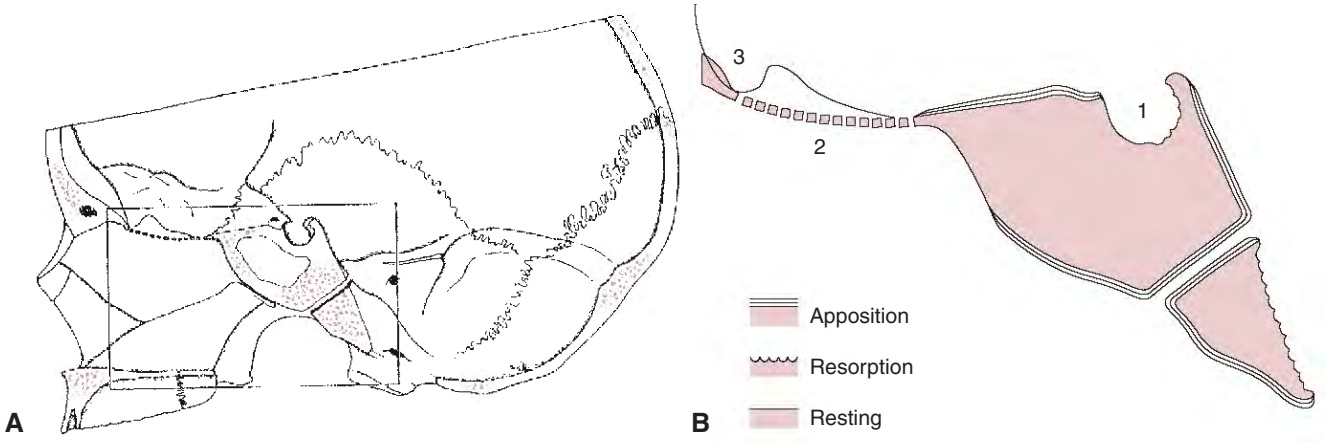


Figure 26-35 **A,** Schematic drawing of a skull showing the tissue block removed at autopsy from a series of growing children and adolescents from birth to 20 years of age. **B,** Diagrammatic representation of the bone modeling patterns of the cranial base in growing children. Histologic and microradiographic analysis established that the three most stable anatomical landmarks are *(1)* the anterior curvature of the sella turcica, *(2)* the cribriform plate, and *(3)* the internal curvature of the frontal bone. *(From Melsen B: The cranial base,* Acta Odontol Scand *32(suppl 62):103, 1974.)*

with a rotating series of six different multifluorochrome labels for 18 weeks. Cross sections of the subcondylar region (Figure 26-36, *A*) were superimposed on original, oldest-labeled, and newest-labeled bone according to fluorescent time markers (see Figure 26-36, *B*). Because all three sections were at the same relative level at a point in time, superimposition on original (unlabeled) bone and the oldest labeled bone (see Figure 26-36, *C*) provided an index of the relative amounts of bone resorbed and formed as the mandible grew superiorly (see Figure 26-36, *D*). This method provides the most accurate assessment to date of cortical bone drift over time. The major mechanism of the increase in interramal width during adolescent growth in rabbits is lateral drift of the entire subchondral region.

The Noorda study also produced important quantitative data on the rates of surface modeling (apposition and resorption) of primary bone (Figure 26-37). During the last 18 weeks of growth to adult stature, the surface apposition rate decreased from more than 25 µm per day to less than 5 µm per day (Figure 26-38, *A*). The secondary osteon census peaked at about 8 to 10 weeks (see Figure 26-38, *B*). Therefore under conditions of relatively rapid growth, primary cortical bone is

WWW.HIGHDENT.IR

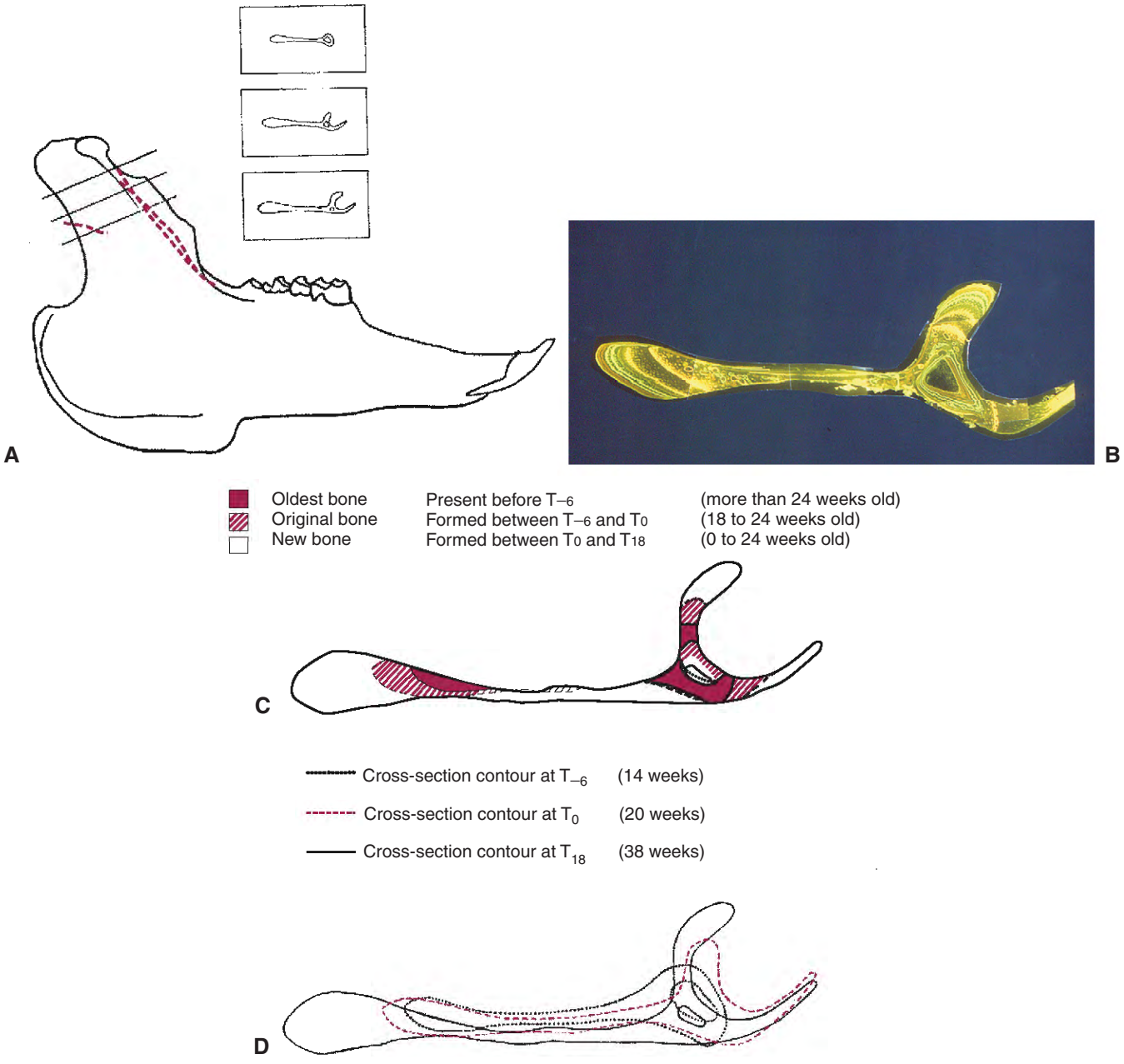


Figure 26-36 **A,** Schematic drawing of a rabbit mandible showing the plane of sectioning in the subcondylar region of the ramus. **B,** Fluorescent light photomicrographs of the most inferior section are arranged in a composite. The weekly deposition of bone labels over 4 months shows the patterns of bone modeling and remodeling associated with the growth and development of the subcondylar region. **C,** Based on the uptake of bone labels, the age of specific areas in a given cross section can be determined accurately. **D,** Because the subcondylar region of the ramus is growing superiorly, superimposition of the three sections on the oldest bone gives an estimation of the patterns of bone resorption (catabolic modeling) associated with growth of the mandibular ramus. *(From Noorda CB:* Modeling and remodeling in the cortical bone of both growing and mature rabbits *[master's thesis], San Francisco, 1986, University of the Pacific.)*

remodeled to secondary osteons in about 2 months. Remodeling therefore is a time-dependent maturation of primary cortical bone.[6,7]

There is little long-term documentation of the bone remodeling response to functional loading of implant-supported restorations. The same methods used for defining the growth and development of the rabbit mandible would provide valuable new information for the field of implantology.

Cutting and Filling Cones

The rate at which cutting and filling cones progress through compact bone is an important determinant of turnover. The progression is calculated by measuring the distance between initiation of labeled bone formation sites along the resorption arrest line in longitudinal sections.[6] Using two fluorescent labels administered 2 weeks apart in adult dogs, the velocity was 27.7 ± 1.9 µm

WWW.HIGHDENT.IR

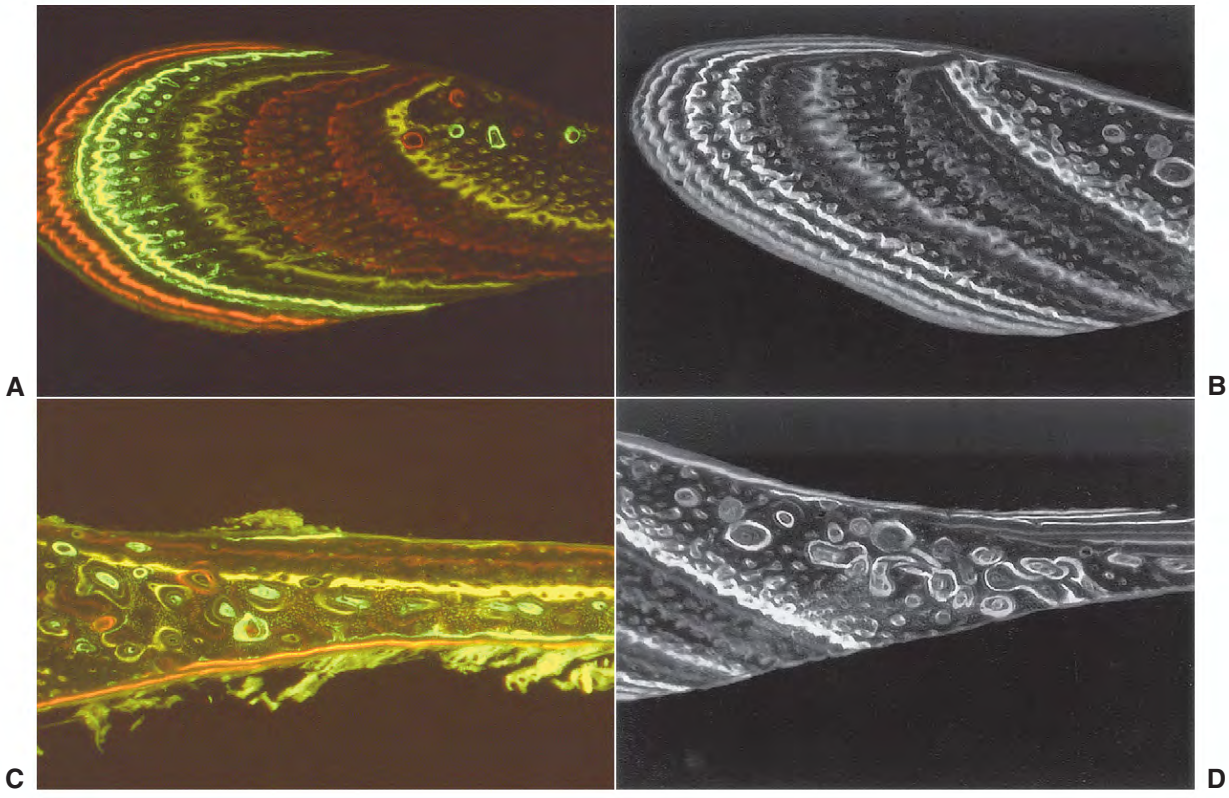


Figure 26-37 **A,** Fluorescent microscopy of weekly bone labels shows the patterns of anabolic modeling (bone apposition) in a rabbit. Note the diminishing space between the labels as growth slows and the animal achieves an adult skeletal form. **B,** A similar section from another rabbit in the same study shows the consistency of the growth pattern. **C,** In the first rabbit the adjacent microscopic field shows several sites of bone remodeling in primary cortical bone formed about 6 to 12 weeks earlier. **D,** In the second rabbit the adjacent microscopic field shows a consistent pattern of remodeling of new cortical bone at about 6 to 12 weeks after formation. *(From Noorda CB:* Modeling and remodeling in the cortical bone of both growing and mature rabbits *[master's thesis], San Francisco, 1986, University of the Pacific.)*

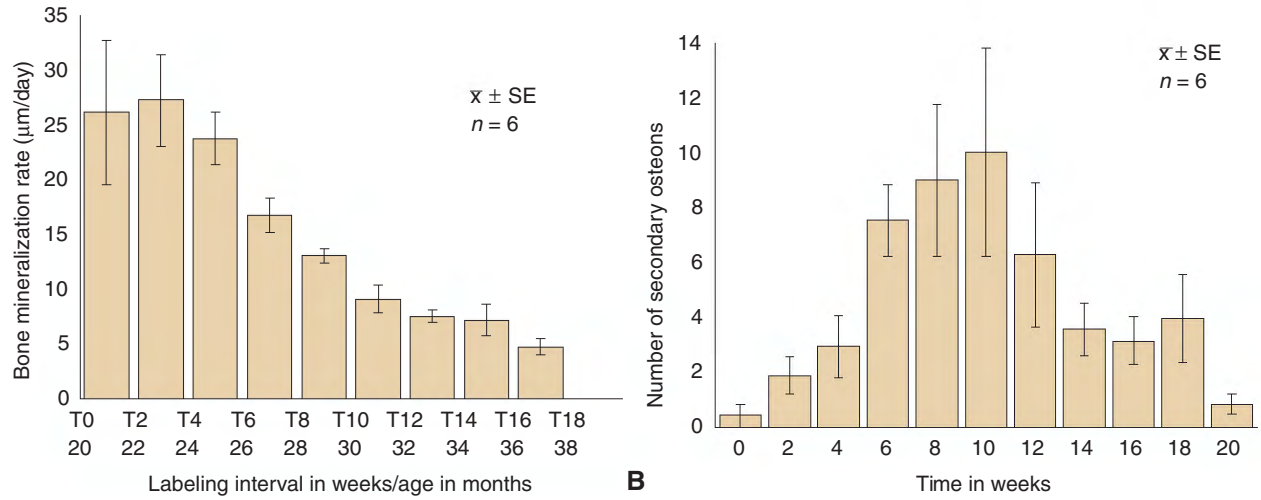


Figure 26-38 **A,** Age-related changes in the rate of periosteal apposition that occur in the posterior border of the mandibular ramus of the rabbit. Note the progressive decrease in the rate of periosteal bone apposition as the adolescent animals mature. **B,** Remodeling of new cortical bone. The highest incidence of remodeling to secondary osteons occurs when new cortical bone is 6 to 12 weeks old. *(From Noorda CB:* Modeling and remodeling in the cortical bone of both growing and mature rabbits *[master's thesis], San Francisco, 1986, University of the Pacific.)*

WWW.HIGHDENT.IR

per day (mean ± SEM [standard error of the mean], n = 4 dogs, 10 cutting and filling cones sampled from each). At this speed, evolving secondary osteons travel about 1 mm in 36 days. Newly remodeled secondary osteons (formed within the experimental period of the dog study) contained an average of 4.5 labels (administered 2 weeks apart); the incidence of resorption cavities is about one third the incidence of labeled osteons.[25] These data are consistent with a remodeling cycle of about 12 weeks in dogs[25] compared with 6 weeks in rabbits[6] and 17 weeks in humans.[7,8] This relationship is useful for extrapolating animal data to human applications. More recent experimental studies have shown that new secondary osteons may continue to fix bone labels for up to 6 months, indicating that terminal filling of the lumen is slow.[38]

Traumatic or surgical wounding usually results in intense but localized modeling and remodeling responses. After an osteotomy or placement of an endosseous implant, callus formation and resorption of necrotic osseous margins are modeling processes; however, internal replacement of the devitalized cortical bone surrounding these sites is a remodeling activity. In addition, a gradient of localized remodeling disseminates through the bone adjacent to any invasive bone procedure. This process, called *regional acceleratory phenomenon*, is an important aspect of postoperative healing.[8,39]

Modeling and remodeling are controlled by an interaction of metabolic and mechanical signals. Bone modeling is largely under the integrated biomechanical control of functional applied loads (Table 26-1). However, hormones and other metabolic agents have a strong secondary influence, particularly during periods of growth and advanced aging. Paracrine and autocrine mechanisms, such as local growth factors and prostaglandins, can override the mechanical control mechanism temporarily during wound healing.[40] Remodeling responds to metabolic mediators such as parathyroid hormone (PTH) and estrogen, primarily varying the rate of bone turnover (Box 26-1). Bone scans with ^{99}Te-bisphosphate, a marker of bone activity, indicate that the alveolar processes, but not the basilar mandible, have a high remodeling rate.[41,42] Uptake of the marker in alveolar bone is similar to uptake in trabecular bone of the vertebral column. The latter is known to remodel at a rate of about 20% to 30% per year compared with most cortical bone, which turns over at a rate of 2% to 10% per year.[43] Metabolic mediation of continual bone turnover provides a controllable flow of calcium to and from the skeleton.

Table 26-1 Control Factors for Bone Modeling

FACTOR	PEAK LOAD IN MICROSTRAIN (με)*
Mechanical†	
Disuse atrophy	<200
Bone maintenance	200 to 2500
Physiologic hypertrophy	2500 to 4000
Pathologic overload	>4000
Endocrine	
Bone metabolic hormones: PTH, vitamin D, calcitonin	
Growth hormones: somatotropin, IGF-I, IGF-II	
Sex steroids: testosterone, estrogen	
Paracrine and autocrine	
Wide variety of local agents	

PTH, Parathyroid hormone; *IGF,* insulin-like growth factor.
*με = percent deformation $\times 10^{-4}$.
†Frost's mechanostat theory (Frost HM: Skeletal structural adaptations to mechanical usage [SATMU]. 2. Redefining Wolff's law: the remodeling problem, Anat Rec 226:414, 1990).

Structural and Metabolic Fractions

The structural fraction of cortical bone is the relatively stable outer portion of the cortex; the metabolic fraction is the highly reactive inner aspect (Figure 26-39, *A*). The primary metabolic calcium reserves of the body are found in trabecular bone and the endosteal half of the cortices. Analogous to orthodontic wires, the stiffness and strength of a bone are related directly to its cross-sectional area. Diaphyseal rigidity quickly is enhanced by adding a circumferential lamella at the periosteal surface. Even a thin layer of new osseous tissue at the periosteal surface greatly enhances bone stiffness because it increases the diameter of the bone. In engineering terms, cross-sectional rigidity is related to the second moment of the area. The same general relationship of round wire diameter and stiffness (strength) is well known to orthodontists. The rigidity of a wire increases as the fourth power of diameter.[44] Therefore when a relatively rigid material (bone or wire) is doubled in diameter, the stiffness increases 16 times.

The addition of new osseous tissue at the endosteal (inner) surface has little effect on overall bone strength. Structurally, the long bones and mandible are modified

Box 26-1 Control Factors for Bone Remodeling

Metabolic

Parathyroid hormone: ↑ activation frequency
Estrogen: ↓ activation frequency

Mechanical

<1000 με: more remodeling
>2000 με: less remodeling

WWW.HIGHDENT.IR

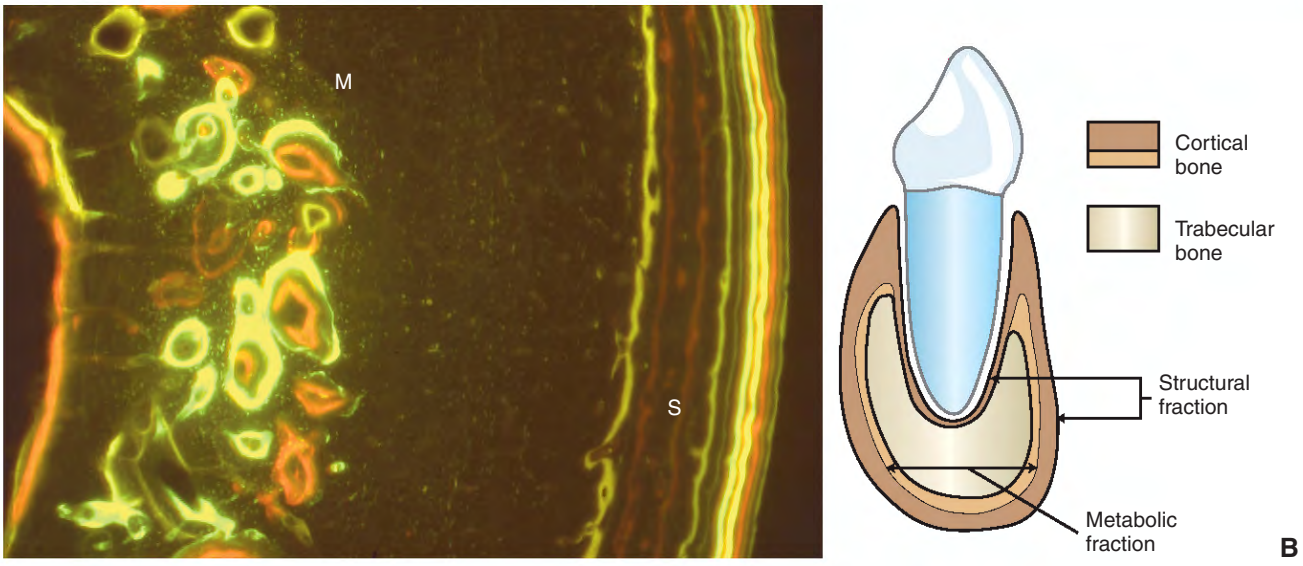


Figure 26-39 **A,** The structural *(S)* and metabolic *(M)* fractions of cortical bone are revealed by multiple fluorochrome labeling of a rabbit femur during the late growth and early adult periods. Continuing periosteal bone formation (right) contributes to structural strength, and high remodeling of the endosteal half of the compacta provides a continual supply of metabolic calcium. **B,** Structural and metabolic fractions of bone in the mandible. *(Adapted from Roberts WE et al:* Bone dynamics in orthodontic and orthopedic treatment: craniofacial growth series, *vol 27, Ann Arbor, Mich, 1991, University of Michigan Press.)*

tubes—an optimal design for achieving maximal strength with minimal mass.[26] Within limits, loss of bone at the endosteal surface or within the inner third of the compacta has little effect on bone rigidity. The inner cortex can be mobilized to meet metabolic needs without severely compromising bone strength (see Figure 26-39, *B*); this is the reason patients with osteoporosis have bones with a normal diameter but thin cortices. Even under severe metabolic stress, the body follows a cardinal principle of bone physiology: *maximal strength with minimal mass.*[45]

BONE METABOLISM

Restoration of esthetics and function with implant-supported prostheses requires substantial bone manipulation. The biomechanical response to altered function and applied loads depends on the metabolic status of the patient. Bone metabolism is an important aspect of clinical medicine that is directly applicable to implant dentistry. This section discusses the fundamentals of bone metabolism with respect to clinical practice.

The skeletal system is composed of highly specialized mineralized tissues that have structural and metabolic functions. Structurally, lamellar, woven, composite, and bundle bone are unique types of osseous tissue adapted to specific functions. Bone modeling and remodeling are distinct physiologic responses to integrated mechanical and metabolic demands. Biomechanical manipulation of bone is the physiologic basis of stomatognathic reconstruction. However, before addressing dentofacial considerations, the clinician must assess the patient's overall health status. Implantology is bone-manipulative therapy, and favorable calcium metabolism is an important consideration. Because of the interaction of structure and metabolism, a thorough understanding of osseous structure and function is fundamental to patient selection, risk assessment, treatment planning, and retention of desired dentofacial relationships.[45,46]

Bone is the primary calcium reservoir in the body (Figure 26-40). Approximately 99% of the calcium in the body is stored in the skeleton. The continual flux of bone mineral responds to a complex interaction of endocrine, biomechanical, and cell-level control factors that maintain the serum calcium level at about 10 mg/dL (10 mg%).

Calcium homeostasis is the process by which mineral equilibrium is maintained. Maintenance of serum calcium levels at about 10 mg/dL is an essential life support function. Life is thought to have evolved in the sea; calcium homeostasis is the mechanism of the body for maintaining the primordial mineral environment in which cellular processes evolved.[45] Calcium metabolism is one of the fundamental physiologic processes of life support. When substantial calcium is needed to maintain the critical serum calcium level, bone structure is sacrificed (see Figure 26-40). The alveolar processes and basilar bone of the jaws also are subject to metabolic bone loss.[47] Even in cases of severe skeletal atrophy, the outer cortex of the alveolar process and the lamina dura around the teeth are preserved. This preservation is analogous to the thin cortices characteristic of osteoporosis.

Calcium homeostasis is supported by three temporally related mechanisms: (1) rapid (instantaneous) flux of

WWW.HIGHDENT.IR

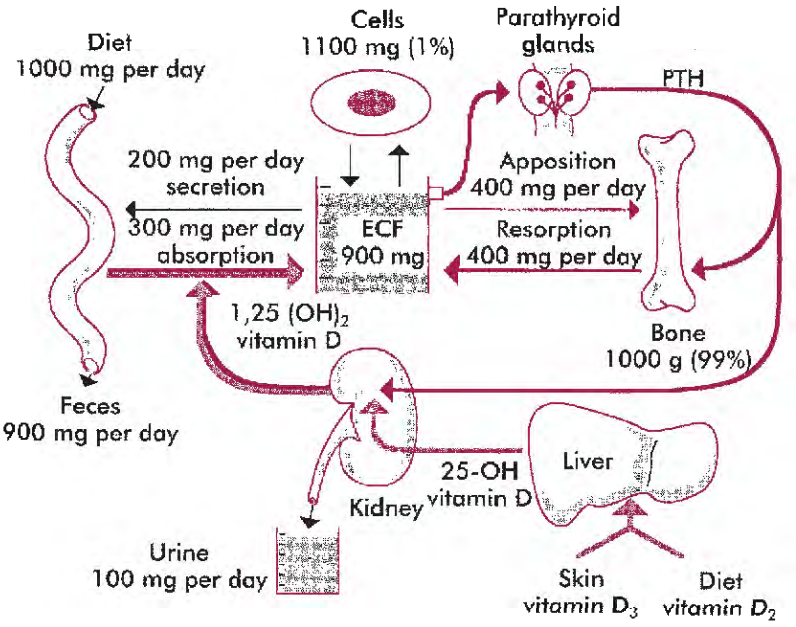


Figure 26-40 Calcium metabolism is a complex physiologic process. Maintaining zero calcium balance requires optimal function of the gut, parathyroid glands, bone, liver, and kidneys. Parathyroid hormone (PTH) and the active metabolite of vitamin D, 1,25 dihydroxycholecalciferol, are the major hormones involved. *(Adapted from Roberts WE et al:* Bone dynamics in orthodontic and orthopedic treatment: craniofacial growth series, *vol 27, Ann Arbor, Mich, 1991, University of Michigan Press.)*

calcium from bone fluid (which occurs in seconds); (2) short-term response by osteoclasts and osteoblasts (which extends from minutes to days); and (3) long-term control of bone turnover (over weeks to months) (Figure 26-41). Precise regulation of serum calcium levels at about 10 mg/dL is essential for nerve conductivity and muscle function. A low serum calcium level can result in tetany and death. A sustained high serum calcium level often is a manifestation of hyperthyroidism and some malignancies. Hypercalcemia may lead to kidney stones and dystrophic calcification of soft tissue. Normal physiology demands precise control of the serum calcium level.[45,46,48]

Instantaneous regulation of calcium homeostasis is accomplished in seconds by selective transfer of calcium ions into and out of bone fluid (see Figure 26-41, *B*). Bone fluid is separated from extracellular fluid by osteoblasts or relatively thin bone-lining cells (the latter are thought to be atrophied remnants of osteoblasts). A decrease in the serum calcium level stimulates secretion of PTH, which enhances transport of calcium ions from bone fluid into osteocytes and bone-lining cells. The active metabolite of vitamin D (1,25-dihydroxycholecalciferol [1,25DHCC]) enhances pumping of calcium ions from bone-lining cells into the extracellular fluid. By means of this sequence of events, calcium is transported across the bone-lining cells, resulting in a net flux of calcium ions from bone fluid to extracellular fluid. Within physiologic limits, support of calcium homeostasis is possible without resorption of bone. Radioisotope studies have confirmed that bone contains a diffuse mineral component that can be mobilized or redeposited without osteoblastic and osteoclastic activity.[24] However, a sustained negative calcium balance can be compensated for only by removing calcium from bone surfaces.[45,46]

Short-term control of serum calcium levels affects rates of bone resorption and formation within minutes through the action of the three calcific hormones: PTH, 1,25-DHCC, and calcitonin. Calcitonin, a hormone produced by interstitial cells in the thyroid gland, is believed to help control hypercalcemia by transiently suppressing bone resorption. Parathyroid hormone, acting in concert with 1,25-DHCC, accomplishes three important tasks: it (1) enhances osteoclast recruitment from promonocyte precursors[49]; (2) increases the resorption rate of existing osteoclasts; and (3) may suppress the rate at which osteoblasts form bone.[45,46]

Long-term regulation of metabolism has profound effects on the skeleton. Biomechanical factors (e.g., normal function, exercise, posture, habits), noncalcific hormones (e.g., sex steroids, growth hormone), and the metabolic mechanisms previously discussed (see Figures 26-40 and 26-41) dictate mass, geometric distribution, and the mean age of bone (Figure 26-42). Mass and geometric distribution of bone are influenced strongly by load history (biomechanics) and sex hormone status. Parathyroid hormone is the primary regulator of the frequency of remodeling (see Box 26-2). Because the adult skeleton is composed almost entirely of secondary (remodeled) bone, the PTH-mediated activation frequency determines mean bone age. Bone age is an important determinant of fragility because old bone presumably has been weakened by fatigue damage.[45,46]

CALCIUM CONSERVATION

Calcium conservation is the aspect of bone metabolism that involves preservation of skeletal mass. A failure in calcium conservation because of one problem or a

WWW.HIGHDENT.IR

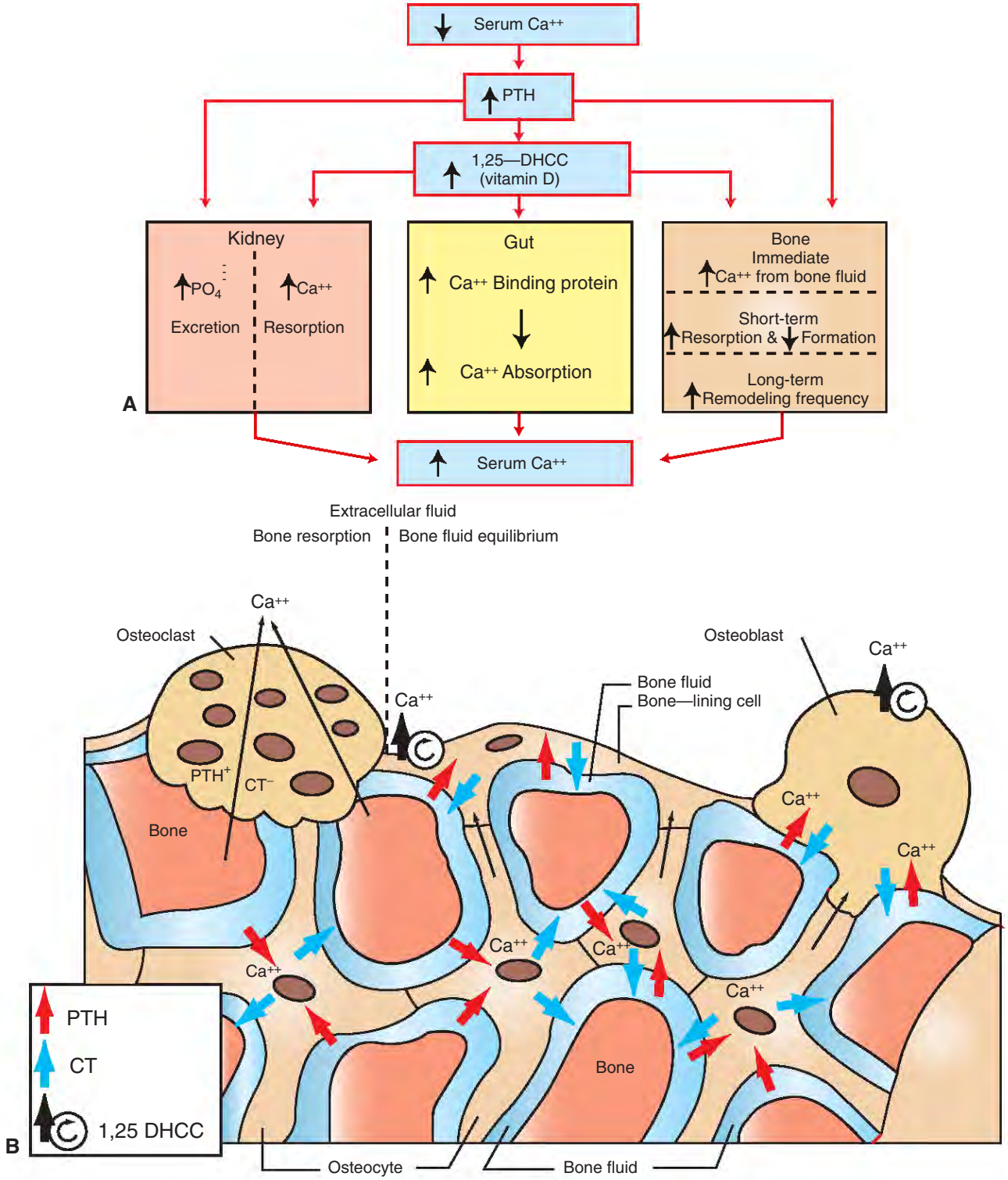


Figure 26-41 **A,** A flowchart of calcium homeostasis shows the roles of parathyroid hormone (PTH), vitamin D, and the kidneys, gut, and bone in maintaining serum calcium levels. Note that bone has immediate, short-term, and long-term responses. **B,** PTH, the active metabolite of vitamin D (1,25 DHCC), and calcitonin (CT) play active roles in transporting ionic calcium(Ca^{++}) between the bone fluid and extracellular fluid compartments. This is the mechanism of immediate homeostatic control of the serum calcium level. *(Redrawn from Roberts WE, Simmons KE, Garetto LP et al: Bone physiology and metabolism in dental implantology: risk factors for osteoporosis and other metabolic bone diseases,* Implant Dent *1:11, 1992.)*

WWW.HIGHDENT.IR

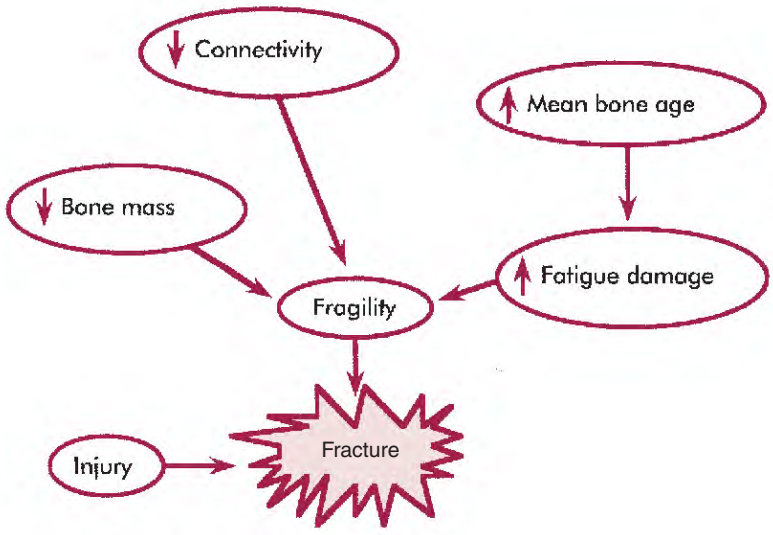


Figure 26-42 The structural integrity (fracture resistance) of osseous tissue is affected by bone mass, connectivity (geometric distribution), mean bone age, and fatigue damage. Fragile bone may be fractured by normal functional loads or minor injuries.

combination of metabolic and biomechanical problems may leave a patient with inadequate bone mass for reconstructive dentistry.

The kidney is the primary calcium conservation organ in the body. Through a complex series of excretion and endocrine functions, the kidney excretes excess phosphate while minimizing the loss of calcium (see Figures 26-40 and 26-41, *A*). A patient with impaired renal function often is a high risk for osseous manipulative procedures such as endosseous implants or orthognathic surgery. Because of its components of secondary hyperparathyroidism and impaired vitamin D metabolism, kidney disease may result in poor bone quality, a condition often referred to as *renal osteodystrophy.*[45,46,50]

Absorption from the small intestine is the primary source of exogenous calcium and phosphate. Phosphate is absorbed passively and rarely is deficient. Optimal calcium uptake, however, requires an active absorption mechanism. A unique factor involved in the gut absorption process is calcium-binding protein, formed in response to the active metabolite of vitamin D.[51] Common clinical profiles associated with poor calcium absorption include a diet deficient in dairy products, vitamin D deficiency, liver disease, and kidney problems.[16,45,46]

Under normal physiologic conditions, the body expends about 300 mg of calcium per day, primarily as a result of secretory processes in the intestines and kidneys. To maintain the serum calcium level, this 300-mg deficit must be recovered by absorption from the gut. However, absorption of calcium from the gut depends on vitamin D and is only about 30% efficient. If less than about 300 mg per day of calcium is absorbed from the intestine, the serum calcium level drops, PTH secretion ensues, and the necessary calcium is removed from the bones (see Figure 26-40).

Positive calcium balance normally occurs during the growing period and for about 10 years thereafter. The skeletal mass of prepubertal children can be enhanced with regular calcium supplements.[52] Peak skeletal mass is attained between 25 and 30 years. After the early adult years, natural aging is associated with a slightly negative calcium balance that progressively erodes bone volume throughout life. Zero calcium balance (see Figure 26-40) is the ideal metabolic state for maintaining skeletal mass. Preservation of bone requires a favorable diet, endocrine balance, and adequate exercise.[45,46]

Diet

Animal studies have documented endosteal bone loss of the alveolar processes of dogs maintained on a low-calcium diet.[47] These data indicate that a low-calcium diet may have severe effects on the bones of the oral cavity. In adult humans the current recommended daily allowance of calcium is 1000 to 1500 mg per day (Table 26-2). Growing adolescents, pregnant or lactating women, and particularly pregnant teenagers need as much as 1500 mg per day. Postmenopausal women who are not receiving estrogen replacement therapy should get 1500 mg of calcium per day. In the United States, dairy products supply about 70% of dietary calcium. As previously mentioned, dietary phosphate deficiency is a rare problem.[16,27,45,46]

Obesity has few health benefits; however, it is a protective factor against osteoporosis. This most probably is a result of the high rates of mechanical loading needed to support an overweight body. Slight stature, however, is a risk factor for osteoporosis. Because weight control is a concern for the population at risk, the calcium-to-calorie ratio is an important consideration in dietary counseling (Table 26-3). The most favorable dairy products with respect to the calcium-to-calorie

WWW.HIGHDENT.IR

Table 26-2 Dietary Calcium Recommendations

GROUP	AGE	mg/DAY
Infants	Birth to 6 months	400
	6 to 12 months	600
Children	1 to 5 years	800
	6 to 10 years	800 to 1200
Adolescents and young adults	11 to 24 years	1200 to 1500
Men	25 to 65 years	1000
Women	25 to 50 years	1000
Pregnant or lactating		1200 to 1500
Postmenopausal		
Receiving estrogen replacement therapy		1000
Not receiving estrogen replacement therapy		1500
Men and women	>65 years	1500

From National Institutes of Health: Consensus statement: optimal calcium intake, 1994.

Table 26-3 Calcium and Calorie Content of Common Dairy Products

PRODUCT	CALCIUM (mg)	CALORIES	RATIO (CALCIUM:CALORIES)
Milk			
Whole, 3.3% fat, 1 cup	291	150	1.9:1
Low-fat, 2% fat, 1 cup	297	120	2.5:1
Buttermilk, 1 cup	285	100	2.8:1
Skim milk, 0% fat, 1 cup*	302	85	3.6:1
Cheese			
American, pasteurized process, 1 oz	174	104	1.7:1
Cheddar, 1 oz	204	115	1.8:1
Cottage, creamed, 4% fat, 1 cup	135	235	0.6:1
Cottage, low-fat, 2%, 1 cup	155	205	0.8:1
Monterey Jack, 1 oz	212	106	2.0:1
Mozzarella, part-skim, 1 oz*	207	80	2.6:1
Swiss, 1 oz*	272	105	2.6:1
Yogurt			
Plain, low-fat, 8 oz	415	145	2.9:1
Plain, non-fat, 8 oz*	452	125	3.6:1
Fruit, low-fat, 8 oz	345	230	1.5:1

*Food or foods with most favorable calcium-to-calorie ratios in each category. Data provided by the American Dairy Association.

ratio are nonfat milk, part-skim mozzarella cheese, Swiss cheese, and plain, low-fat yogurt. Typical servings of these products have about 300 mg of calcium and 100 to 200 calories. Some adults avoid milk because of intolerance to lactose. These patients often assume that they have a milk allergy, which should be determined according to symptoms. Lactose intolerance usually is manifested by an upset stomach rather than a classic anaphylaxis. Even patients who are intolerant of milk usually can tolerate cultured products such as buttermilk, cheese, and yogurt. Calcium supplements are indicated if a patient is allergic to milk or fails to achieve a calcium-sufficient diet for any other reason. Other foods, particularly green, leafy vegetables (e.g., turnip greens, spinach), contain substantial amounts of calcium, but the calcium is tightly bound and little ionic calcium is absorbed. In effect, to consume adequate calcium in a diet that excludes dairy products is difficult.[45,46] Calcium supplements of many varieties are available in pharmacies and health food stores, and most provide adequate calcium when used as directed. However, consumers should beware of toxic contaminants in some natural supplements, such as bone meal and dolomite, which may contain significant amounts of lead, arsenic, or other heavy metals. Among the least expensive, readily tolerated supplements are calcium carbonate (e.g., Tums, calcium-rich Rolaids). To determine the amount of elemental calcium in a

WWW.HIGHDENT.IR

supplement, consumers must remember to use the molecular weight. For instance, calcium carbonate is only 40% calcium; this means that a 500-mg tablet provides only 200 mg of calcium.[45,46]

Endocrinology

Peptide hormones (e.g., PTH, growth hormone, insulin, calcitonin) bind receptors at the cell surface and may be internalized with the receptor complex. Steroid hormones (e.g., vitamin D, androgens, estrogens) are lipid soluble and pass through the plasma membrane to bind receptors in the nucleus.[45,46] PTH increases serum calcium by direct and indirect vitamin D–mediated effects. Vitamin D (cholecalciferol) originally was thought to be an essential dietary factor. However, vitamin D is not a vitamin at all; it is a hormone. Cholecalciferol is synthesized in skin irradiated by ultraviolet light, hydroxylated in the liver at the No. 25 position, and then hydroxylated in the kidney at the No. 1 position to produce the active metabolite 1,25-DHCC. The last step is feedback controlled; hydroxylation at the No. 1 position is induced by a low serum calcium level, probably through PTH (see Figure 26-40). Clinically a major effect of 1,25-DHCC is induction of active absorption of calcium from the gut. Because of the complexity of vitamin D synthesis and the metabolic pathway, calcium adsorption may be inhibited at many levels; some of these inhibitors are (1) lack of skin exposure to adequate sunlight of the proper wavelength; (2) failure to consume vitamin D through the diet, thereby not compensating for the lack of vitamin D synthesis; (3) a genetic defect in the skin; (4) liver disease; and (5) kidney failure.[45,46]

Sex hormones have profound effects on bone. Androgens (testosterone and other anabolic steroids) build and maintain musculoskeletal mass. The primary hypertrophic effect of androgens is to increase muscle mass. The anabolic effect on bone is a secondary biomechanical response to increase loads generated by the enhanced muscle mass. Estrogen, however, has a direct effect on bone; it conserves skeletal calcium by suppressing the activation frequency of bone remodeling.[53] At menopause, enhanced remodeling activation increases turnover.[54] Because a slight negative calcium balance is associated with each remodeling event, a substantial increase in the turnover rate can result in rapid bone loss, leading to symptomatic osteoporosis. Even young women are susceptible to significant bone loss if the menstrual cycle (menses) stops.[55] Bone loss is a common problem in women who have low body fat and who exercise intensely (e.g., running, gymnastics) and in women who are anorexic.[56] Clearly, estrogen protects the female skeleton from bone loss during the childbearing years. Lack of menses in women of any age is a high-risk factor for the development of osteoporosis later in life.[45,46]

Estrogen replacement therapy is widely recommended for calcium conservation and the prevention of osteoporosis in postmenopausal women.[57,58] A major concern of many patients and of some physicians is the relationship of estrogen therapy to the incidence and progression of breast cancer.[59] It generally is accepted that estrogen replacement therapy increases the risk of breast cancer by about 2% but decreases the risk of osteoporosis, heart disease, colon cancer, and Alzheimer disease by as much as 50%. For many women, estrogen replacement therapy remains a wise health measure.

The antiestrogen tamoxifen is used to treat some forms of breast cancer. Fortunately, in postmenopausal women, tamoxifen has a beneficial effect on bone similar to that of estrogen.[60] Recently raloxifene (Evista) has been shown to reduce the risk of osteoporosis and heart disease without increasing the risk of breast cancer. Some studies have even shown a substantial anticancer protective effect.[61]

SKELETAL COMPROMISE

Skeletal health is related to diet, exercise, lifestyle, and proper functioning of numerous organ systems. To provide optimal support over a broad spectrum of conditions, the skeletal system has evolved complex mechanical, endocrine, and cell-level regulatory mechanisms. A failure of one or more of these homeostatic mechanisms can result in metabolic bone disease. Low skeletal mass and/or poor osteogenic capability may make some patients poor candidates for orthodontic or orthognathic procedures. Skeletally compromised patients who seek unrelated dental treatment provide dentists with unique diagnostic opportunities. Timely medical referral of individuals with high-risk profiles can result in substantial health benefits. If osteopenia is confirmed, corrective medical therapy can be started before the onset of the debilitating symptoms associated with osteoporosis.[45,46]

The concept of structural and metabolic fractions (see Figure 26-39) has considerable clinical significance. The dietary requirement for calcium increases during the growing years. A high dietary calcium intake (1200 mg per day) is essential during the adolescent period (see Table 26-2) to provide structural strength without compromising the metabolic reserve. Pregnancy and lactation before the age of 19 may be precipitating factors for osteopenia later in life. Multiple births during the teenage years are of particular concern. Under these circumstances, young women may fail to deposit sufficient skeletal reserves to withstand the sustained negative calcium balance that follows menopause.[45,46,61]

Although the metabolic fraction of cortical bone can make a substantial contribution, the major source of serum calcium under steady-state conditions is trabecular bone. The primary reason for this is the differential

WWW.HIGHDENT.IR

remodeling rates. Cortical bone turns over about 2% to 10% per year, whereas trabecular bone, which is much more active, remodels at 20% to 30% per year.[62] Because it is more labile, trabecular bone is more susceptible to loss under conditions of negative calcium balance. For this reason, patients with osteoporosis have a tendency to suffer structural failure at sites primarily dependent on trabecular bone: the spine (compression fracture), the wrist (Colles' fracture), and the hip (femoral neck fracture). Degenerative changes in the TMJ have not been related directly to skeletal atrophy. However, some relationship is likely because these problems tend to affect the same high-risk group—postmenopausal women.[45,46,63]

Women depend on estrogen to maintain skeletal mass. Lack of normal menses, even in young women, usually indicates an estrogen deficiency and probable negative calcium balance. Numerous national and international consensus conferences[64,57] have recommended that most postmenopausal Caucasian and Asian women be treated with estrogen to prevent osteoporosis. Surveys indicate that some physicians fail to prescribe estrogen for their postmenopausal patients[65]; however, the most common problem is the failure of many women to comply despite the recommendations of their physicians. For this reason many women in Western society are estrogen deficient. About 20% will develop frank osteoporosis, and as many as 50% will have some signs or symptoms.[46] All health care providers should be concerned particularly about the skeletal status of postmenopausal white and Asian women. However, even low-risk groups, such as men and black women, have an incidence of osteoporosis that approaches 5%. Osteopenia and osteoporosis therefore are significant health risks for almost everyone. Bone metabolic evaluation is an important diagnostic concern for all patients being considered for dental implants or any other bone-manipulative therapy.[16,45,66]

METABOLIC BONE DISEASE

Osteoporosis is a generic term for low bone mass (osteopenia). The most important risk factor for the development of osteoporosis is age: after the third decade, osteopenia is related directly to longevity. Other high-risk factors are (1) a history of long-term glucocorticoid treatment, (2) slight stature, (3) smoking, (4) menopause or dysmenorrhea, (5) lack of or little physical activity, (6) low-calcium diet, (7) excessive consumption of alcohol, (8) vitamin D deficiency, (9) kidney failure, (10) liver disease (cirrhosis), and (11) a history of fractures. These risk factors are effective in identifying 78% of those with the potential for osteopenia.[61,67] This is a particularly good screening method for skeletally asymptomatic dental patients. However, one must realize that more than 20% of individuals who eventually develop osteoporosis have a negative history for known risk factors. Any clinical signs or symptoms of low bone mass (e.g., low radiographic density of the jaws, thin cortices, excessive bone resorption) are grounds for referral. A thorough medical workup, including bone mineral density measurement, usually is necessary to establish the diagnosis of osteopenia. The term *osteoporosis* usually is reserved for patients with evidence of fracture or other osteoporotic symptoms. The treatment of metabolic bone diseases such as osteoporosis depends on the causative factors. Medical management of these often complex disorders is best handled by physicians specifically trained in bone metabolism.[45,46]

Because the loss of teeth is an important risk factor for osteoporosis, dental patients, especially adult women, are at high risk for developing osteoporosis. A sampling of all adult female dental patients at a midwestern dental school showed that about 65% were at high risk for developing osteoporosis (estrogen deficient or had at least two other risk factors).[68]

See Box 26-1 for a relevant case study.

BIOMECHANICS

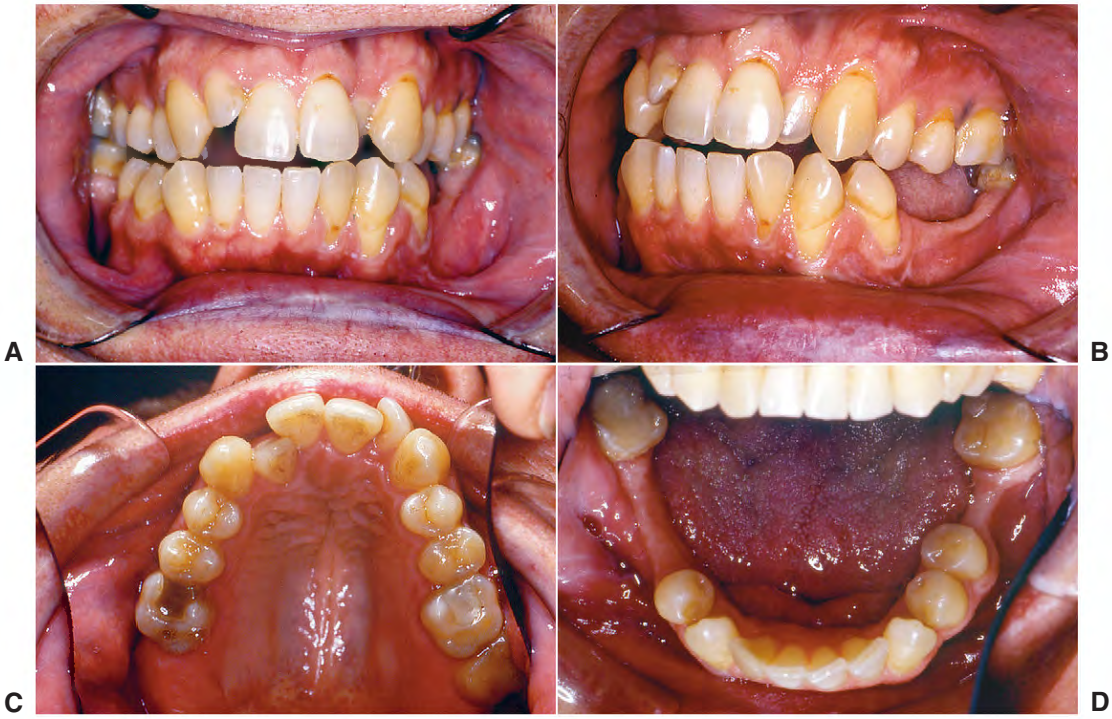
Gravitational loads have a substantial influence on normal skeletal physiology. Osteoblast differentiation that leads to new bone formation is stimulated by mechanical loading[12] but inhibited by weightlessness.[28,69] Space flight studies have established that gravity helps maintain skeletal mass.[70,71] A substantial part of the physiologic loading of the mandible is related to antigravity posturing. In erect posture, gravity tends to open the mouth; muscular force is used to hold the mouth closed. Apparently, growth of the rat mandibular condyle may be inhibited during space flight because of weightlessness and the decrease in functional loading.[72] Gravity may prove to be an important factor in the secondary growth mechanism of the mandible.

Mechanical loading is essential to skeletal health. Control of most bone modeling (see Table 26-1) and some remodeling processes (see Box 6-1) are related to strain history, which usually is defined in microstrain (με).[73] Repetitive loading generates a specific response, which is determined by the peak strain.[74-78] In an attempt to simplify the often conflicting data, Frost[79] proposed the mechanostat theory. Reviewing the theoretical basis of this theory, Martin and Burr[24] proposed that (1) subthreshold loading of less than 200 με results in disuse atrophy, manifested as a decrease in modeling and an increase in remodeling; (2) physiologic loading of about 200 to 2500 με is associated with normal, steady-state activities; (3) loads exceeding the minimal effective strain (about 2500 με) result in a hypertrophic increase in modeling and a concomitant decrease in remodeling; and (4) after peak strains exceed about 4000 με, the

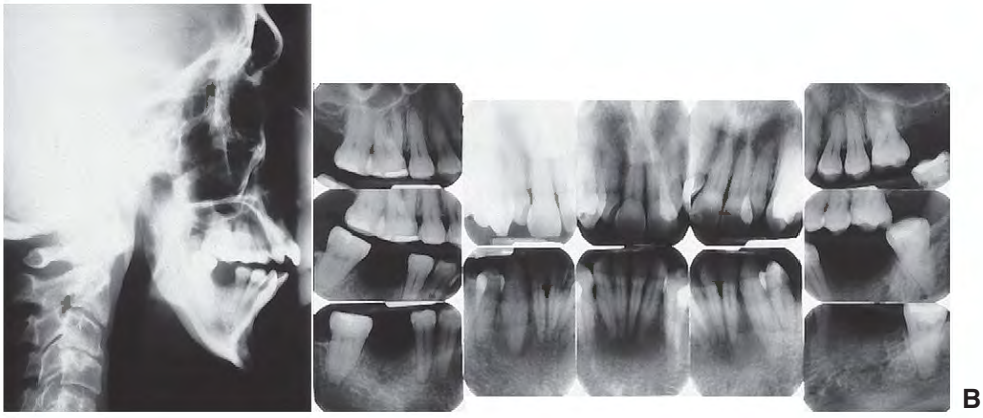
WWW.HIGHDENT.IR

Box 26-2 Temporomandibular Discrepencies: A Case Study

A 52-year-old man sought treatment for a long history of facial pain, occlusal dysfunction, and an internal derangement of the right mandibular condyle (see figures). Intracapsular surgery was performed on the right temporomandibular (TMJ) accelerated, the pain increased, and a progressive anterior open bite malocclusion developed. Masticatory function deteriorated, and an internal derangement of the left TMJ was noted. Bilateral intracapsular surgery was performed to restore "normal jaw function." After the second surgical procedure, the patient suffered for 10 years with chronic pain and progressive bilateral degeneration of both TMJs. Orthodontic and orthotic (splint) therapy failed to relieve the pain and functional debilitation. The patient declined further treatment and is being managed with pain medication. From a physiologic perspective, intracapsular surgery usually is contraindicated because it inhibits the natural ability of the joint to adapt to changing biomechanical demands. The TMJ is remarkably regenerative and adaptive joint if its physiologic limits are respected.



Frontal view **(A),** lateral view **(B),** maxillary occlusal view **(C),** and mandibular occlusal view **(D)** of the dentition of a 52-year-old man with a partly edentulous open bite malocclusion. Note the atrophic extraction sites and gingival rescession. *(From Roberts WE: Adjunctive orthodontic therapy in adults over 50 years of age: clinical management of compensated, partially edentulous malocclusion,* J Indiana Dent Assoc *76:33-41, 1997.)*



A, Cephalometric radiograph of the patient shown above, shows a skeletal open bite with a steep mandibular plane and a relatively short ramus. The thin symphyseal cortex is consistent with a systemic osteopenia. **B,** A full-mouth radiographic survey shows a generalized lack of cortical bone at the alveolar crest and a pattern of indistinct lamina dura and trabeculae. This generalized ground-glass approach of the alveolar bone is consistent with high-turnover metabolic bone disease. *(From Roberts WE: Adjunctive orthodontic therapy in adults over 50 years of age: clinical management of compensated, partially edentulous malocclusion,* J Indiana Dent Assoc *76:33-41, 1997.)*

WWW.HIGHDENT.IR

structural integrity of bone is threatened, resulting in pathologic overload. Figure 26-43 is a representation of the mechanostat. Many of the concepts and microstrain levels are based on experimental data.[24] The strain range for each given response probably varies between species and may be site specific in the same individual.[16,24,74,76,78] However, the mechanostat provides a useful clinical reference for the hierarchy of biomechanical responses to applied loads.

Normal function helps build and maintain bone mass. Suboptimally loaded bones atrophy as a result of increased remodeling frequency and inhibition of osteoblast formation.[80] Under these conditions, trabecular connections are lost and cortices are thinned from the endosteal surface. Eventually the skeleton is weakened until it cannot sustain normal function. Assuming that the negative calcium balance is corrected and adequate bone structure remains, patients with a history of osteoporosis or other metabolic bone disease are viable candidates for reconstructive dental procedures. The crucial factor is the residual bone mass in the area of interest after the disease process has been arrested (Figure 26-44).

When flexure (strain) exceeds the normal physiologic range, bones compensate by adding new mineralized tissue at the periosteal surface. Adding bone is an essential compensating mechanism because of the inverse relationship between load (strain magnitude) and the fatigue resistance of bone.[81] When loads are less than 2000 με, lamellar bone can withstand millions of loading cycles, more than a lifetime of normal function. However, increasing the cyclic load to 5000 με, about 20% of the ultimate strength of cortical bone, can produce fatigue failure in 1000 cycles, which is achieved easily in only a few weeks of normal activity. Repetitive overload at less than one fifth of the ultimate strength of lamellar bone (25,000 με, or 2.5% deformation) can lead to skeletal failure, stress fractures, and shin splints.

From a dental perspective, occlusal prematurities or parafunction may lead to compromise of periodontal bone support. Localized fatigue failure may be a factor in periodontal clefting, alveolar recession, tooth oblation (cervical ditching), or TMJ arthrosis. Guarding against occlusal prematurities and excessive tooth mobility, while achieving an optimal distribution of occlusal loads, are important objectives for orthodontic treatment. The human masticatory apparatus can achieve a biting strength of more than 2200 N, or more than 500 lb of force.[82,83] Because of the high magnitude and frequency of oral loads, functional prematurities during reconstructive treatment could contribute to isolated incidences of alveolar clefting (Figure 26-45, *A*) and root resorption (see Figure 26-45, *B*). Excessive tooth mobility should be monitored carefully. Prevention of

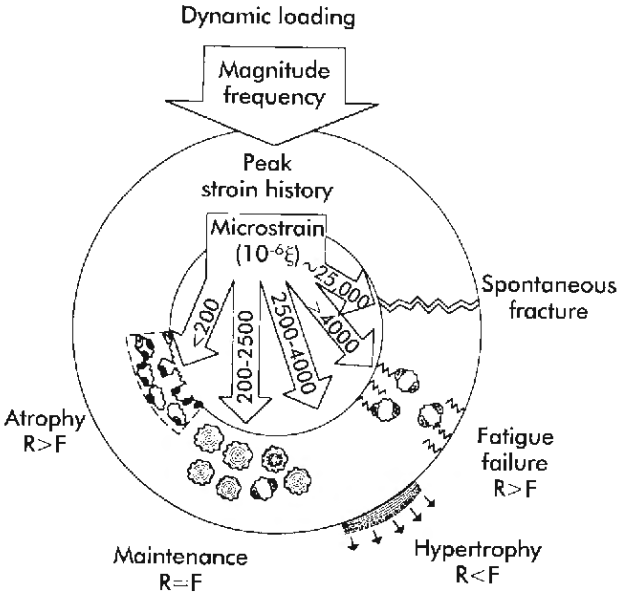


Figure 26-43 The mechanostat concept of Frost as defined by Martin and Burr. Bone formation *(F)* and resorption *(R)* are the modeling phenomena that change the shape and/or form of a bone. The peak strain history determines whether atrophy, maintenance, hypertrophy, or fatigue failure occurs. Note that the normal physiologic range of loading (maintenance $R = F$) is only at less than 10% of maximal bone strength (spontaneous fracture). Fatigue damage can accumulate rapidly at greater than 4000 με.

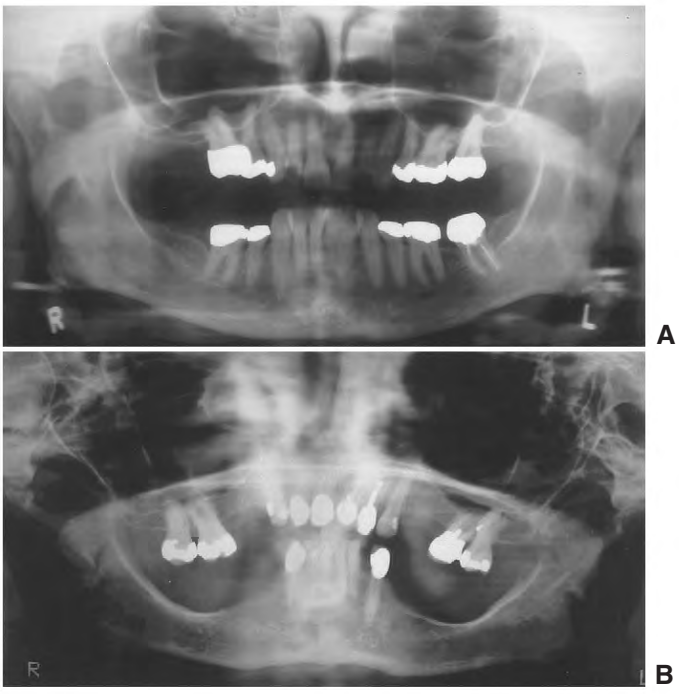


Figure 26-44 Two postmenopausal females with systemic osteopenia present widely varying patterns of lower posterior bone loss. **A,** Alveolar bone in the buccal segments is well preserved by functional loading of natural teeth. **B,** Severe resorption of the alveolar process and basilar mandible has occurred in the absence of adequate functional loading. *(From Roberts WE: Fundamental principles of bone physiology, metabolism and loading. In Naert I, van Steenberghe D, Worthington D et al, editors:* Osseointegration in oral rehabilitation, *London, 1993, Quintessence.)*

WWW.HIGHDENT.IR

occlusal prematurities is a particular concern in treating periodontally compromised teeth.

SUTURES

The facial sutures are important mediators of skeletal adaptation to craniofacial growth and biomechanical therapy. Expansion of the midpalatal suture often is a key objective in dentofacial orthopedic treatment. Although the potential for sutural expansion has been appreciated since the middle of the nineteenth century, Andrew Haas[84] introduced the modern clinical concepts of rapid palatal expansion in the last half of the twentieth century. Despite the long history of this important clinical procedure, little was known of the cell kinetics of osteogenesis and the bone remodeling response associated with it. Sutures and the PDL were widely assumed to have similar mechanisms of osseous adaptation.

Chang et al.[85,86] compared the osteogenic reaction in the expanded midpalatal suture with orthodontically induced osteogenesis in the PDL of adjacent incisors (Figures 26-46 to 26-49). The widened PDL resulted in direct osteogenic induction of new bone, whereas the adjacent expanded suture experienced hemorrhage, necrosis, and a wound healing response. Vascular invasion of the blood clot in the expanded suture was a prerequisite for new bone formation. Chang et al.[85] also defined the angiogenic capillary budding process associated with the propagation of perivascular osteogenic cells (Figure 26-50). After its vascularity had been reestablished, the expanded midpalatal suture and adjacent widened PDL produced new osteoblasts by the same mechanism. Pericytes, the osteogenic cells that are perivascular to the venules (Figure 26-51), are the cells

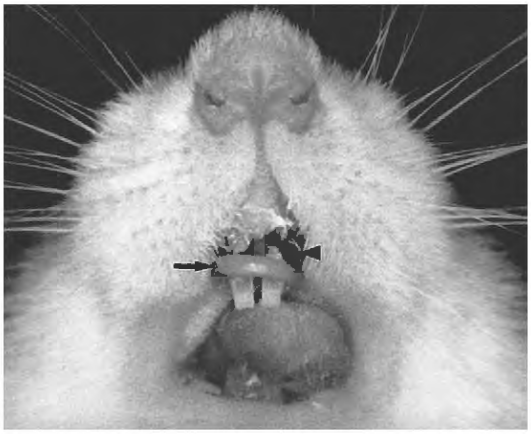


Figure 26-46 An expansion appliance is placed on the maxillary incisors of a rat. A 1-mm-diameter elastomeric ring *(arrowhead)* was fitted into the left incisor; a 2-mm-diameter elastomeric ring *(arrow)* encircled both incisors, 2 mm from cutting edges. The 2-mm ring constricts the incisors while the interproximal elastic elicits a parallel separation of the interpremaxillary suture. *(From Chang HN, Garetto LP, Potter RH et al: Angiogenesis and osteogenesis in an orthopedically expanded suture,* Am J Orthod Dentofacial Orthop *3:382-390, 1997.)*

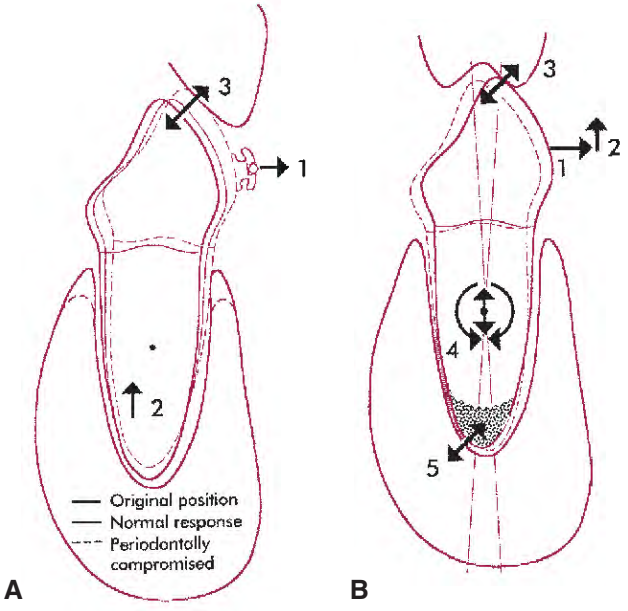


Figure 26-45 **A,** A moderate load in the buccal direction *(1)* results in tipping displacement of the crown. In the absence of vertical constraint, a normal healthy tooth would be expected to extrude slightly because of the inclined plane effect of the root engaging the tapered alveolus *(2)*. As a result of diminished bone support and destruction of restraining collagen fibers at the alveolar crest, a periodontally compromised tooth may tip and extrude considerably more. Depending on the occlusion, this displacement may cause an occlusal prematurity *(3)*. **B,** Orthodontic tipping *(1)* with an extrusive component *(2)* may produce an occlusal prematurity *(3)* and mobility *(4)*. An individual tooth in chronic occlusal trauma is expected to fatigue the root apex continuously. This combination of physical failure in a catabolic environment may lead to progressive root resorption *(5)*.

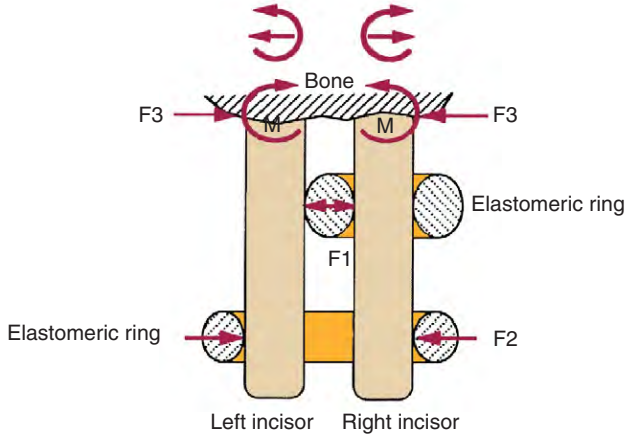


Figure 26-47 Forces *(F)* and moments *(M)* on a tooth. F1 and F2 were produced by inner and outer elastomeric rings, respectively. This illustration of the device demonstrates the formation of a couple thatresulted in parallel separation of the interpremaxillary suture. As measured in a pilot study using a Dontrix tension gauge, the outer elastomeric ring exerted about 200 g of initial separation force (F2), of which 90 g remained at the end of day 3. This force level (90 g) is suitable for premaxillary expansion in rats. *(From Chang HN, Garetto LP, Potter RH et al: Angiogenesis and osteogenesis in an orthopedically expanded suture,* Am J Orthod Dentofacial Orthop *3:382-390, 1997.)*

WWW.HIGHDENT.IR

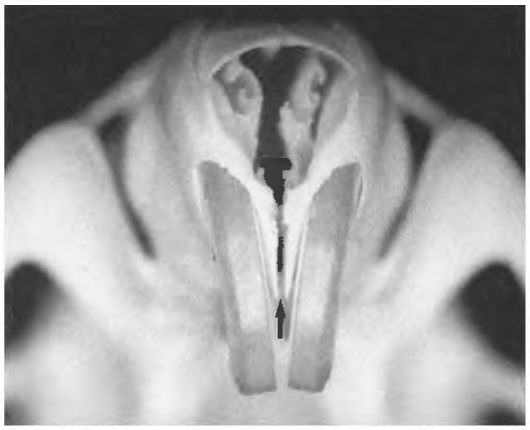


Figure 26-48 A dry skull, expanded as illustrated in Figure 26-55, shows parallel separation of the interpremaxillary suture *(arrow). (From Chang HN, Garetto LP, Potter RH et al: Angiogenesis and osteogenesis in an orthopedically expanded suture,* Am J Orthod Dentofacial Orthop *3:382-390, 1997.)*

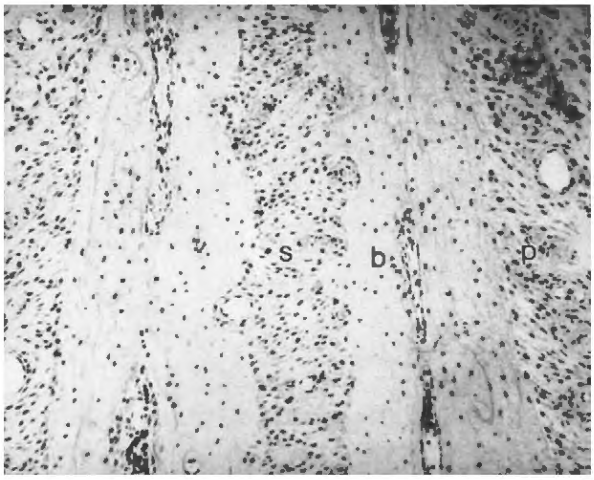


Figure 26-49 Photomicrograph of a sagittal section of the interpremaxillary suture, showing the relationship of expanded suture *(s),* alveolar bone *(b),* and periodontal ligament *(p).* (Stained with hematoxylinand eosin; original magnification ×40.) *(From Chang HN, Garetto LP, Potter RH et al: Angiogenesis and osteogenesis in an orthopedically expanded suture,* Am J Orthod Dentofacial Orthop *3:382-390, 1997.)*

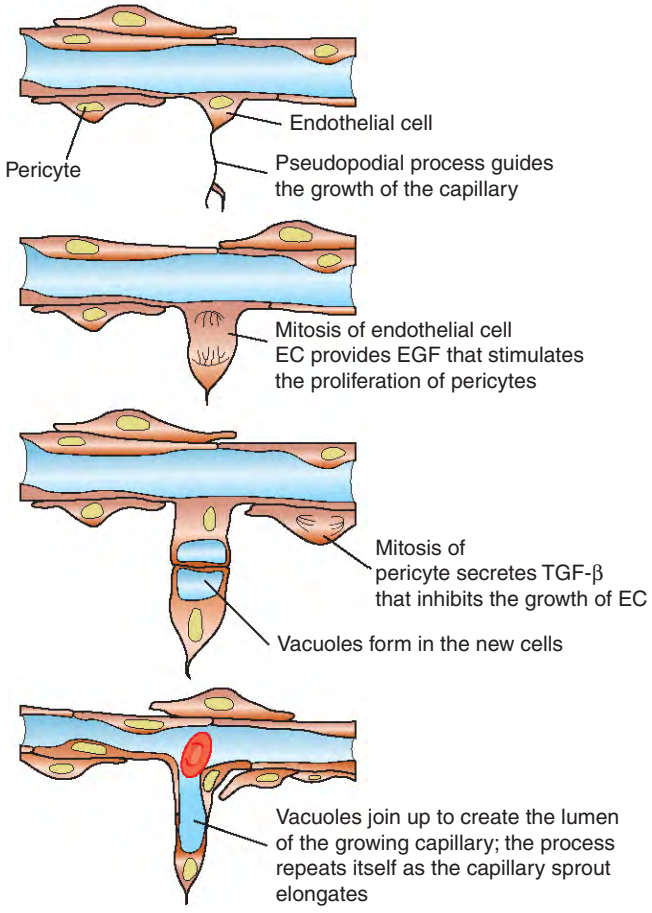


Figure 26-50 Angiogenesis involves a well-defined sequence of capillary budding followed by an extension of the perivascular network of pericytes, which are the source of osteoprogenitor cells. *EC,* Endothelial cell; *EGF,* epidermal growth factor; *TGF-β,* transforming growth factor β. *(Redrawn from Chang HN, Garetto LP, Katona TR et al: Angiogenic induction and cell migration in an orthopaedically expanded maxillary suture in the rat,* Arch Oral Biol *41:985-994, 1996.)*

of origin for preosteoblasts.[85,86] The role of perivascular cells in the origin of PDL osteoblasts first was reported in 1987.[87] Over the past decade a number of investigators have reported the same mechanism for the production of osteoblasts throughout the body. Doherty et al.[88] recently reviewed the literature and provided evidence that vascular pericytes express osteogenic potential in vivo and in vitro. What is now clear is that perivascular osteogenesis is not a mechanism unique to the PDL and sutures, but rather, is the source of osteoblasts all over the body under a variety of osteogenic conditions. Parr et al.[89] used an innovative endosseous implant mechanism (see Figure 26-17) to expand the nasal bones in young adult rabbits with forces from 1 to 3 N. Injection of multiple fluorochrome bone labels documented the bone modeling and remodeling reactions that occurred not only adjacent to the suture but also throughout the nasal bones. Expansion of a suture results in a regional adaptation of adjacent bones similar to the postoperative regional acceleratory phenomenon that is characteristic of bone wound healing.[24] Parr et al.[89] described the bone formation rate and mineral apposition rate for new bone formed in the suture (Figures 26-52 to 26-54). Sutural expansion, relative to load decay, is shown for repeatedly reactivated 1- to 3-N loads (Figure 26-55). Osseointegrated implants were excellent abutments for sutural expansion mediated by loads as large as 3 N.

Overall, expanded sutures are less efficient at initiating osteogenesis because of postactivation necrosis. However, after a wound healing response has occurred to reestablish sutural vitality, the vascularly mediated origin of osteoblasts is the same as for the PDL and other skeletal sites. Expansion of a suture results in a regional

WWW.HIGHDENT.IR

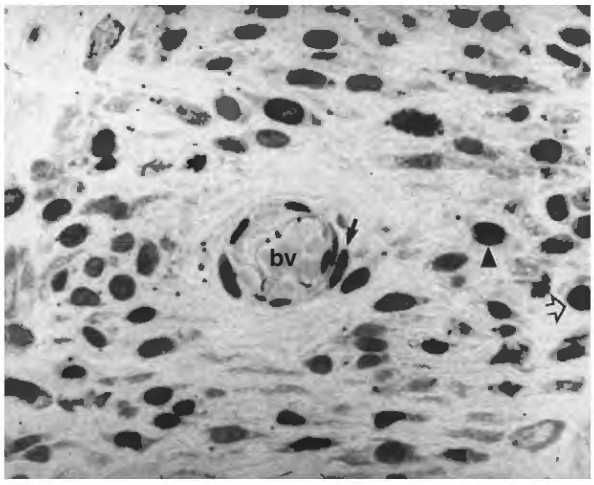


Figure 26-51 Photomicrograph of autoradiography of an expanded interpremaxillary suture, showing blood vessel *(bv)* and paravascular cells. Note the relationship of pericyte *(solid arrow)*, fibroblast-like cells *(arrowhead)*, and mature osteoblast *(open arrow)* lining the suture-bone interface. (Stained with hematoxylin and eosin; original magnification ×400.) *(From Chang HN, Garetto LP, Potter RH et al: Angiogenesis and osteogenesis in an orthopedically expanded suture,* Am J Orthod Dentofacial Orthop *3:382-390, 1997.)*

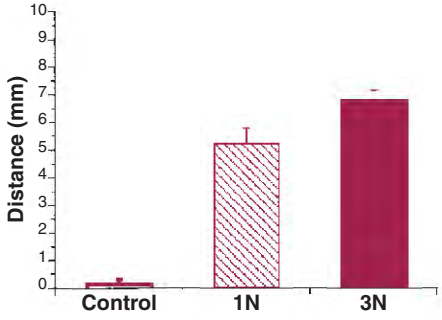


Figure 26-52 Expansion of the suture between the nasal bones of a rabbit (Figure 26-17, *A*) is expressed as the mean difference of initial and final measurements between implants for the three loading groups (mean ± SEM, all groups significant at $p<0.05$). *(From Parr JA Garetto LP, Wohlford ME et al: Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits,* Angle Orthod *67:287, 1987.)*

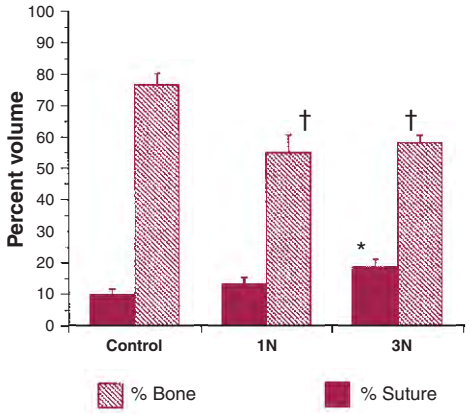


Figure 26-53 Volume percent of suture and bone for three loading groups (mean ± SEM); an asterisk (*) indicates significant difference in the percentage of sutural expansion from the control at $p<0.05$; a dagger (†) indicates significant difference in the percentage of bone from the control at $p<0.05$. *(From Parr JA, Garetto LP, Wohlford ME et al: Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits,* Angle Orthod *67:283-290, 1987.)*

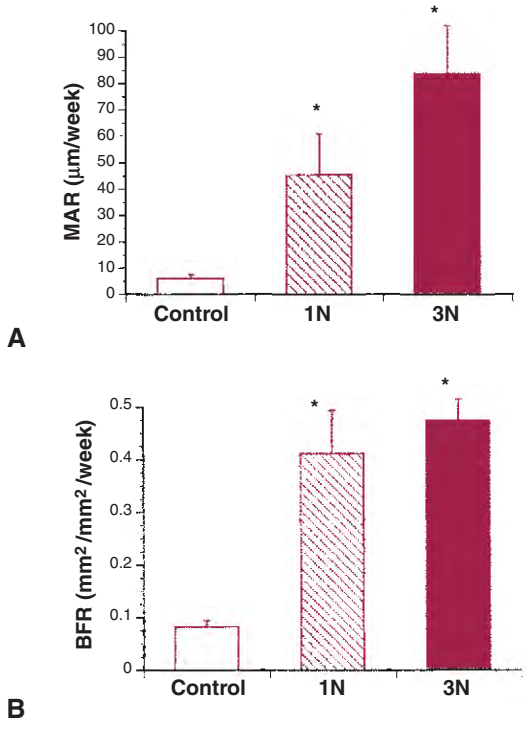


Figure 26-54 **A,** Mineral apposition rate (MAR). **B,** The bone formation rate (BFR) was calculated at the suture during the final 6 weeks of loading for three loading groups (mean ± SEM; an asterisk [*] indicates significant difference from the control at $p<0.05$). *(From Parr JA, Garetto LP, Wohlford ME et al: Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits,* Angle Orthod *67:283-290, 1987.)*

acceleration of bone adaptive activity, which allows for extensive adaptation of the affected bones to new biomechanical conditions. These results indicate that sutural expansion within physiologic limits is a clinically viable means of repositioning the bones of the craniofacial complex to improve esthetics and function. With respect to fundamental bone physiology, sutural expansion is similar to surgically mediated distraction osteogenesis.

Using sequential labels of ^{3}H-thymidine and bromodeoxyuridine in rabbits, Sim[90] demonstrated that the osteoblast histogenesis sequence for evolving secondary osteons was a perivascular process (Figure 26-56) similar to that previously demonstrated for the PDL[91] and the intermaxillary suture.[85,86] The Sim data confirmed the hypothesis that the perivascular connective tissue cells proliferate and migrate along the surface of the invading capillaries or venules. Figure 26-57 is a three-dimensional perspective of a remodeling foci (cutting/filling cone) in cortical bone, which demonstrates that perivascular cells,

WWW.HIGHDENT.IR

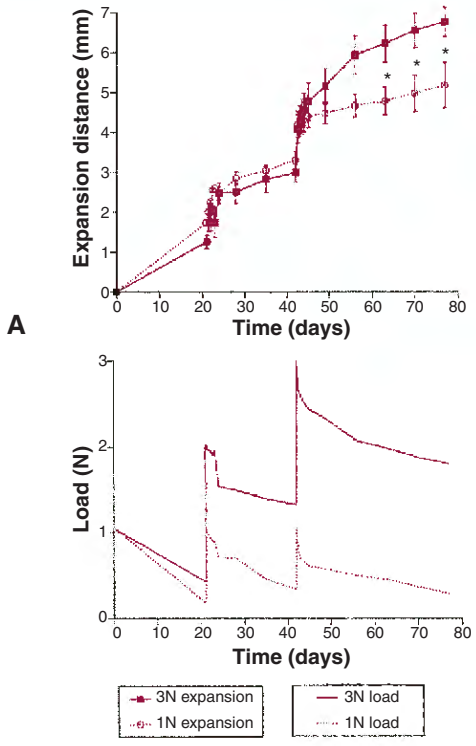


Figure 26-55 **A,** Sutural expansion measured as an increase in the distance between implants. The slope of this curve is the rate of sutural expansion. Three N is significantly greater than 1 N at these time points $p<0.05$. **B,** Load on the suture as a function of time. Load was calculated using the formula $F = kx$, where k is the spring constant and x is the distance between implants. As sutural expansion occurs, force decays. Loads were placed at day 0 and adjusted at days 21 and 42. *(From Parr JA, Garetto LP, Wohlford ME et al: Sutural expansion using rigidly integrated endosseous implants: an experimental study in rabbits,* Angle Orthod *67:283-290, 1987.)*

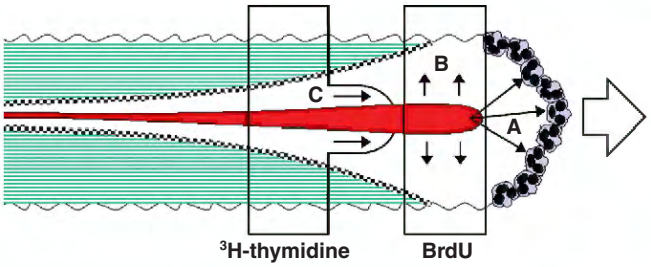


Figure 26-56 A cutting/filling cone in rabbit cortical bone shows the intravascular origin of osteoclasts *(A)*. The perivascular proliferation and migration away for the perivascular surface *(B)* is demonstrated by bromodeoxyuridine (BrdU) labeling and nuclear volume morphometry. A sequence of ^{3}H-thymidine labels from 2 to 72 hours before sacrifice and nuclear morphometric analysis revealed migration of proliferating perivascular cells in the direction of vascular invasion *(C)*. These data demonstrate the perivascular origin of osteoblasts in evolving secondary osteons. *(From Sim Y:* Cell kinetics of osteoblast histogenesis in evolving rabbit secondary haversian systems using a double labeling technique with ^{3}H-thymidine, and bromodeoxyuridine *[doctoral thesis], Indianapolis, 1995, Indiana University School of Dentistry.)*

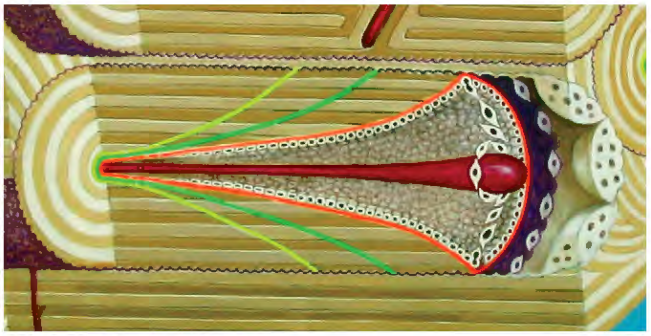


Figure 26-57 An evolving secondary osteon, moving to the right, shows a head of multinucleated osteoclasts *(right)*, followed by a layer of mononuclear cells secreting cement substance *(blue)* to cover the scalloped resorption arrest line. The perivascular osteogenic cells proliferate and differentiate to osteoblasts, which form the new secondary osteon. Three sequential colored bone labels (*yellow, green and orange*) allow the calculation of the velocity of the cutting/filling cone through cortical bone.

near the head of the proliferating blood vessel, are the source of osteoblasts for the filling cone. Confirmation of a perivascular origin of osteoblasts in PDL, sutures, and cortical bone remodeling foci strongly suggests that all osteoblasts, at least in the peripheral skeleton, are derived from perivascular precursors. These data suggest that less differentiated osteogenic cells grow along the surface of bone-related blood vessels (capillaries and venules) as they invade blood clots or other connective tissue spaces in preparation for osteogenesis. From a clinical perspective, the perivascular origin of osteoblasts confirms an important surgical principle: preservation of the blood supply is essential for optimal healing of bone.

IMPLANT-ANCHORED ORTHODONTICS

A major problem in orthodontics and facial orthopedics is anchorage control.[30] Undesirable movement of the anchorage units is a common problem that limits the therapeutic range of biomechanics.[92] An important application of the basic principles of bone physiology is the use of rigid endosseous implants for orthodontic and orthopedic anchorage. Animal studies[25] and clinical trials of custom orthodontic devices[34] have established that rigidly integrated implants do not move in response to conventional orthodontic and orthopedic forces. These devices are opening new horizons in the management of asymmetry, mutilated dentition, severe malocclusion, and craniofacial deformity.[93]

A preclinical study in dogs tested the anchorage potential of two prosthetic-type titanium implants: (1) a prototype of an endosseous device with a cervical post, asymmetric threads, and an acid-etched surface; and (2) a commercially available implant with symmetric threads (Figure 26-58). Based on label incidence (Figure 26-59, *A*) and the relative number of new osteons in microradiographs (see Figure 26-59, *B*), the rate of bone remodeling near the implant was higher

WWW.HIGHDENT.IR

A B

Figure 26-58 **A,** Two titanium implants of different design were placed in the partly edentulous mandible of young adult dogs. **B,** After 2 months of unloaded healing, a 3-N compressive load was applied between the implants for 4 months. Increased periosteal apposition (*) was noted between the implants of some dogs. None of the rigidly integrated fixtures was loosened by the continuous load superimposed on function. *(From Roberts WE, Helm FR, Marshall KJ et al: Rigid endosseous implants for orthodontic and orthopedic anchorage,* Angle Orthod *59:247-256, 1989.)*

compared with the basilar mandible only a few millimeters away.[94] Compared with titanium implants with a smooth surface, the degree of remodeling at the interface is greater for threaded implants placed in a tapped bone preparation.[30] This may be related to the increased resistance of threaded implants to torsional loads over time.[95]

Direct bone apposition at the endosseous interface results in rigid fixation (osseointegration).[96] From an anchorage perspective, a rigid endosseous implant is the functional equivalent of an ankylosed tooth. Complete bony encapsulation is not necessary for an implant to serve as a rigid anchorage unit. The crucial feature is indefinite maintenance of rigidity despite continuous orthodontic loads. Over time, orthodontically loaded implants achieve a greater fraction of direct osseous interface.[34,95] From an orthodontic and orthopedic perspective, titanium implants can resist substantial continuous loads (1 to 3 N superimposed on function) indefinitely. Histologic analysis with multiple fluorochrome labels and microradiography confirm that rigidly integrated implants do not move relative to adjacent bone (see Figure 26-59).[25] By definition, maintaining a fixed relationship with supporting bone is true osseous anchorage. Endosseous (osseointegrated) implants are well suited to many demanding orthodontic applications.[30,34]

Routine use of rigid implants for prosthetic or orthodontic applications requires that fixtures be placed between or near the roots of teeth. Inadvertent impingement on the PDL and the root of an adjacent tooth still may provide an acceptable result (Figure 26-60). Cementum repair occurs where the root is cut, the PDL reorganizes, and the implant surface is integrated rigidly with osseous tissue. No evidence exists of ankylosis of the tooth.[25]

Retromolar Implant Anchorage

The isolated loss of a lower first molar with a retained third molar is a common problem. Rather than extract the third molar and replace the first molar with a three-unit bridge, mesial translation of second and third molars to close the edentulous spaces often is preferable (Figure 26-61). The first case with long-term follow-up has been published.[34] Because of the increasing incidence of progressive bone loss and fatigue fracture associated with single-tooth implants in lower first and second molar areas, the orthodontic option for mesially translating the molars to close the space is increasing in popularity.

External Abutment Mechanism

An anchorage wire that is secured to a retromolar implant can be used to intrude and protract mandibular second and third molars to close an atrophic first molar extraction site (see Figure 26-61, *B*).[16] The tipping and extrusion of residual lower molars limits potential orthodontic repositioning. Rigid retromolar implants offer a unique capability for intrusion and alignment. Figure 26-62 demonstrates the mechanics for achieving three-dimensional control to intrude the third molar to the plane of occlusion and translate both teeth mesially. Cephalometric tracings (Figure 26-63) document the more than 10 mm of mesial translation and its stability. Panoramic radiographs show the initial alignment (Figure 26-64, *A*) and the final space closure (see Figure 26-64, *B*). Clinical details are published.[34]

Histologic analysis of implants recovered after completion of treatment has revealed important information about the continuous remodeling process that maintains the rigid integration and anchorage value of the endosseous device. Two intravital bone labels, administered within 2 weeks of implant recovery, have

WWW.HIGHDENT.IR

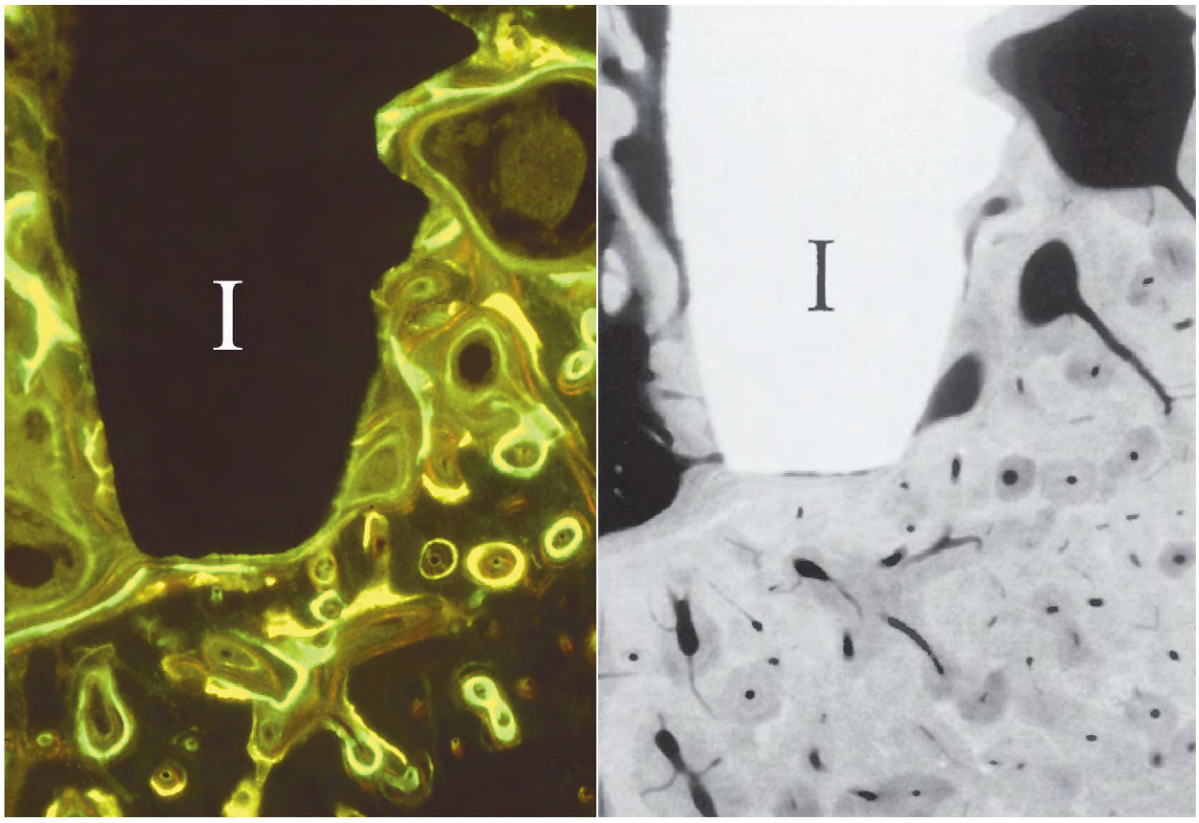


Figure 26-59 **A,** Multiple fluorochrome labels in bone adjacent to an implant *(I)* show a high rate of remodeling at the bone-implant surface. **B,** Microradiographic image of the same section shows direct bone contact on the surface of the implant. *(From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors:* Bone biodynamics in orthodontic and orthopedic treatment, *Ann Arbor, Mich, 1992, University of Michigan Press.)*

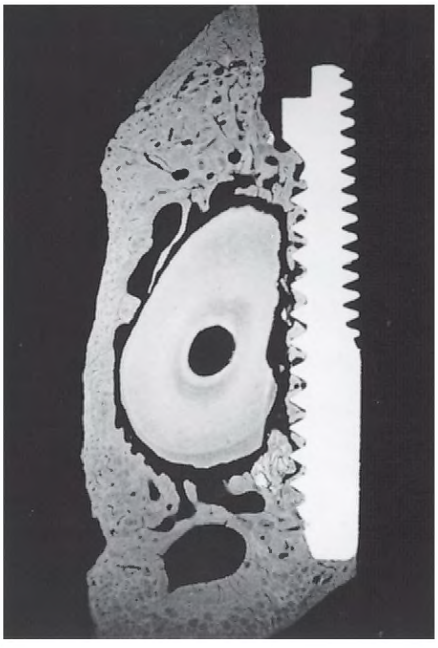


Figure 26-60 An endosseous implant inadvertently impinged on the root of a canine. The implant successfully integrated with bone and served as a rigid anchor for orthopedic loading. *(From Roberts WE et al: Rigid endosseous implants for orthodontic and orthopedic anchorage,* Angle Orthod *59:247, 1989.)*

shown a continuing high rate of bone remodeling (more than 500% per year) within 1 mm of the implant surface (see Figure 26-64, *C, D*). This biological mechanism apparently is the means by which rigid osseous integration is maintained indefinitely.[8,34] If no fracture is present at the implant interface or in its supporting bone, rigid implants are not moved by orthodontic loads.[6,25,34] Well-integrated endosseous implants remain rigid despite continued remodeling of the bone supporting them because only a portion of the osseous resorbed interface is turned over at any given time.[34] Figure 26-65 shows the mechanics for mesial translation of molars to close an edentulous space when a premolar is congenitally missing. Rigid endosseous implants show great promise for considerably extending the therapeutic possibilities of orthodontics and dento-facial orthopedics.

Internal Abutment Mechanism

The 0.019 × 0.025-inch titanium-molybdenum alloy anchorage wire (Ormco Corp., Orange, Calif.) is secured to the endosseous implant when the implant is placed (Figure 26-66). A 7 to 10 × 3.75-mm Brånemark implant (Nobel Biocare, Gothenburg, Sweden) is placed in the

WWW.HIGHDENT.IR

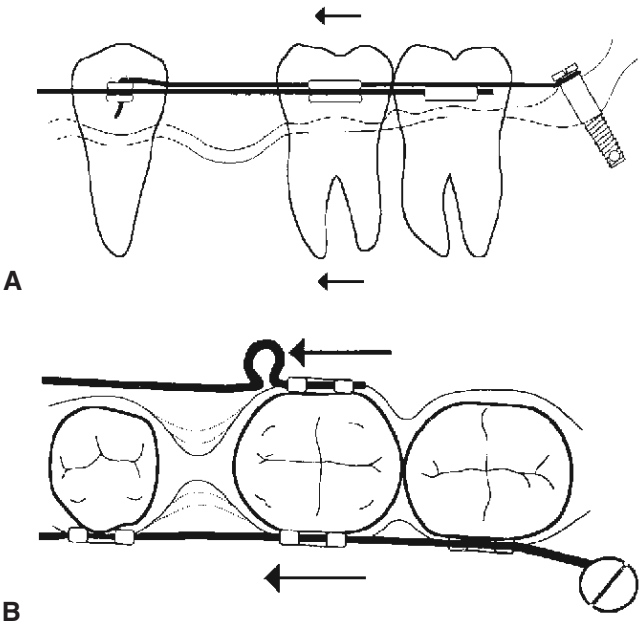


Figure 26-61 **A,** The mechanics of using a retromolar implant with an external abutment as anchorage to stabilize the premolar anterior to an extraction site. **B,** Using buccal and lingual mechanics to balance the load and shield the periosteum in the extraction site, the atrophic extraction site is closed without periodontal compromise of any of the adjacent teeth. *(From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors:* Bone biodynamics in orthodontic and orthopedic treatment, *Ann Arbor, Mich, 1992, University of Michigan Press.)*

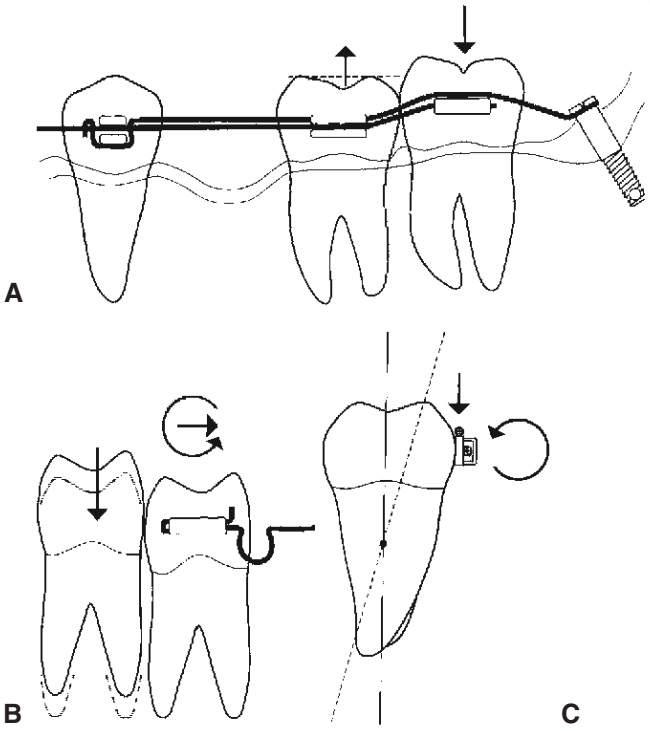


Figure 26-62 **A,** The mechanics of intruding a third molar with implant anchorage before space closure. **B,** A removable lingual arch prevents extrusion of the second molar. **C,** Because the intrusive force on the third molar is buccal to the center of resistance, the tooth tends to tip buccally. This problem is controlled by placing lingual crown torque in the rectangular wire inserted in the tube. *(From Roberts WE, Garotto LP, Katona TR: Principle of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors:* Bone biodynamics in orthodontic and orthopedic treatment, *Ann Arbor, Mich, 1992, University of Michigan Press.)*

Figure 26-63 **A,** Pretreatment, finish, and 3-year postretention cephalometric tracings document 10 to 12 mm of molar translation to close an atrophic first molar extraction site. **B,** Mandibular superimposition shows the mesial movement of the second and third molars, as well as lingual root torque of the lower incisors. *(From Roberts WE, Marshall KJ, Mozsary PG: Rigid endosseous implant utilized as anchorage to protract molars and close an atrophic extraction site,* Angle Orthod *60:135, 1990.)*

WWW.HIGHDENT.IR

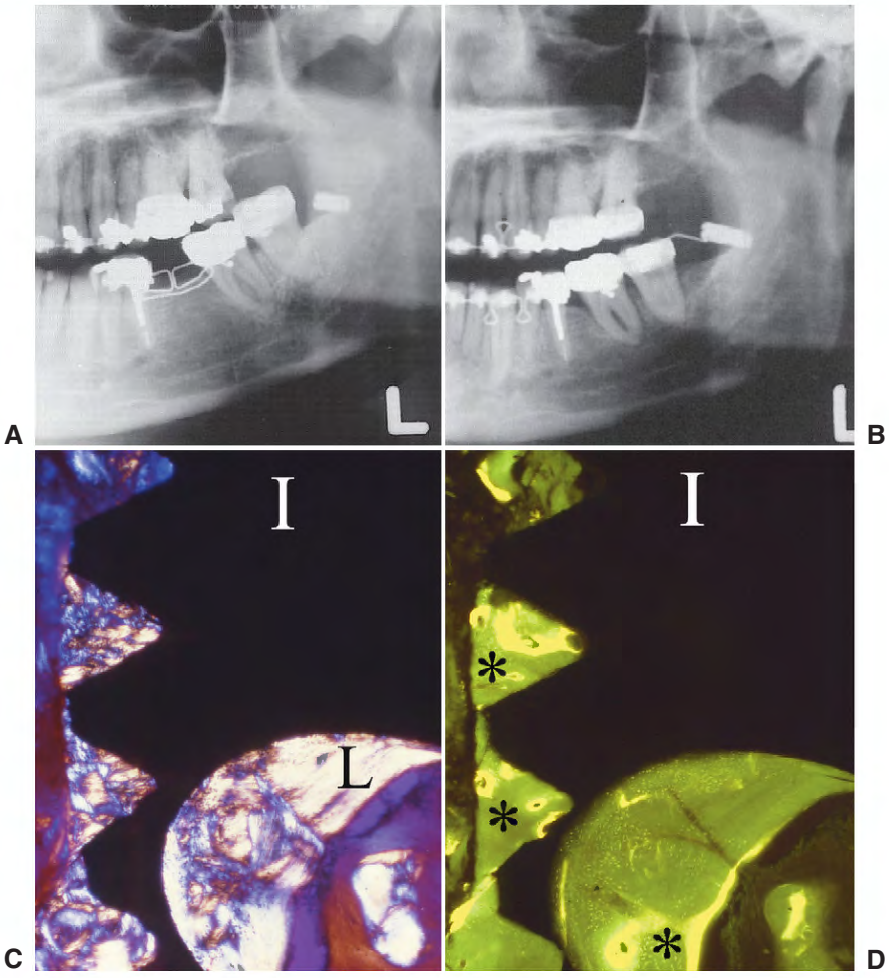


Figure 26-64 **A,** Panoramic radiograph of the initial buccal alignment before an implant is uncovered. **B,** Panoramic radiograph of the closed extraction site. **C,** Polarized light microscopy of lamellar bone *(L)* around the implant *(I)* recovered after completion of treatment. **D,** Two demeclocycline labels (*) in bone adjacent to the implant *(I)* document the high rate of bone remodeling that apparently is the mechanism for long-term maintenance of rigid osseous fixation (osseointegration). *(From Roberts WE, Marshall KJ, Mozsary PG: Rigid endosseous implant utilized as anchorage to protract molars and close an atrophic extraction site,* Angle Orthod *60:135, 1990.)*

Figure 26-65 A 44-year-old female has a partly edentulous mandibular arch and a long history of temporomandibular dysfunction and pain. **A,** A progress radiograph shows restoration of occlusion in the left mandibular buccal segment with implants. **B,** The molars on the right side are being intruded and rotated mesially with the retromolar implant anchorage mechanism. **C,** By the end of active treatment, the mandibular curve of Spee has flattened and ideal alignment of the residual dentition has been achieved. *(From Epker BN, Stella JP, Fish LC:* Dentofacial deformities: integrated orthodontic and surgical correction, *ed 2, vol 4, St Louis, 1999, Mosby.)*

WWW.HIGHDENT.IR

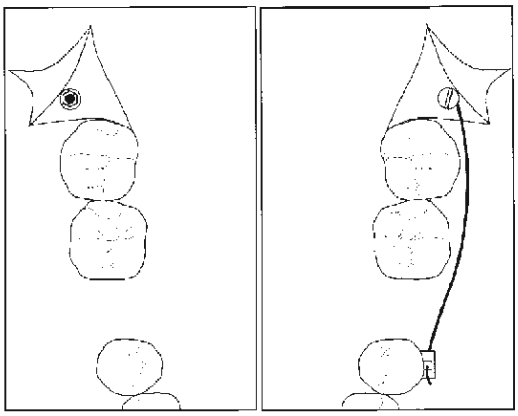


Figure 26-66 The drawing on the left shows the soft tissue flap design for placing a retromolar anchorage implant distal to a mandibular right third molar. The drawing on the right illustrates the attachment of the 0.019 × 0.025-inch titanium-molybdenum alloy wire to the implant with a standard cover screw. Note that the free end of the passive anchorage wire is inserted into the vertical slot of a bracket bonded to the buccal surface of a mandibular left second premolar. *(From Epker BN, Stella JP, Fish LC:* Dentofacial deformities: integrated orthodontic and surgical correction*, ed 2, vol 4, St Louis, 1999, Mosby.)*

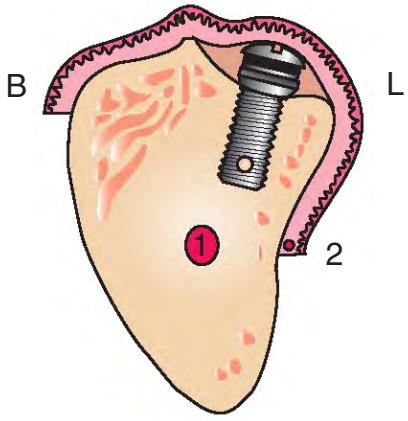


Figure 26-67 A cross section of the mandible distal to the third molar shows that the retromolar implant is inclined buccally *(B)*. Using the shelf of bone on the lingual *(L)*, the 7- to 10-mm implant is oriented toward the inferior alveolar nerve *(1)* and away from the lingual nerve *(2)*. *(Redrawn from Epker BN, Stella JP, Fish LC:* Dentofacial deformities: integrated orthodontic and surgical correction*, ed 2, vol 4, St Louis, 1999, Mosby.)*

retromolar area 3 to 5 mm buccal and distal to the terminal molar. The end of the anchorage wire is bent into a circle and firmly attached to the implant with a standard healing cap (Figures 26-67 and 26-68). This "internal abutment" approach offers a number of advantages over the original external abutment method as follows:

- Minimal surgery: no postoperative uncovering is required.
- Less expense: only one surgical procedure is needed, and no transmucosal abutment is required.
- Better hygiene: wire exiting in the depth of the buccal fold requires little or no periodontalmaintenance.
- Immediate loading: no healing period is necessary.
- More versatile intrusive force: control of the intrusive load on the mandibular molars is easier.

Nineteen years of experience with the internal abutment mechanism (Figure 26-69) has established its utility as an implant anchorage mechanism for managing edentulous spaces in the mandibular buccal segments.[97-100]

Indirect anchorage with a retromolar implant is proving to be useful for closing missing second premolar spaces in growing children. However, an increased tendency for soft tissue irritation exists if the anchorage wire is positioned in the depth of the mucobuccal fold (Figure 26-70, *A*). When the wire is repositioned to just under the brackets of the molars (see Figure 26-70, *B*), soft tissue irritation ceased to be a problem and the second molar space was closed in about 10 months.

Figure 26-68 A schematic drawing of the implant anchorage mechanism shows an internal abutment (i.e., the titanium-molybdenum alloy anchorage wire) attached to the endosseous base with the cover screw (healing cap). The anchorage wire passes through the mucosa in the depth of the buccal fold on the buccal aspect of the terminal molar. Extrusion of the molars during axial alignment and space closure is controlled by the intrusive force *(arrow)* generated by ligating the molar bracket to the anchorage wire with a steel ligature. Sagittal anchorage for mesial movement of the molars is achieved by inserting the passive anchorage wire into the vertical tube of a bracket anterior to the extraction site (*). To achieve unidirectional space closure, the "keyhole" vertical loop is activated by pulling the arch wire at the distal of the terminal molar and bending it down. *(Redrawn from Epker BN, Stella JP, Fish LC:* Dentofacial deformities: integrated orthodontic and surgical correction*, ed 2, vol 4, St Louis, 1999, Mosby.)*

Mini-Implants for Orthodontic Anchorage

Kanomi[101] introduced a series of miniscrews as miniature implants for orthodontic anchorage. Although some of the nonintegrated titanium screws served as adequate anchorage units, some loosened and failed during treatment. A new series of osseointegrated mini-implants was developed and tested in animals.[43] Deguchi et al.[102] found that 97% of 96 implants placed in eight dogs successfully integrated and 100% of the implants that achieved osseointegration were successful

WWW.HIGHDENT.IR

Figure 26-69 **A,** A mock-up demonstrates retromolar implant anchorage. Orthodontic mechanics are designed to align mandibular second and third molars and close the edentulous spaces by translating the molars mesially. The left retromolar implant shows the relationship of the fixture to supporting bone (surgical view). The right implant is covered with wax to simulate the closure of soft tissue over the implant with the titanium-molybdenum alloy anchorage wire attached. **B,** Mechanics are shown for mesial root movement of the second molar into the first molar extraction site. Note that the mesial arm on the root spring is immediately adjacent to the first premolar bracket to prevent the second molar from moving distally as it is positioned upright. To prevent space from opening mesial to the first premolar, a steel ligature ("rope tie") connects the first premolar to the canine. A steel ligature connecting the bracket of the second molar to the titanium-molybdenum alloy anchorage wire controls molar extrusion. **C,** Similar mechanics as shown in **B** are used for simultaneous alignment of both molars. A rectangular arch wire segment connects the two molars. Extrusion is controlled by tying the second molar to the anchorage wire with a steel ligature.

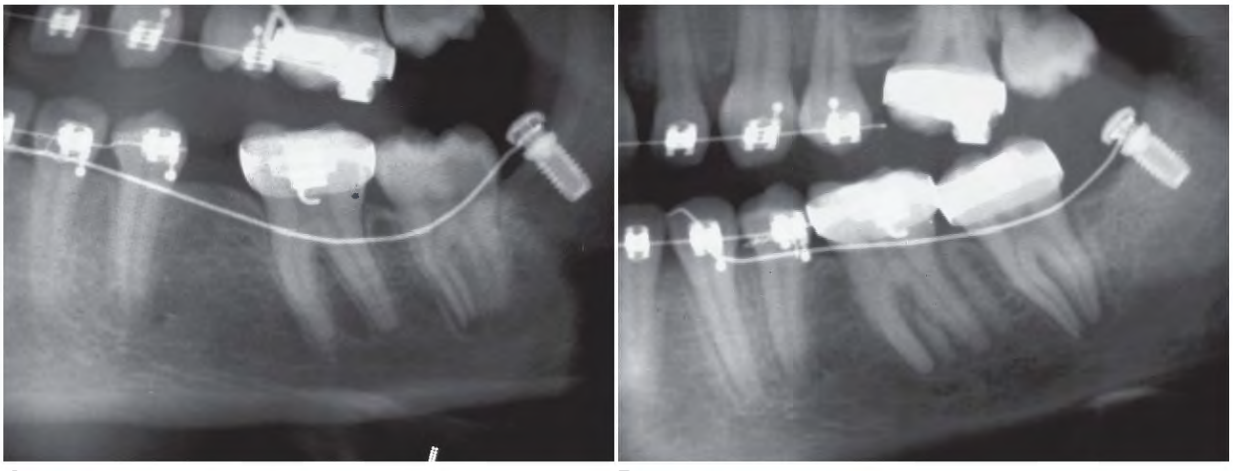


Figure 26-70 **A,** A postoperative panoramic radiograph reveals a retromolar implant to be used for indirect anchorage to close the space caused by a missing second premolar in an 11-year-old girl. Note that the anchorage wire is positioned too far apically. **B,** A panoramic radiograph shows the rapid space closure associated with mesial movement of the molars. Note the anchorage wire has been repositioned just under the molar brackets to lessen soft tissue irritation.

WWW.HIGHDENT.IR

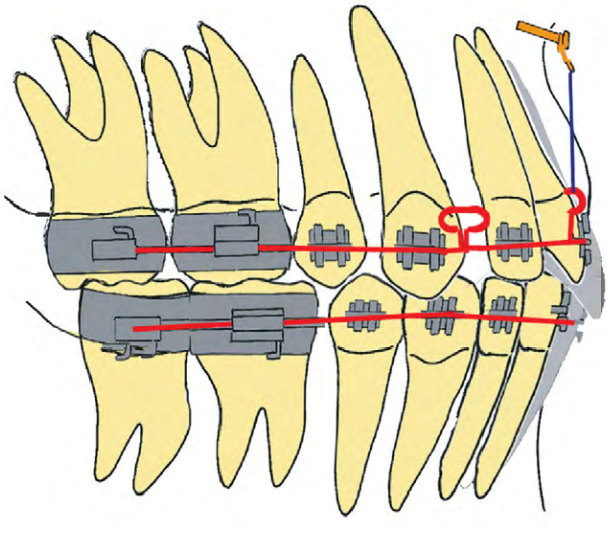
as anchorage units. Clinical use of these simple devices have produced some impressive results. Figure 26-71 documents the treatment of a 15-year-old girl with a gummy smile and bimaxillary protrusion. Four first premolars were extracted and the maxillary anterior segment was intruded with mini-implant anchorage (see Figure 26-71, *A*, *B*). Comparison of frontal smile photographs pretreatment (see Figure 26-71, *C*) and posttreatment (see Figure 26-71, *D*) demonstrate the effective anchorage of the mini-implants. Figure 26-71, *E* is a cephalometric superimposition that demonstrates intrusion of the maxillary anterior segment and a horizontal vector of mandibular growth. Clearly, miniscrews are effective osseous anchorage for some types of malocclusion.

SUMMARY

Bone physiologic, metabolic, and cell kinetic concepts have important clinical applications in all phases of stomatognathic reconstruction: implant surgery, orthodontics, prosthodontics, and long-term functional loading. The application of fundamental concepts is limited only by the knowledge and imagination of the clinician. Modern clinical practice is characterized by a continual evolution of methods based on fundamental and applied research.

ACKNOWLEDGMENTS

This research was supported by National Institute of Dental Research (part of the National Institutes of Health [NIH]) Grants No. DE09237 and DE09822; NASA-Ames grant numbers NCC 2-594 and NAG 2-756; and private donors through Indiana University Foundation. The author gratefully acknowledges the assistance of faculty and staff members at the University of the Pacific School of Dentistry and Indiana University School of Dentistry. Ryuzo Kanomi provided the material on mini-implants for orthodontic anchorage.



A

B

Figure 26-71 **A,** A drawing of space closure mechanics demonstrates the use of a miniimplant apical to the central incisors to intrude the maxillary anterior segment. **B,** An occlusal radiograph shows the two miniscrews apical to the maxillary incisors. *Continued*

WWW.HIGHDENT.IR

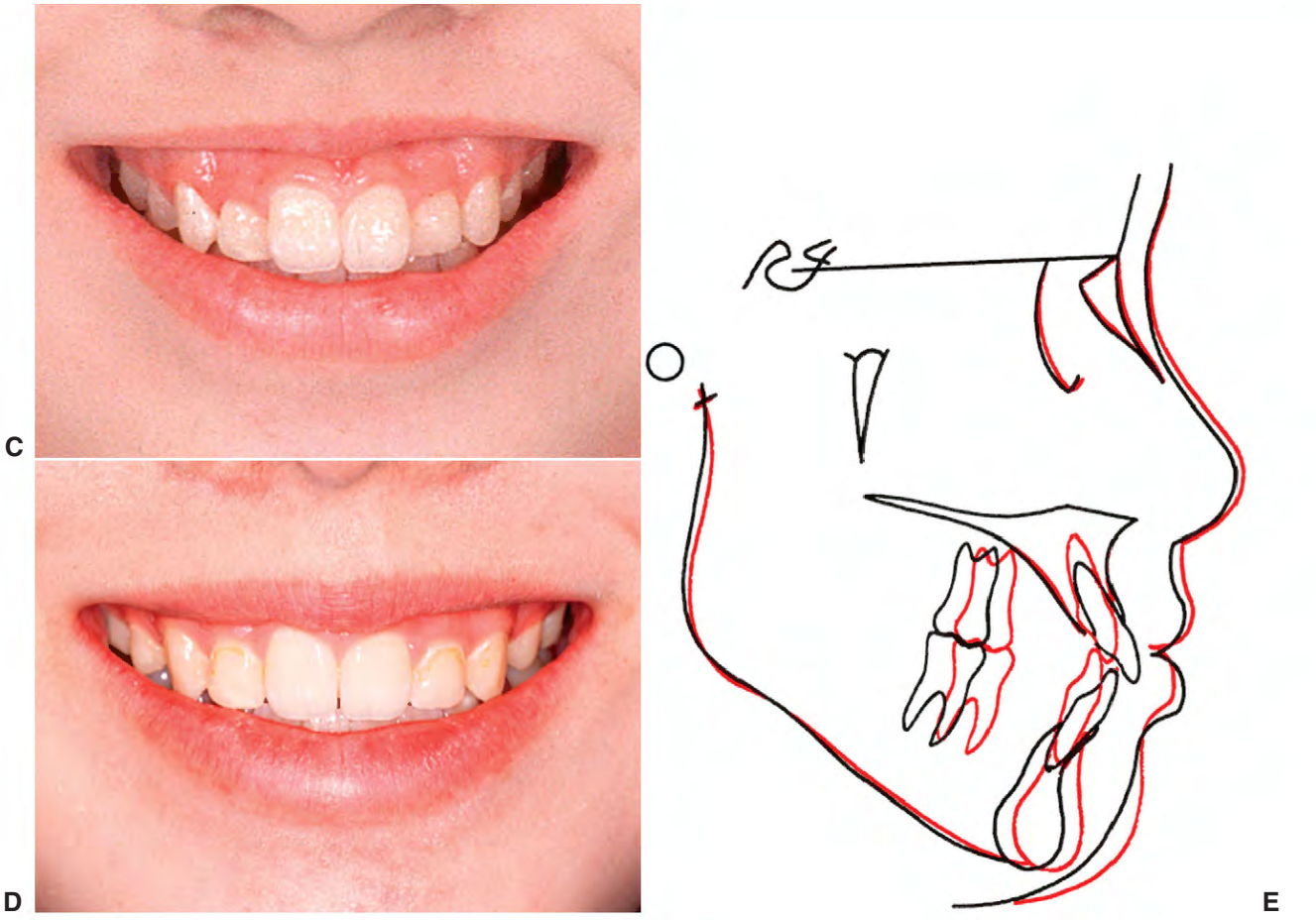


Figure 26-71, cont'd **C,** Pretreatment photograph of a 15-year-old girl reveals a gummy smile. **D,** Posttreatment photograph shows a pleasant smile line with ideal gingival exposure. **E,** Pretreatment *(black)* and posttreatment *(red)* tracings of cephalometricradiographs are superimposed on the anterior cranial base. Note the intrusion of the maxillary anterior incisors and the horizontal component to mandibular growth. *(Courtesy Ryuzo Kanomi.)*

References

1. Atkinson SR: Balance: the magic word, *Am J Orthod* 50:189, 1964.
2. Hylander WL: Mandibular function in *Galago crassicaudatus* and *Macaca fasicularis:* an in vivo approach to stress analysis, *J Morphol* 159:253, 1979.
3. Hylander WL: Patterns of stress and strain in the macaque mandible. In Carlson DS, editor: *Craniofacial biology,* Ann Arbor, Mich, 1981, Center for Human Growth and Development.
4. Behrents RG: Adult facial growth. In Enlow DH, editor: *Facial growth,* ed 3, Philadelphia, 1990, WB Saunders.
5. Pead MJ, Lanyon LE: Indomethacin modulation of load-related stimulation of new bone formation in vivo, *Calcif Tissue Int* 45:34, 1989.
6. Roberts WE, Smith RK, Zilberman Y et al: Osseous adaptation to continuous loading of rigid endosseous implants, *Am J Orthod* 86:95, 1984.
7. Roberts WE, Turley PK, Brezniak N et al: Implants: bone physiology and metabolism, *CDA J* 15:54, 1987.
8. Roberts WE: Bone tissue interface, *J Dent Educ* 52:804, 1988.
9. Roberts WE: Advanced techniques for quantitative bone cell kinetics and cell population dynamics. In Jaworski ZFG, editor: *Proceedings of the first workshop on bone morphometry,* Ottawa, 1976, University of Ottawa Press.
10. Roberts WE, Chase DC, Jee WSS: Counts of labeled mitoses in orthodontically stimulated periodontal ligament in the rat, *Arch Oral Biol* 19:665, 1974.
11. Roberts WE, Jee WSS: Cell kinetics of orthodontically stimulated and nonstimulated periodontal ligament in the rat, *Arch Oral Biol* 19:17, 1974.
12. Roberts WE, Mozsary PG, Klingler E: Nuclear size as a cell kinetic marker for osteoblast differentiation, *Am J Anat* 165:373, 1982.
13. Roberts WE, Ferguson DJ: Cell kinetics of the periodontal aligament. In Norton LA, Burstone CJ, editors: *The biology of tooth movement,* Boca Raton, Fla, 1989, CRC Press.
14. Katona TR et al: Stress analysis of bone modeling response to rat molar orthodontics, *J Biomech* 28:27, 1995.
15. Rapperport DJ, Carter DR, Schurman DJ: Contact finite element stress analysis of porous ingrowth acetabular cup implantation, ingrowth, and loosening, *J Orthop Res* 5:548, 1987.
16. Roberts WE, Garetto LP, Katona TR: Principles of orthodontic biomechanics: metabolic and mechanical control mechanisms. In Carlson DS, Goldstein SA, editors: *Bone biodynamics in orthodontic and orthopedic*

WWW.HIGHDENT.IR

treatment, Ann Arbor, Mich, 1992, University of Michigan Press.
17. Siegele D, Dr-Ing US: Numerical investigations of the influence of implant shape on stress distribution in the jaw bone, *Int J Oral Maxillofac Implants* 4:333, 1989.
18. Roberts WE, Smith RK, Cohen JA: Change in electrical potential within periodontal ligament of a tooth subjected to osteogenic loading. In Dixon A, Sarnat B, editors: *Factors and mechanisms influencing bone growth,* New York, 1982, Alan R Liss.
19. Huja SS, Roberts WE: Mechanism of osseointegration: characterization of supporting bone with indentation testing and backscattered imaging, *Semin Orthod* 10:162-173, 2004.
20. Yip G, Schneider P, Roberts WE: Micro-computed tomography: high resolution imaging of bone and implants in three dimensions, *Semin Orthod* 10:174-187, 2004.
21. Huja SS, Katona TR, Moore BK, et al: Microhardness and anisotropy of the vital osseous interface and endosseous implant supporting bone, *J Orthop Res* 16:54-60, 1998.
22. Roberts WE, Poon LC, Smith RK: Interface histology of rigid endosseous implants, *J Oral Implantol* 12:406, 1986.
23. Ascenzi A, Baschieri P, Benvenuti A: The bending properties of single osteons, *J Biomech* 8:763, 1990.
24. Martin RB, Burr DB: *Structure, function, and adaptation of compact bone,* New York, 1989, Raven Press.
25. Roberts WE et al: Rigid endosseous implants for orthodontic and orthopedic anchorage, *Angle Orthod* 59:247, 1989.
26. Currey JD: *The mechanical adaptations of bones,* Princeton, NJ, 1984, Princeton University Press.
27. Roberts WE: Rigid endosseous anchorage and tricalcium phosphate (TCP)-coated implants, *Calif Dent Assoc J* 12:158, 1984.
28. Roberts WE, Morey ER: Proliferation and differentiation sequence of osteoblast histogenesis under physiological conditions in rat periodontal ligament, *Am J Anat* 174:105, 1985.
29. Keeting PE et al: Lack of a direct effect of estrogen on proliferation and differentiation of normal human osteoblast-like cells, *J Bone Miner Res* 7:S369, 1992.
30. Roberts WE, Garetto LP, Simmons KE: Endosseous implants for rigid orthodontic anchorage. In Bell WH, editor: *Surgical correction of dentofacial deformities,* vol 2, Philadelphia, 1992, WB Saunders.
31. Enlow DH: *Facial growth,* ed 3, Philadelphia, 1990, WB Saunders.
32. Colditz GA, Stampfer MJ, Willett WC et al: Prospective study of estrogen replacement therapy and risk of breast cancer in postmenopausal women, *J Am Dent Assoc* 264:2648, 1990.
33. Roberts WE, Garetto LP, DeCastro RA: Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants with threaded or smooth surfaces: indications for provisional loading and axially directed occlusion, *J Indiana Dent Assoc* 68:19, 1989.
34. Roberts WE, Marshall KJ, Mozary PG: Rigid endosseous implant utilized as anchorage to protract molars and close an atrophic extraction site, *Angle Orthod* 60:135, 1990.
35. Mundy GR, Bonewald LF: Transforming growth factor beta. In Gowen M, editor: *Cytokines and bone metabolism,* Boca Raton, Fla, 1992, CRC Press.
36. Melsen B: The cranial base, *Acta Odontol Scand* 32:1, 1974.
37. Noorda CB: *Modeling and remodeling in the cortical bone of both growing and adult rabbits* [master's thesis], San Francisco, 1986, University of the Pacific.
38. Brockstedt H, Bollerslev J, Melsen F et al: Cortical bone remodeling in autosomal dominant osteopetrosis: a study of two different phenotypes, *Bone* 18:67, 1996.
39. Frost HM: The regional acceleratory phenomenon: a review, *Henry Ford Hosp Med J* 31:3, 1983.
40. Roberts WE, Garetto LP: Physiology of osseous and fibrous integration, *Alpha Omegan* 85:57, 1992.
41. Jeffcoat MK, Williams RC, Kaplan ML et al: Nuclear medicine techniques for the detection of active alveolar bone loss, *Adv Dent Res* 1:80, 1987.
42. Reddy MS, English R, Jeffcoat MK et al: Detection of periodontal disease activity with a scintillation camera, *J Dent Res* 70:50, 1991.
43. Ohmae M, Saito S, Morohashi T et al: A clinical and histological evaluation of titanium mini-implants as anchors for orthodontic intrusion in the beagle dog, *Am J Orthod Dentofacial Orthop* 119:489-497, 2001.
44. Nikolai RJ: *Bioengineering analysis of orthodontic mechanics,* Philadelphia, 1985, Lea & Febiger.
45. Roberts WE, Garetto LP, Arbuckle GR et al: What are the risk factors of osteoporosis? Assessing bone health, *J Am Dent Assoc* 122:59-61, 1991.
46. Roberts WE, Simmons KE, Garetto LP et al: Bone physiology and metabolism in dental implantology: risk factors for osteoporosis and other metabolic bone diseases, *Implant Dent* 1:11, 1992.
47. Midgett RJ, Shaye R, Fruge JF: The effect of altered bone metabolism on orthodontic tooth movement, *Am J Orthod* 80:256, 1981.
48. Rhodes R, Pflanzer R: *Human physiology,* Philadelphia, 1989,WB Saunders.
49. Holtrop ME, Raisz LG, Simmons HA: The effects of parathyroid hormone, colchicine, and calcitonin on the ultrastructure and the activity of osteoclasts in organ culture, *J Cell Biol* 60:346, 1974.
50. Malluche HH, Faugere MC: Renal bone disease 1990: an unmet challenge for the nephrologist, *Kidney Int* 38:193, 1990.
51. Carter DR, Smith DJ, Spengler DM et al: Measurement and analysis of in vivo bone strains on the canine radius and ulna, *J Biomech* 13:27, 1980.
52. Johnston CC Jr, Miller JZ, Slemenda CW et al: Calcium supplementation and increases in bone mineral density in children, *N Engl J Med* 327:82, 1992.
53. Frost HM: *Bone remodeling and its relationship to metabolic bone diseases,* Springfield, Ill, 1973, Charles C Thomas.
54. Heaney RP: Estrogen-calcium interactions in postmenopause: a quantitative description, *Bone Miner* 11:67, 1990.
55. Drinkwater BL, Nilson K, Chesnut CH 3rd et al: Bone mineral content of amenorrheic and eumenorrheic athletes, *N Engl J Med* 311:277, 1984.
56. Rigotti NA, Neer RM, Skates SJ et al: The clinical course of osteoporosis in anorexia nervosa, *JAMA* 265:1133, 1991.
57. Consensus conference report on osteoporosis, *J Am Dent Assoc* 252:799, 1984.
58. Eriksen EF, Mosekilde L: Estrogens and bone, *J Bone Miner Res* 7:273, 1990.
59. Henrich JB: The postmenopausal estrogen/breast cancer controversy, *JAMA* 268:1900, 1992.

WWW.HIGHDENT.IR

60. Love RR, Mazess RB, Barden HS et al: Effects of tamoxifen on bone mineral density in postmenopausal women with breast cancer, *N Engl J Med* 326:852, 1992.
61. Slemenda CW et al: Predicators of bone mass in premenopausal women, *Ann Intern Med* 112:96, 1990.
62. Parfitt AM: The physiological and clinical significance of bone histomorphometric data. In Recker RR, editor: *Bone histomorphometry: techniques and interpretation*, Boca Raton, Fla, 1983, CRC Press.
63. Heaney RP: Calcium, bone health in osteoporosis. In Peck WA, editor: *Bone and mineral research*, Amsterdam, 1986, Elsevier Science.
64. Christiansen C: Consensus development conference: prophylaxis and treatment of osteoporosis, *Am J Med* 90:107, 1991.
65. Grisso JA, Baum CR, Turner BJ: What do physicians in practice do to prevent osteoporosis? *J Bone Miner Res* 5:213, 1990.
66. Roberts WE, Arbuckle GR, Katona TR: Bone physiology of orthodontics: metabolic and mechanical control mechanisms. In Witt E, Tammoscheit U-G, editors: *Symposion der Deutschen Gesellschaft für Kieferorthopadie*, Munich, 1992, Urban & Vogel.
67. Johnston CC: Osteoporosis: extent and cause of the disease, *Proc Soc Exp Biol Med* 191:258, 1989.
68. Becker AR, Handick KE, Roberts WE et al: Osteoporosis risk factors in female dental patients, *J Indiana Dent Assoc* 76:15, 1997.
69. Roberts WE, Mozsary PG, Morey ER: Suppression of osteoblast differentiation during weightlessness, *Physiologist* 24(suppl 6):S75, 1981.
70. Morey ER, Baylink DJ: Inhibition of bone formation during space flight, *Science* 201:1138, 1978.
71. Simmons DJ, Russell JE, Winter F et al: Effect of space flight on the non-weight-bearing bones of rat skeleton, *Am J Physiol* 244:319, 1983.
72. Jackson CB, Roberts WE, Morey ER: Growth alterations of the mandibular condyle in Spacelab-3 rats (abstract), *ASGSB Bull* 1:33, 1988.
73. Cowin SC: *Bone mechanics*, Boca Raton, Fla, 1989, CRC Press.
74. Lanyon LE: Control of bone architecture by functional load bearing, *J Bone Miner Res* 7:S369, 1992.
75. Riggs BL: Overview of osteoporosis, *West J Med* 154:63, 1991.
76. Rubin CT, Lanyon LE: Regulation of bone mass by mechanical strain magnitude, *Calcif Tissue Res* 37:411, 1985.
77. Rubin CT, Lanyon LE: Osteoregulatory nature of mechanical stimuli: function as a determinant for adaptive modeling in bone, *J Orthop Res* 5:300, 1987.
78. Rubin CT, McLeod KJ, Bain SD: Functional strains and cortical bone adaptation: epigenetic assurance of skeletal integrity, *J Biomech* 23:43, 1990.
79. Frost HM: Skeletal structural adaptations to mechanical usage (SATMU). 2. Redefining Wolff's law: the remodeling problem, *Anat Rec* 226:414, 1990.
80. Frost HM: *Intermediary organization of the skeleton*, vol 1, Boca Raton, Fla, 1986, CRC Press.
81. Carter DR: Mechanical loading history and skeletal biology, *J Biomech* 20:1095, 1987.
82. Brunski JB: Forces on dental implants and interfacial stressed transfer. In Laney WR, Tolman DE, editors: *Tissue integration in oral, orthopedic, and maxillofacial reconstruction*, Chicago, 1992, Quintessence.
83. Brunski JB, Shalak R: Biomechanical considerations. In Worthington P, Brånemark PI, editors: *Advanced osseointegration surgery*, Chicago, 1992, Quintessence.
84. Haas AJ: The treatment of maxillary deficiency by opening the midpalatal suture, *Angle Orthod* 35:200-217, 1965.
85. Chang HN, Garetto LP, Katona TR et al: Angiogenic induction and cell migration in an orthopaedically expanded maxillary suture in the rat, *Arch Oral Biol* 41:985-994, 1996.
86. Chang HN, Garetto LP, Potter RH et al: Angiogenesis and osteogenesis in an orthopedically expanded suture, *Am J Orthod Dentofacial Orthop* 111:382-390, 1997.
87. Roberts WE, Wood HB, Chambers DW et al: Vascularly oriented differentiation gradient of osteoblast precursor cells in rat periodontal ligament: implications for osteoblast histogenesis and periodontal bone loss, *J Periodontal Res* 22:461, 1987.
88. Doherty MJ, Ashton BA, Walsh S et al: Vascular pericytes express osteogenic potential in vitro and in vivo, *J Bone Miner Res*13:828, 1998.
89. Parr JA, Garetto LP, Wohlford ME et al: Sutural expansion using rigidly integrated endosseous implants, *Angle Orthod* 67:283, 1997.
90. Sim Y: *Cell kinetics of osteoblast histogenesis in evolving rabbit secondary haversian systems using a double labeling technique with ^{3}H-thymidine and bromodeoxyuridine* [doctoral thesis], Indianapolis, 1995, Indiana University School of Dentistry.
91. Roberts WE, Wood HB, Chambers DW et al: Vascularly oriented differentiation gradient of osteoblast precursor cells in rat periodontal ligament: implications for osteoblast histogenesis and periodontal bone loss, *J Periodontal Res* 22:461-467, 1987.
92. Arbuckle GR, Nelson CL, Roberts WE: Osseointegrated implants and orthodontics, *Oral Maxillofac Surg Clin North Am* 3:903, 1991.
93. Goodacre CJ, Brown DT, Roberts WE et al: Prosthodontic considerations when using implants for orthodontic anchorage, *J Prosthet Dent* 77:162, 1997.
94. Helm FR et al: Bone remodeling response to loading of rigid endosseous implants [abstract], *J Dent Res* 66:186, 1987.
95. Albrektsson T, Jacobsson M: Bone-metal interface in osseointegration, *J Prosthet Dent* 60:75, 1987.
96. Brånemark P-I: Osseointegration and its experimental background, *J Prosthet Dent* 50:399, 1983.
97. Roberts WE: Dental implant anchorage for cost-effective management of dental and skeletal malocclusion. In Epker BN, Stella JP, Fish LC, editors: *Dentofacial deformities*, vol 4, ed 2, St Louis, 1999, Mosby.
98. Roberts WE, Arbuckle GR, Analoui M: Rate of mesial translation of mandibular molars utilizing implant-anchored mechanics, *Angle Orthod* 66:331, 1996.
99. Roberts WE, Hartsfield JK Jr: Multidisciplinary management of congenital and acquired compensated malocclusions: diagnosis, etiology and treatment planning, *Indiana Dent Assoc J* 76:42, 1997.
100. Roberts WE, Nelson CL, Goodacre CJ: Rigid implant anchorage to close a mandibular first molar extraction site, *J Clin Orthod* 28:693, 1994.
101. Kanomi R: Mini-implant for orthodontic anchorage, *J Clin Orthod* 31:763-767, 1997.
102. Deguchi T, Takano-Yamamoto T, Kanomi R et al: The use of small titanium screws for orthodontic anchorage, *J Dent Res* 82:377-381, 2003.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 27

Dental Implant Surfaces: A Review

Adriano Piattelli, Carl E. Misch, Ana Emília Farias Pontes,
Giovanna Iezzi, Antonio Scarano, Marco Degidi

The implant design and surface condition influences the dynamics of osteointegration. The following four distinct phases occur in the development of the bone-implant interface:

1. Surgical integration
2. Healing dynamics
3. Early loading period
4. Mature loading period

The overall implant design and the surface condition affect these four processes, often as independent features (Table 27-1). The surgical process of implant dentistry requires initial fixation and lack of relative movement during the initial phases of the development of the implant-bone interface. The implant design is of primary importance to accomplish this initial step; however, the surface condition of the implant may also be a contributing factor. For example, rougher surfaces will aid the implant to have more friction and fixation during insertion of the implant. When the overall design of the implant is a cylinder and/or the bone quality is poor, the surface roughness of the implant will improve the surgical fixation of the implant.

The initial healing period of an implant (which is not immediately loaded) is the phase of the osteointegration process that is primarily affected by the surface condition of the implant. As a general rule, roughened surfaces increase the bone-implant contact (BIC) percent during the initial bone-healing process. As a consequence, this chapter mainly addresses the contribution of the surface condition of an implant to the healing phase of integration.

Steigenga et al.[1] compared three thread shapes with endosteal implants of similar width, length, thread number, thread depth, and surface condition. The V-shaped and reversed buttress thread had similar BIC and reverse torque values. The square threaded implants had higher BIC and higher reverse torque values. Therefore thread shape may also influence the implant-bone interface during the healing period of an implant, but it is a secondary factor.

The surface condition alone may also decrease the biological width, which causes marginal bone loss when the implant extends through the mucosal tissues. Hermann et al.[2] inserted a combination of smooth- and rough-surface implants with smooth collars of 1.5 mm below the bone and other implants with the rough surface placed at the bone crest. Within 1 month the implants with smooth collars lost 1.5 mm of bone, even though the implants received no occlusal load. The implants with a roughened surface to the crest of bone and no occlusal load maintained the bone for the 6 months of the study. Therefore roughened surfaces improve the initial BIC during initial healing and decrease the marginal bone loss when the implant extends through the soft tissue, before occlusal load.

The early loading period of an implant has considerations from an implant body design and the surface condition of the implant, and both criteria are similar in importance. Stress equals force divided by the area over which it is applied and is directly related to the strain observed in the bone. Therefore BIC will directly relate to the amount of strain at the bone-implant interface. When the strain conditions are within the physiologic zone of bone, the bone-implant interface may maintain a lamellar bone organization, which is organized and mineralized and is best to resist occlusal loads to the interface. The roughened surface of an implant that improves unloaded healing may also help bear the initial occlusal load to an implant interface. For example, hydroxyapatite (HA)-coated cylinder implants reported a high BIC after healing. This implant design also reported high survival rates during the 3 initial years of loading.

The implant design also may affect the early loading period of an implant. For example, smooth-surface cylinder implants do not respond favorably to occlusal load. On the other hand, smooth-surface threaded implants have early loading success, especially in good bone types.

The mature loading period of an implant begins to occur after 3 to 5 years and continues throughout the

WWW.HIGHDENT.IR

Table 27-1 Studies That Evaluated the Role of Implant Design and Surface Condition on the Bone-Implant Interface

PHASES	IMPLANT SURFACE VERSUS DESIGN	AUTHORS	FINDINGS	EXPERIMENTAL DESIGN
1. Surgical integration	Surface condition	Shalabi et al.*	Removal torque values higher for roughed surface (76.6 N-cm) versus machined surface (50.2 N-cm)	Implants with etched or machined surfaces were evaluated after surgical insertion into the femoral condyle of goats
	Implant design	O'Sullivan et al.†	In tibia, resonance frequency values at placement were higher for 1-degree tapered implants (8131 Hz) compared with control (7782 Hz)	Experimental implants (1- or 2-degree taped) were compared with standard Brånemark implants, which were used as control; implants were evaluated after surgical insertion into the tibiae and femurs of rabbits
2. Healing dynamics	Thread shape	Steigenga et al.‡	Square-threaded implants had higher BIC value (74.37%) compared with V-shape (65.46%) and reverse buttress–threaded (63.05%) ones	Implants were placed in the tibiae of rabbits and evaluated after a 12-week healing period
	Surface condition	Hermann et al.§	No significant changes concerning bone loss were detected around implants with the rough surface placed at the bone crest; however, implant inserted with the smooth collars of 1.5 mm below the crest resulted in bone loss (1.7 mm in the first month) to the level of the rough/smooth implant interface	Implants were placed in the mandibles of dogs and evaluated at a monthly interval until sacrifice at 6 months; six combinations of smooth- and rough-surface implants were used in the study; however, data from only two groups are presented
3. Early loading period	Surface condition	Degidi et al.‖	Immediately loaded implants, despite the surface condition, presented high BIC values (average 80.6%)	Immediately loaded implants (two HA-coated and one with a sandblasted surface) were retrieved from two patients and evaluated after a 6-month loading period
	Implant design	Degidi et al.¶	Vertical bone loss was more pronounced for cylindric implants; overall BIC approximately 60% to 65%	Eleven implants submitted to immediate loading were retrieved from patients after a loading period between 10 and 11 months
4. Mature loading period	Implant design	Jeffcoat et al.#	HA cylindric implants had the highest success rate 5 years after restoration (99.0%), followed by HA threaded implants (97.9%)	Implants (threaded and cylindrical HA-coated implants, as well as threaded titanium implants) were inserted between the mental foramina of 120 edentulous patients; prostheses were attached no sooner than 3 months after implant placement; patients were observed for 60 months

BIC, Bone-implant contact; *HA,* hydroxyapatite.

*Shalabi MM, Wolke JG, Jansen JA et al: The effects of implant surface roughness and surgical technique on implant fixation in an in vitro model, *Clin Oral Implants Res* 17:172-178, 2006.

†O'Sullivan D, Sennerby L, Meredith N: Influence of implant taper on the primary and secondary stability of osseointegrated titanium implants, *Clin Oral Implants Res* 15: 474-480, 2004.

‡Steigenga J, Al-Shammari K, Misch C et al: Effects of implant thread geometry on percentage of osseointegration and resistance to reverse torque in the tibia of rabbits, *J Periodontol* 75:1233-1241, 2004.

§Hermann JS, Cochran DL, Nummikoski PV et al: Crestal bone changes around titanium implants: a radiographic evaluation of unloaded nonsurbmerged and submerged implants in the canine mandible, *J Periodontol* 68:1117-1130, 1997.

‖Degidi M, Scarano A, Iezzi G et al: Periimplant bone in immediately loaded titanium implants: histologic and histomorphometric evaluation in human: a report of two cases, *Clin Implant Dent Relat Res* 5:170-175, 2003.

¶Degidi M, Scarano A, Petrone G et al: Histologic analysis of clinically retrieved immediately loaded titanium implants: a report of 11 cases, *Clin Implant Dent Relat Res* 5:89-94, 2003.

#Jeffcoat MK, McGlumphy EA, Reddy MS et al: A comparison of hydroxyapatite (HA)-coated threaded, HA-coated cylindric, and titanium threaded endosseous dental implants, *Int J Oral Maxillofac Implants* 18:406-410, 2003.

WWW.HIGHDENT.IR

life span of the implant interface. In general, the surface condition of the implant is least important during this time frame. For example, roughened surfaces on cylinders often lose bone after 5 years of occlusal load. The roughened surface on a cylinder can withstand the initial loads, but fatigue factors of the bone interface begin to break down over continued cycles, similar to shin splints in long-distance runners. The higher the bone turnover rates are because of high strain conditions, the more likely the bone microstrain condition is found to be in the pathologic overload range—and the bone is lost. On the other hand, the implant design is the major feature of an implant body during the mature loading period and is primarily responsible for the bone turnover rates adjacent to the implant. Therefore the implant design becomes most important in the mature loading period.

Before the advent of osteointegration, the use of implant-supported prostheses often resulted in the formation of a fibrous structure around the implants, resulting in mobility and subsequent loss of the implant. The observation that bone can grow over titanium structures started a new pathway in the study of the intraosseous anchorage for dental prostheses.[3] As a result, long-term implant success rates are reported to be as high as 90% to 100%.[4]

Once an implant is inserted in a bone site, a cascade of biological events is initiated. Initially, osteoconduction occurs, which implies the recruitment and migration of osteogenic cells to the implant surface. Secondly, new bone formation takes place, which results in the formation of a mineralized interfacial matrix, followed by a bone-remodeling process. This phenomenon may influenced by the microtopography of the surface.[5]

However, it is important to emphasize that the surface condition of an implant is not the only requirement to ensure long-lasting implant anchorage. The implant material, status of the bone, surgical technique, surface quality, implant design, and implant-loading conditions all relate to the long-term success of a direct bone-implant interface.[6]

The percentage of BIC may be used to evaluate the stability of an implant, and values higher than 50% appear to be satisfactory for stable prosthetic results.[7] Moreover, torque removal force has been used to describe the anchorage of an implant, in such a way that the higher the value, the greater the biomechanical strength of the bone-implant interface.[8] These values are influenced by factors such as the healing period, the loading protocol, and the experimental methodology of the study. It also appears that results obtained in studies performed in humans are more reliable than the findings obtained in studies performed in animals or in vitro. However, it is more prudent to perform some analyses using an animal model (e.g., histologic or torque removal evaluations of successful implants), and in vitro studies are also important to clarify the biological response of cells in contact with different surfaces.

Scientific studies frequently compare the values of one-dimensional parameters to describe the roughness of a surface, based on the evaluation of its peaks and valleys. For example, it is common to use only parameters representative of the height: the arithmetic mean height deviation from a mean plane (also called R_a, if accessed with bidimensional evaluation, or S_a, if accessed with three-dimensional evaluation).[9] Each surface should be described by the combination of parameters representative of height and space. To simplify the description and comparison of surfaces found on an implant body, both the values of R_a and S_a are addressed in this chapter.

TITANIUM

The "osseointegration" phenomenon was firstly described by Brånemark et al.,[3] and is defined as the "direct contact between living bone and a functionally loaded implant surface without interposed soft tissue at the light microscope level."[10]

Titanium is a metal that presents low weight, high strength/weight ratio, low modulus of elasticity, excellent corrosion resistance, excellent biocompatibility, and easy shaping and finishing.[11] Because of these properties, it is the material most widely used in the manufacture of dental implants, in the commercially pure titanium (cpTi) form or as an alloy. The most frequently used alloy (titanium-6 aluminum-4 vanadium [Ti6Al4V]) is composed of 90% titanium, 6% aluminum (decreases the specific weight and improves the elastic modulus), and 4% vanadium (decreases thermal conductivity and increases the hardness).[12]

Lincks et al.[13] observed in cell cultures that the osteoblast-like cells responded in a differential manner on plates of cpTi and Ti6Al4V surfaces and speculated that the mosaicism of the alloy components would result in a more complex surface chemistry. Moreover, the authors theorized that ions released from these surfaces would have a negative effect on cell adhesion and could impair normal bone formation.[14,15] In addition, they speculated cpTi surfaces were more likely to optimize the osteoblastic differentiation[16] and to attach more strongly to bone than to the alloy.[17] A study was performed inserting implants in the tibiae of rabbits; results were observed at 1, 6, and 12 months. The removal torque of unloaded implants was not statistically different at 1 month, but at 6 months (29 N-cm and 23 N-cm, respectively, in cpTi and alloy) and 12 months (38 N-cm and 35 N-cm, respectively, in cpTi and alloy) the cpTi implants were significantly more stable. BIC values were not statistically different between

WWW.HIGHDENT.IR

the materials, but the authors emphasized a tendency of cpTi implants to have higher values as compared with the alloys implants.[18]

However, not all studies suggest cpTi improved the BIC. In a report on baboons, cpTi and Ti6Al4V dental implants were inserted in the mandible and maxilla of baboons and analyzed 3 and 6 months after implant placement. BIC values significantly increased over time in cpTi (39.1% to 56.2%) and Ti6Al4V sites (40.0% to 55.2%), but differences at 6 months were not considered statistically significant.[19] The primary use of Ti6Al4V in the industry relate to the mechanical advantages over cpTI. The alloy is as much as four times stronger than grade 1 cpTi and 2.4 times stronger than grade 3 titanium. Because the BIC and reverse torque valves are similar before loading, the alloy is often used to decrease the complications of component and/or implant body fracture or wearing of antirotational features of the abutment, which increase the risk of screw loosening.

An interesting aspect of titanium implants (cpTi or Ti6Al4V) is that immediately after its exposure to the air, an oxide layer is formed over the surface (about 2 to 5 nm thickness). This layer is the material that is in contact with the body tissues,[20,21] playing an important role in corrosion resistance, biocompatibility, and osseointegration; however, the mechanisms are not completely understood.[22-24] This most likely is the reason cpTi and Ti6Al4V have similar BIC values. The titanium oxide layer is mainly composed of titanium dioxide (TiO_2),[25] and the crystalline structure—the thickness and stability of this layer—varies according to the surfaces of the implant.[26-28]

TURNED SURFACES

The turned surfaces were the most commonly used surfaces in the past; they were submitted only to a decontamination process after the turning process. These surfaces are also called *machined* or *smooth*, but microscopic observation reveals the presence of a slight roughness because of the grooves and ridges produced during the turning process. For this reason, the use of the term *smooth* should be avoided.[12] One of the main characteristics of the turned surfaces is that it is possible to observe a distance osteogenesis (Figure 27-1).

Modifications were proposed to change the characteristics of the surface from turned to roughened, to improve the stabilization of the implant, and to increase the surface area.[18,29,30] Moreover, it was observed that the morphology of the surface plays a role in the cellular behavior.[31,32] The interaction of the surface with the culturing media and serum seems to directly affect the attachment of osteoblasts and their subsequent proliferation and differentiation,[33,34] as well as to influence the production of local cell regulators such as transforming growth factor β (TGF-β) and prostaglandin E_2 (PGE_2).[35,36,37,38]

On the preparation of modified surfaces, additive methods (e.g., plasma spraying, HA coating) or subtractive methods (e.g., sandblasting, acid etching) have been used; the optimal type of surface is still to be defined. In one study the ideal roughness for a turned surface, which ideally would result in a stronger bone response, was suggested to be 0.9 to 1.3 µm in average.[39] However, another study showed that in the 4.0-µm range, osteoblastic proliferation was reduced but not blocked, phenotypic differentiation was enhanced, and cells on smoother surfaces presented a loss of a differentiated osteoblastic phenotype.[13] In fact, the roughness average values seem to be useful only as a reference, and other factors, such as wettability and free energy,[38] have been studied to clarify their influences on bone formation, together with the average roughness.

SANDBLASTED SURFACES

Sandblasting the metal core with gritting agents creates these modified surfaces. This process is influenced by the number and the speed of the rotations to which the implant is submitted, as well as by the pressure and the size of the particles used.[12,40] The blasting procedure is performed with the aim of increasing the irregularity of the surface of the implant, using agents such as aluminum oxide (Al_2O_3, also called *alumina*) (Figures 27-2 and 27-3) and TiO_2. The analyses of different implant surfaces revealed that sandblasted samples showed the largest variability in surface appearance. Researchers have suggested that this characteristic may be explained by an inappropriate refreshing cycle of the used particles because they can break after use; these fragments may be reused, instead of being discarded after a single run.[41] In Table 27-2, comparative studies in which sandblasted implants were evaluated are presented.

Sandblasting has been shown in some studies to allow the adhesion, proliferation, and differentiation

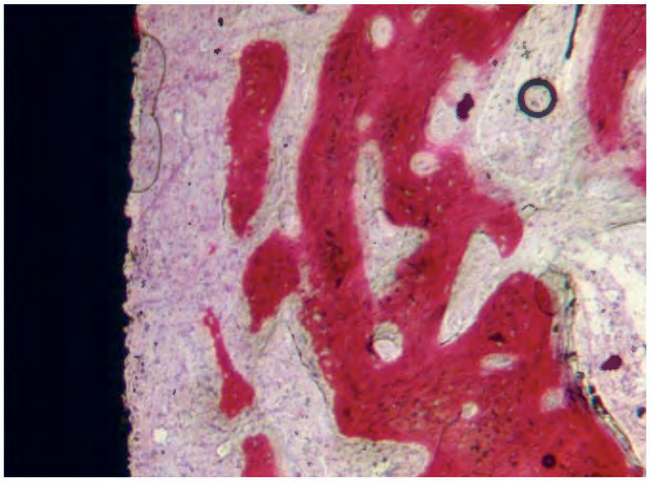


Figure 27-1 Machined surface. Distance osteogenesis. Newly formed bone is growing toward the implant surface. (Acid fuchsin-toluidine blue; ×40.)

WWW.HIGHDENT.IR

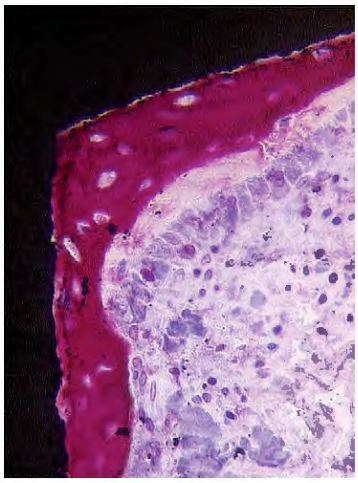


Figure 27-2 Surface sandblasted with Al_2O_3. Contact osteogenesis. A rim of osteoblasts has deposited osteoid matrix directly on the implant surface. Strongly stained, newly formed bone with large osteocyte lacunae is present at the interface with the implant. (Acid fuchsin-toluidine blue; ×100.)

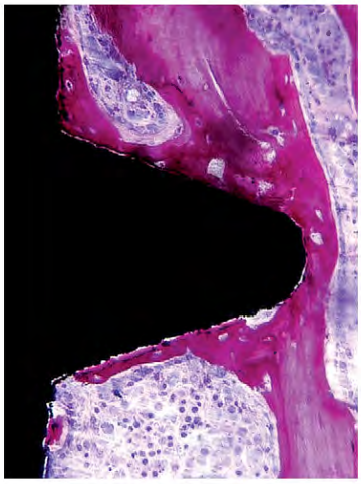


Figure 27-3 Surface sandblasted with Al_2O_3. Newly formed bone is in close, tight contact with the implant surface. No gaps are present at the bone-implant interface. (Acid fuchsin-toluidine blue; ×40.)

of osteoblasts.[33,42] On the other hand, fibroblasts were found to adhere with more difficulty to this surface; this could limit the soft tissue proliferation and potentially benefit bone formation.[43,44]

Studies have compared the BIC of turned titanium and sandblasted surfaces with Al_2O_3 particles. Implants were inserted in the femoral knee joints of rabbits, and the blasted sites presented BIC values statistically higher than the turned surface sites.[45] In another study in rabbits, turned implants (S_a = 0.96 µm) were compared with implants blasted with Al_2O_3 particles of different sizes, to obtain surfaces with different roughnesses. Mean roughness values (S_a) of 1.2 µm, 1.43 µm, and 2.2 µm were observed respectively, with particles of 25 µm, 75 µm, and 250 µm. After 12 weeks, all blasted surfaces (BIC values ranging from 31% to 47%) demonstrated more bone in contact with the implant surface compared with turned surfaces (BIC values ranging from 18% to 23%). Additionally, a higher percentage of bone was found in close contact with the implant surfaces in the sites in which 75-µm particles were used.[46]

Blasting procedures leave residual particles on the surface of the implant, and this could modify the bone-healing process. Some authors believe that the presence of Al_2O_3 particles remaining may be beneficial to osseointegration, catalyzing this process,[47] whereas other researchers believe that aluminum ions may impair bone formation by a possible competitive action to calcium.[44,48-50] A study was developed with the aim of describing the effects of residual Al_2O_3 particles on the implant surface on the integration of titanium surfaces. Dental implants were sandblasted (Al_2O_3 100- to 120-µm particles) or sandblasted and decontaminated (ASTM Committee F [ASTM F] 86-68 decontamination process in an ultrasonic bath), and inserted in rabbits. After 4 weeks, no statistically significant differences were observed between the groups based on the following analyses: BIC value, quantification of multinucleated cells or osteoclasts in contact with the implant surface, quantification of multinucleated cells or osteoclasts 3 mm from the implant surface. The authors suggested these histological results did not provide evidence to support the hypothesis that residual Al_2O_3 particles on the implant surface could affect the osseointegration of titanium dental implants.[51]

Alternatives to blasting with Al_2O_3 particles have also been tested. Blasting a surface with TiO_2 particles was proposed to promote a modification on the implant by using a component of the oxide layer naturally formed around titanium implants. In a rabbit comparison study, between surfaces blasted with Al_2O_3 25-µm particles (R_a = 0.84 µm) or TiO_2 25-µm particles (R_a = 0.96 µm), implants were inserted in the tibia. After 12 weeks, no statistically different values, concerning torque removal (26.5 N-cm and 24.9 N-cm, respectively, in Al_2O_3 and TiO_2 sites) or BIC values (49.2% and 47.6%, respectively, in Al_2O_3 and TiO_2 sites), were observed between these surfaces blasted with the same size of particles.[40]

Dental implants with TiO_2-blasted surface (25-µm particles) (R_a = 0.88 µm) have also been compared with turned implants (R_a = 0.39 µm) in the tibiae of rabbits. Twelve weeks after the insertion, TiO_2-blasted implants showed a statistically significant greater removal torque force (35.4 N-cm) than turned implants (29.2 N-cm). However, BIC values were not significantly different between the implants but were generally higher in the blasted group (40.9% and 34.5%, respectively).[39] In another animal model, a comparison between TiO_2-blasted and turned surfaces was performed by placing dental implants in fresh extraction sockets in dogs. After a 12-week healing period (no prosthetic rehabilitation was performed), no significant differences were observed between the surfaces concerning BIC (69% for both groups); blasted implants, however, showed higher remo-

WWW.HIGHDENT.IR

Table 27-2 Comparative Studies That Used Sandblasted Implants

AUTHOR	IMPLANT SURFACE	RESULTS	FINDINGS	EXPERIMENTAL DESIGN
Wennerberg et al.*	1. Al_2O_3 blasted (25-µm, 75-µm, and 250-µm particles) 2. Turned	BIC values: 1. 31% to 47% 2. 18% to 23%	Blasted surfaces demonstrated more bone in contact to implant surface compared with turned surface	Implants inserted in the tibiae of rabbits Healing period: 12 weeks
Wennerberg et al.†	1. Al_2O_3 blasted (25-µm particles) 2. TiO_2 blasted (25-µm particles)	BIC values: 1. 49.2% 2. 47.6% Removal torque: 1. 26.5 N-cm 2. 24.9 N-cm	No statistically different values concerning torque removal BIC values between surfaces blasted with same-size particles	Implants inserted in the tibiae of rabbits Healing period: 12 weeks
Wennerberg et al.‡	1. TiO_2 blasted (25-µm particles) 2. Turned	BIC values: 1. 40.9% 2. 34.5% Removal torque: 1. 35.4 N-cm 2. 29.2 N-cm	BIC values were not significantly different between the implants; however, TiO_2-blasted implants demanded a statistically significant greater removal torque force than turned implants	Implants inserted in the tibiae of rabbits Healing period: 12 weeks
Gotfredsen et al.§	1. TiO_2 blasted (10- to 53-µm particles) 2. Turned	Removal torque: 1. 150 N-cm 2. 60 N-cm	BIC not significantly difference (data not shown), but blasted implants presented higher removal torque values compared with turned sites	Implants placed immediately in dogs; no prosthetic rehabilitation performed Healing period: 12 weeks
Ivanoff et al.‖	1. TiO_2 blasted (25-µm particles) 2. Turned	BIC values: 1. 37% 2. 9%	Analysis of results revealed a significantly higher BIC for blasted implants versus turned groups	Microimplants inserted in the ridges of 27 patients Mean healing period: 3.9 to 6.3 months.

BIC, Bone-implant contact.

*Wennerberg A, Hallgren C, Johansson C et al: A histomorphometric evaluation of screw-shaped implants each prepared with two surface roughnesses, *Clin Oral Implants Res* 9(1):11-19, 1998.

†Wennerberg A, Albrektson T, Andersson B: Bone tissue response to commercially pure titanium implants blasted with fine and coarse particles of aluminum oxide, *Int J Oral Maxillofac Implants* 11:38-45, 1996.

‡Wennerberg A, Albrektsson T, Andersson B et al: A histomorphometric and removal torque study of screw-shaped titanium implants with three different surface topographies, *Clin Oral Implants Res* 6:24-30, 1995.

§Gotfredsen K, Nimb L, Hjorting-Hansen E et al: Histomorphometric and removal torque analysis for TiO2-blasted titanium implants. An experimental study on dogs, *Clin Oral Implants Res* 3:77-84, 1992.

‖Ivanoff CJ, Hallgren C, Widmark G et al: Histologic evaluation of the bone integration of TiO(2) blasted and turned titanium microimplants in humans, *Clin Oral Implants Res* 12:128-134, 2001.

val torque values (150 N-cm) compared with turned sites (60 N-cm).[52]

In a comparison study between TiO_2-blasted (25-µm particles) (S_a = 1.43 µm) and turned surfaces (S_a = 1.47 µm) in humans, microimplants were inserted in 27 patients and retrieved after a mean healing time of 6.3 months in the maxillae and 3.9 months in the mandible. The analysis of the results revealed a significantly higher BIC for the blasted implants than turned groups (37% and 9%, respectively).[53] However, these findings may not necessarily modify a clinical outcome. For example, in a clinical study, implants with TiO_2-blasted (S_a values of approximately 1.5 µm) and turned (S_a values of approximately 0.7 µm) surfaces were placed in the edentulous ridges of 18 patients. Prosthetic rehabilitation was performed, and after 2-year follow-up, no statistically significant differences between groups were found in the cumulative success rate (100% and 97.7%, respectively) or in marginal bone loss (–0.2 mm and 0.0 mm, respectively).[54]

Modifications on zirconium oxide (ZrO_2) surfaces have also been proposed to improve the bone-implant interface.[55] Implants with modified surfaces were investigated in monkeys. ZrO_2 implants (Al_2O_3 sandblasting with 50-µm particles) were compared with titanium (Al_2O_3 sandblasting with 50-µm particles; followed by hydrogen peroxide [H_2O_2] and hydrofluoric acid [HF] etching). Nine months after implant placement, metallic prostheses were installed, and 5 months later, BIC values corresponded to 67.4% for ZrO_2 and 72.9% for the titanium implants. However, differences were not statistically significant.[56]

WWW.HIGHDENT.IR

PLASMA-SPRAYED SURFACES

The use of implants with plasma-sprayed surfaces has been reported in orthopedics studies since the 1970s.[57] Later it was observed that, around dental implants, bone was formed without an intervening layer of connective tissue.[58] Plasma-sprayed implants are prepared by spraying molten metal on the titanium base, which results in a surface with irregularly sized and shaped valleys, pores, and crevices,[59] increasing the microscopic surface area by 6 to 10 times.[12] This topography may improve the fixation of implants by the growth of bone into the coating, forming a mechanical interlock.[60]

SURFACE COATING

Titanium Plasma Spray

The titanium plasma spray (TPS) surface has been reported to increase the surface area of the bone-implant interface and acts similarly to a three-dimensional surface, which may stimulate adhesion osteogenesis (Figures 27-4 and 27-5). The surface area increase has been reported to be as great as 600%. Although tremendous increase in total surface area occurs at the microscopic level, the actual load-bearing capability of the coating increases the functional area by 25% to 30%, which is still substantial. Porous surfaces in the range of TPS (150 to 400 µm) also increase the tensile strength of the bone-implant interface, resist shear forces, and improve load transfer. The increased surface roughness may also improve the initial fixation of the implant, especially in softer bone. Some evidence indicates that the interface may form faster, but no consensus exists regarding whether that may shorten clinical healing times.

A comparison of the biological response of plasma-sprayed (R_a = 7.345 µm) and turned surfaces (R_a = 0.350 µm) was performed in baboons. Six months after the implantation (no prosthetic rehabilitation was performed), no statistically significant differences could be observed between groups concerning the BIC percentage (55.9% and 56.2%, respectively, in plasma-sprayed and turned surfaces).[61] Plasma-sprayed implants were used in studies in rabbits,[62] monkeys,[63,64] and humans,[65-68] even under immediate placement (i.e., implants placed in fresh extraction sites) and immediate-loading conditions.[69] In Table 27-3, comparative studies in which plasma-sprayed implants were evaluated are presented.

Plasma-sprayed surfaces obtained with more reactive materials have been proposed to accelerate and enhance the bone ingrowth into pores of the implant surface. An alkali modification was proposed after plasma spraying, using sodium hydroxide solutions at 40° C for 24 hours. The oxide layer in modified surfaces (R_a = 18.2 µm) measured about 150 nm, whereas in plasma-sprayed sites (R_a = 17.6 µm) it measured less than 20 nm. Modified and nonmodified plasma-sprayed implants were placed in the femurs of dogs and analyzed 1, 2, and 3 months after insertion. After the first month, values of the push-out test (2.6 MPa for modified implants, which is about 1.5 times of that of the nonmodified implants) and of BIC (60.5% for alkali-modified implants and 20.2% for nonmodified implants) were significantly higher statistically in modified sites. No differences were observed after the second and third month (values were not presented). The authors suggested that the alkali modification may be beneficial to reduce clinical healing times and thus to improve implant success rates.[60]

One disadvantage of using the plasma-sprayed implants is the detachment of titanium after implant insertion. Franchi et al.[70] investigated the detachment of particles around plasma-sprayed, sandblasted, and

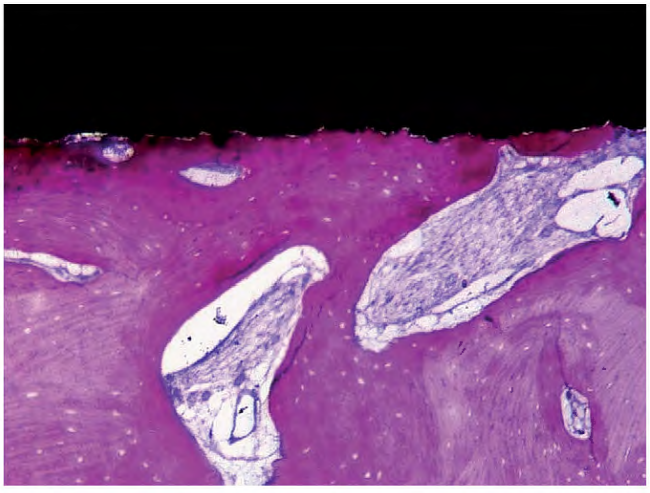


Figure 27-4 Titanium plasma spray surface. Close adaptation of the bone to the implant surface. No gaps or connective tissue are present at the bone-implant interface. (Acid fuchsin-toluidine blue; ×40.)

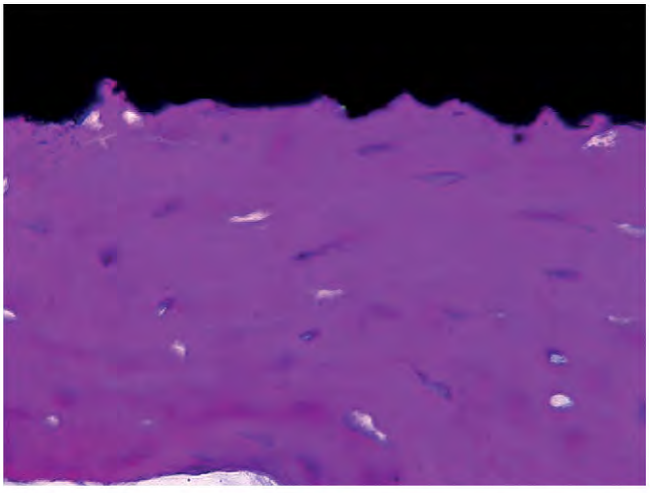


Figure 27-5 Titanium plasma spray surface. At higher magnification, it is possible to observe that bone has grown into the finest irregularities of the implant surface. (Acid fuchsin-toluidine blue; ×200.)

WWW.HIGHDENT.IR

Table 27-3 Comparative Studies That Used Acid-Etched and Plasma-Sprayed Implants

AUTHOR	SURFACE TREATMENT	RESULTS	FINDINGS	EXPERIMENTAL DESIGN
Klokkevold et al.*	1. Acid etched (HCl/H_2SO_4) 2. Turned	Removal torque: 1. 20.50 N-cm 2. 4.95 N-cm	Resistance to torque removal was four times greater for acid-etched implants compared with the turned surfaces	Implants were inserted in the femurs of rabbits Healing period: 2 months
Cho and Park†	1. Acid etched (HF and HCl/H_2SO_4) 2. Turned	Removal torque: 1. 34.7 N-cm‡ 2. 15.2 N-cm	Dual acid-etched implants required a higher removal torque average force versus the turned surface implants	Implants were inserted in the tibiae of rabbits Healing period: 12 weeks
Weng et al.§	1. Acid etched (Osseotite) 2. Turned (ICE)	BIC values: 1. 62.5% 2. 39.5%	BIC values were significantly higher in dual acid-etched sites compared with turned sites	Implants were inserted in areas with poor bone quality in the mandibles of dogs Healing period: 4 months
Klokkevold et al.‖	1. Acid etched (HCl/H_2SO_4) 2. Plasma spray 3. Turned	Removal torque: 1. 27.40 N-cm 2. 59.23 N-cm 3. 6.73 N-cm	Statistically significant differences were observed between acid-etched and turned implants and between plasma-sprayed and turned implants; however, differences between acid etched and plasma sprayed were not statistically different	Implants were inserted in the femurs of rabbits Healing period: 3 months¶
Carr et al.#	1. Plasma spray 2. Turned	BIC values: 1. 55.9% 2. 56.2%	No significant differences could be observed between groups concerning the BIC percentage	Implants were inserted in the mandibles of baboons; no prosthetic rehabilitation was performed Healing period: 6 months

HCl, Hydrochloric acid; *H_2SO_4,* sulfuric acid; *HF,* hydrofluoric acid; *ICE*, incremental cutting edge; *BIC,* bone-implant contact.
*Klokkevold PR, Nishimura RD, Adachi M et al: Osseointegration enhanced by chemical etching of the titanium surface. A torque removal study in the rabbit, *Clin Oral Implants Res* 8:442-447, 1997.
†Cho SA, Park KT: The removal torque of titanium screw inserted in rabbit tibia treated by dual acid etching, *Biomaterials* 24:3611-3617, 2003.
‡These results refer to a 24% HF and 70% HCl/H_2SO_4 group.
§Weng D, Hoffmeyer M, Hurzeler MB et al: Osseotite vs. machined surface in poor bone quality. A study in dogs, *Clin Oral Implants Res* 14:703-708, 2003.
‖Klokkevold PR, Johnson P, Dadgostari S et al: Early endosseous integration enhanced by dual acid etching of titanium: a torque removal study in the rabbit, *Clin Oral Implants Res* 12:350-357, 2001.
¶Data from the first and second healing periods are not included.
#Carr AB, Gerard DA, Larsen PE: Histomorphometric analysis of implant anchorage for 3 types of dental implants following 6 months of healing in baboons, *Int J Oral Maxillofac Implants* 15:785-791, 2000.

acid-etched (Al_2O_3 with 100-µm particles or ZrO_2 with 120-µm particles) and turned implants. Fourteen days after placement in femoral and tibial diaphyses of sheep, titanium debris was observed only in the plasma-sprayed samples. In another investigation in which the tissues around plasma-sprayed and HA-coated implants were evaluated, the presence of granules of titanium was found only around the plasma-sprayed surfaces.[71,72] This phenomenon could be related to the friction between the implant surface and the host bone cavity during implant placement, but its implications are not clear.

Acid-Etched Surfaces

Acid-etching a titanium base was proposed to modify the implant surface without leaving the residues found after the sandblasting procedure, to avoid the nonuniform treatment of the surface, and to control the loss of metallic substance from the body of the implant (Figures 27-6 and 27-7).[12] This is performed using baths of hydrochloric acid (HCl), sulfuric acid (H_2SO_4), HF, and nitric acid (HNO_3) in different combinations. The roughness before etching, the acid mixture, the bath temperature, and the etching time all affect the acid-etching process. Table 27-4 lists the results from histologic evaluation of retrieved implants from humans.

Researchers compared etched (HCl and H_2SO_4) and turned surfaces by inserting implants in rabbit femurs.[8] The roughened surface was characterized by "an even distribution of very small (1 to 2 µm) peaks and valleys." The resistance to torque removal was evaluated 2 months after implant placement in rabbits, and it was found

WWW.HIGHDENT.IR
دندانسازان و دندانپزشکان

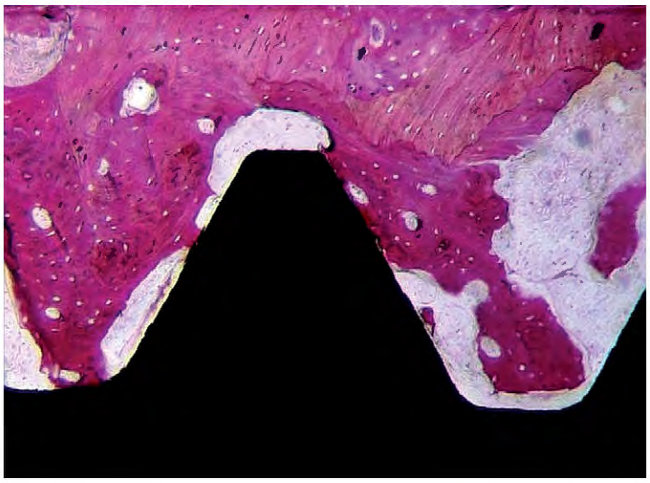


Figure 27-6 Acid-etched surface. Newly formed bone is growing toward the implant surface. (Acid fuchsin-toluidine blue; ×40.)

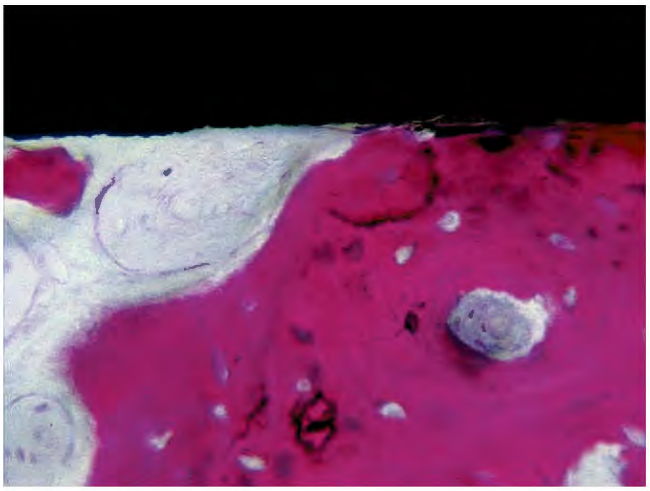


Figure 27-7 Acid-etched surface. At higher magnification it is possible to observe on the right that bone is in close contact with the implant surface. On the left, a marrow space is found at the interface with the implant surface. (Acid fuchsin-toluidine blue; ×200.)

Table 27-4 Histologic Studies in which Acid-Etched Implants Were Retrieved from Humans

AUTHOR	SURFACE TREATMENT	RESULTS	FINDINGS	EXPERIMENTAL DESIGN
Testori et al.*	Acid etched (Osseotite)	BIC values: 78% to 85%	Implants used successfully in immediately loaded protocol	Histologic analysis involved two retrieved, immediately loaded implants Healing period: 4 months
Degidi et al.†	Acid etched (HCl and H_2SO_4)	Mean BIC value: 61.3%	No gaps or fibrous tissues were observed at the interface	Histologic analysis involved two retrieved implants; no prosthetic rehabilitation was performed Healing period: 6 months
Trisi et al.‡	1. Acid etched (Osseotite) 2. Turned	BIC values: 1. 72.35% 2. 35.32%	BIC values in dual acid–etched sites were statistically higher than in turned sites	Histologic analysis involved implants inserted in the posterior maxilla of 11 patients Healing period: 6 months

BIC, Bone-implant contact.

*Testori T, Szmukler-Moncler S, Francetti L et al: Immediate loading of Osseotite implants: a case report and histologic analysis after 4 months of occlusal loading, *Int J Periodontics Restorative Dent* 21:451-459, 2001.

†Degidi M, Petrone G, Iezzi G et al: Bone contact around acid-etched implants: a histological and histomorphometrical evaluation of two human-retrieved implants, *J Oral Implantol* 29:13-18, 2003.

‡Trisi P, Lazzara R, Rao W et al: Bone-implant contact and bone quality: evaluation of expected and actual bone contact on machined and Osseotite implant surfaces, *Int J Periodontics Restorative Dent* 22:535-545, 2002.

to be four times greater for acid-etched implants (20.50 N-cm) compared with the turned ones (4.95 N-cm).

A dual acid–etched technique has been proposed to produce a microtextured (instead of a macrotextured) surface, which could be more predisposed to achieve desirable results.[73] This is because higher adhesion of platelet genes and higher expression of extracellular genes were observed in this dual acid–etched surface.[74,75] Dental implants were inserted in areas with poor bone quality in dogs (mandibular premolar teeth were extracted and left healing for 8 months). Four months later (no prosthetic rehabilitation was performed), BIC values were significantly higher for dual acid–etched implants (62.9%) as compared with turned sites (39.5%).[76]

Degidi et al.[77] presented the histologic analysis of two dual acid–etched implants (HCl and H_2SO_4) retrieved from humans. The implants were removed 6 months after implantation because of the positioning of the implants in contact with the inferior alveolar nerve and

WWW.HIGHDENT.IR

Figure 27-8 Sandblasted and acid-etched surface. Thin lamellae of newly formed bone line almost all the implant surface. (Acid fuchsin-toluidine blue; ×40.)

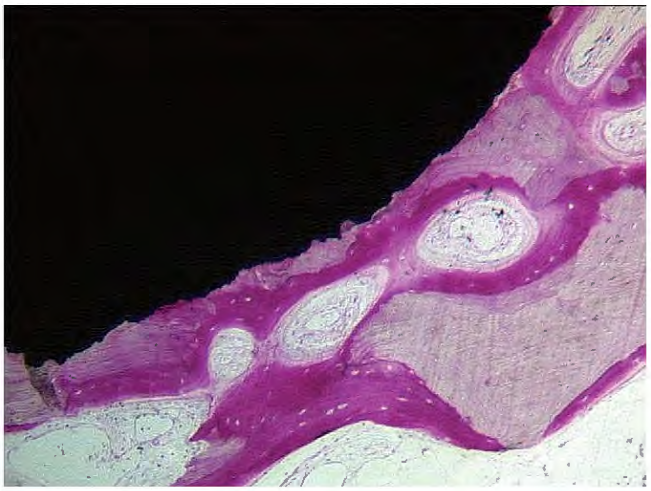


Figure 27-9 Sandblasted and acid-etched surface. At higher magnification, newly formed bone is in close contact and lines almost completely the implant perimeter. (Acid fuchsin-toluidine blue; ×100.)

resultant discomfort. These two integrated implants presented a mean BIC percentage of 61.3%, with no gaps or fibrous tissues present at the interface.

The use of dual acid–etched implants in immediate-loading procedures was histologically investigated in humans. Two implants were inserted in the mandible of a patient to immediately support a provisional fixed partial denture (FPD). Four months after occlusal loading, during the uncovering procedure of the submerged implants, those immediately loaded implants were retrieved, and histomorphometric evaluation revealed high levels of BIC, ranging from 78% to 85%.[78] These results were confirmed in an experimental study in humans in which microimplants (measuring 2 × 5 mm, with one side treated by dual acid etching and the other side left turned) were used. These implants were inserted in the posterior maxillae of 11 patients; 6 months later, the BIC values in dual acid–etched sites (72.35%) were statistically higher than in turned sites (35.32%).[79]

In a comparison among dual acid–etched (R_a = 494.16 nm), plasma-sprayed (R_a = 7.01 nm), and turned (R_a = 185.14 nm) surfaces, mean torque values were evaluated in rabbit femurs, 1, 2, and 3 months after implant placement. Values obtained at the third month were 27.40 N-cm, 59.23 N-cm, and 6.73 N-cm, respectively. Moreover, at the earliest time point, the stability of dual acid-etched implants was comparable to that of plasma-sprayed implants, whereas the stability of the turned implants was comparatively lower.[73]

Cho and Park[80] tested different acid concentrations in implants placed in the tibiae of rabbits. For the preparation of the dual acid–etched surfaces, the first bath was performed using HF in different concentration (12%, 24%, or 48%), and the second bath was performed with 70% HCl and H_2SO_4. Turned surfaces were used as the control. Twelve weeks after implant placement, the removal torque required for dual acid–etched implants (34.3 N-cm, 34.7 N-cm, and 38.7 N-cm respectively) was statistically higher than that for the turned implants (15.2 N-cm). The authors concluded that less correlation exists between removal torque and the difference in HF volume percent and that rough acid-etched implants achieved greater resistance to reverse torque removal than machined implants.

Sandblasted and Acid-Etched Surfaces

In the 1990s the study of a modified surface resultant from blasting (to produce a macrotexture) followed by acid etching (to produce a final microtexture) showed promising results. The resultant surface was constituted by uniformly scattered gaps and holes, and it appeared to be slightly less rough than the plasma-sprayed surface, which presented a deeply irregular texture that provided a less favorable environment for cell spreading (Figures 27-8 to 27-11).[81] In Table 27-5, comparative studies in which acid-etched implants were evaluated are presented; Table 27-6 lists the results from histologic evaluation of retrieved implants from humans.

A comparison study between sandblasted and acid-etched surfaces (R_a = 2.0 µm) and acid-etched surfaces (R_a = 1.3 µm) was performed in miniature pigs. Up to 3 months, implants with sandblasted and acid-etched surfaces presented significantly high torque removal values (186.8 N-cm), which were 75% to 125% higher than those observed in acid-etched sites (95.7 N-cm).[82] In a dog model, Abrahamsson et al.[83] evaluated the pattern of bone formation in blasted and acid-etched (S_a = 2.29 µm) and turned (S_a = 0.35 µm) surfaces. BIC values were significantly greater in sandblasted and acid-etched sites than in turned surfaces 1, 2, 4, 6, 8, and 12 weeks after implant placement. Most of the data were presented as a graphic, without corresponding numerical values; however, in the twelfth week the BIC

WWW.HIGHDENT.IR

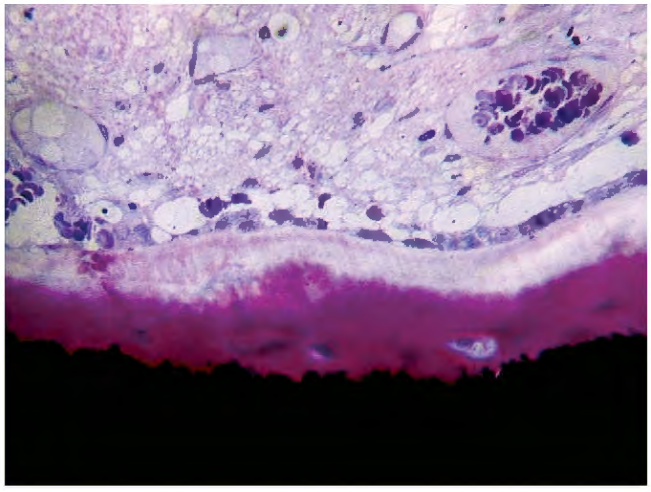


Figure 27-10 Sandblasted and acid-etched surface. At higher magnification it is possible to see that bone is in close contact with the implant surface with no intervening gaps. A rim of osteoblasts is depositing osteoid matrix. (Acid fuchsin-toluidine blue; ×200.)

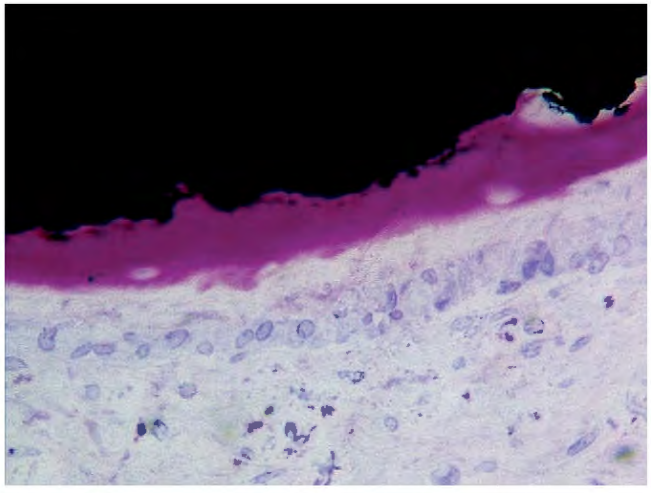


Figure 27-11 Sandblasted and acid-etched surface. Bone has grown inside the irregularities of the implant surface. (Acid fuchsin-toluidine blue; ×200.)

was approximately 60% in sandblasted and acid-etched implants and approximately 40% in turned implants. The authors concluded that the rate and degree of osseointegration is superior in sandblasted and acid-etched implants.

Sandblasted and acid-etched implants tend to promote greater osseous contact at earlier time points compared with plasma-sprayed, coated implants. This conclusion was derived from a dog study in which the test surface was prepared by blasting with 250- to 500-µm corundum particles, and the acid etching was done with HCl and H_2SO_4. Histologic analyses were performed 3 months after implant placement (3 months of healing), 3 months after loading (6 months of healing), and 12 months after loading (15 months of healing). Sandblasted and acid-etched implants had a significantly higher percentage of BIC than did the plasma-sprayed implants after 3 months of healing (72.33% and 52.15%, respectively). Differences were not observed after 6 months of healing on sandblasted and acid-etched and plasma-sprayed implants (68.21% and 78.18%, respectively). After 12 months of loading, BIC values were significantly higher in sandblasted and acid-etched than in plasma-sprayed sites (71.68% and 58.88%, respectively).[84] Sandblasted and acid-etched surfaces also present better osteoconductive properties and a higher capability to induce cell proliferation than plasma-sprayed surfaces.[71,81,85]

In the maxillae of miniature pigs, the removal torque values were evaluated in sandblasted and acid-etched (sandblasted with 0.25- to 0.50-µm particles, and acid-etched with HCl and H_2SO_4) (R_a = 2.0 µm), plasma-sprayed (R_a = 3.1 µm), and turned (R_a = 0.15 µm) surfaces, after 4, 8, and 12 weeks of healing. The values in sandblasted and acid-etched (values ranged between 1.31 and 1.43 N-cm) and plasma-sprayed (values ranged between 1.14 and 1.54 N-cm) implants were significantly higher statistically than turned implants (values ranged between 0.15 and 0.26 N-cm). Values between sandblasted and acid-etched and plasma-sprayed sites were not statistically significant.[86]

The analysis of sandblasted and acid-etched implants in humans was performed in studies by analyzing retrieved implants from patients. In a case report, an implant was inserted in the palatal bone of the maxilla of a patient as anchorage for orthodontic treatment, and 6 months later the implant was retrieved; the BIC was 76.6%.[85] In another case report, an implant was removed after 40 months of function, for a fracture of the abutment screw, and a 75.4% BIC value was observed.[87]

Some authors suggest that, in cases of healthy patients with sufficient bone volume and of patients with good bone quality (Classes I to III), sandblasted and acid-etched implants can be restored after approximately 6 weeks of healing, with a high predictability of success.[88] Of course, it is also reported that immediate loading after implant insertion is possible, with a wide range of implants and surface conditions.

Sandblasting can be performed using different abrasive particles. For example, the surface obtained from acid etching and sandblasting with ZrO_2 particles is reported to favor a better bone deposition as compared with plasma-sprayed and turned surfaces.[89]

In another situation, the response of osteoblasts to acid-etched and sandblasted surfaces prepared with Al_2O_3 or ZrO_2 particles was compared with plasma-sprayed (R_a = 9.5 µm) and turned (R_a = 0.56 µm) surfaces. Blasting with Al_2O_3 was performed with 100-µm (R_a = 1.98 µm) and 150-µm (R_a = 1.14 µm) particles, and blasting with ZrO_2 was performed with 60-µm (R_a = 1.52 µm) and 120-µm (R_a = 1.32 µm) particles. The

WWW.HIGHDENT.IR

Table 27-5 Comparative Studies That Used Sandblasted and Acid-Etched Implants

AUTHOR	SURFACE TREATMENT	RESULTS	FINDINGS	EXPERIMENTAL DESIGN
Orsini et al.*	1. Sandblasted and acid etched 2. Turned		Roughened surfaces could improve initial cell anchorage, providing better osseointegration	Implants were evaluated in vitro concerning the cellular responsiveness to the surfaces
Abrahamsson et al.†	1. Sandblasted and acid etched 2. Turned		BIC values (data not shown) were significantly greater in sandblasted and acid-etched sites versus turned surfaces	Implants were inserted in the mandibles of dogs; no prosthetic rehabilitation was performed Healing period: 1, 2, 4, 6, 8, and 12 weeks
Cochran et al.‡	1. Sandblasted and acid etched (250- to 500-μm corundum particles and etched with HCl/H_2SO_4) 2. Plasma sprayed	BIC values: 1. 71.68% 2. 58.88%	The sandblasted and acid-etched implants had a significantly greater BIC percentage than did the plasma-sprayed implants; however, no qualitative differences in bone tissue were observed between groups	Implants were inserted in the mandibles of dogs Loading period: 12 months Healing period: 15 months
Buser et al.§	1. Sandblasted and acid etched (0.25- to 0.50-μm particles etched with HCl/H_2SO_4) 2. Plasma sprayed 3. Turned	Removal torque: 1. 1.43 N-cm 2. 1.54 N-cm 3. 0.26 N-cm	Statistically significant differences were observed between sandblasted and acid-etched and turned implants, as well as between plasma-sprayed and turned implants; however, differences between sandblasted and acid etched and plasma sprayed were not statistically different	Implants were inserted in the maxillae of miniature pigs; no prosthetic rehabilitation was performed Healing period: 12 weeks‖

HCl, Hydrochloric acid; *H_2SO_4,* sulfuric acid; *BIC,* bone-implant contact.

*Orsini G, Assenza B, Scarano A et al: Surface analysis of machined versus sandblasted and acid-etched titanium implants, *Int J Oral Maxillofac Implants* 15:779-784, 2000.

†Abrahamsson I, Berglundh T, Linder E et al: Early bone formation adjacent to rough and turned endosseous implant surfaces. An experimental study in the dog, *Clin Oral Implants Res* 15:381-392, 2004.

‡Cochran DL, Schenk RK, Lussi A et al: Bone response to unloaded and loaded titanium implants with a sandblasted and acid-etched surface: a histometric study in the canine mandible, *J Biomed Mater Res* 40:1-11, 1998.

§Buser D, Nydegger T, Oxland T et al: Interface shear strength of titanium implants with a sandblasted and acid-etched surface: a biomechanical study in the maxilla of miniature pigs, *J Biomed Mater Res* 45:75-83, 1999.

‖Data from the first and second healing periods are not included.

best growth and differentiation patterns were obtained in microroughed surfaces blasted with 60-μm ZrO_2 particles, which in this study were also the cleanest surface, with no toxic contaminating residues. The authors suggested that the depth and distribution of irregularities, the cavity morphology, and the presence of contaminating elements derived from the treatment procedures seems to play an important role in cell behavior.[90]

A specific method to produce HA-blasted implants was proposed—the "resorbable blast material" (Figure 27-12). The base of titanium is submitted to blasting, followed by a passivity procedure to remove the calcium phosphate ($CaPO_4$) and, finally, cleaning. The blast media is resorbed during these processes, and a surface of pure TiO_2 is produced that is free of contaminants.[91] In a comparison performed in rabbits, 8 weeks after implantation the BIC value in the modified surface (62.3%) (R_a = 2.14 μm) was significantly higher than the 51% observed in the turned surface (R_a = 0.78 μm).[92] In a 50-month follow-up study, 1077 implants were placed in 348 patients, resulting in a 99.3% success rate in the mandible and 100% in the maxilla.[93]

Anodized Surfaces

The oxidation process has been used in dental implants to change the characteristics of the oxide layer and, consequently, to improve the biocompatibility of the surface (Figure 27-13).[27] The advantage is to modify

WWW.HIGHDENT.IR
دانلود از سایت دندانسازان و دندانپزشکان

Table 27-6 Histologic Studies in which Sandblasted and Acid-Etched Implants Were Retrieved from Humans

AUTHOR	SURFACE TREATMENT	RESULTS	FINDINGS	EXPERIMENTAL DESIGN
Hayakawa et al.*	Sandblasted and acid etched (Straumann)	BIC value: 76.6%	Bone surrounding the implant was uniformly and maturely structured	Histologic analysis involved one retrieved implant that was inserted in the palatal bone of the maxilla of a patient as anchorage for orthodontic treatment Healing period: 6 months
Sakakura et al.†	Sandblasted and acid etched	BIC value: 75.4%	"The surrounding bone healed in a well-organized pattern and could not be differentiated from the original alveolus"	Histologic analysis involved one retrieved implant of a patient Loading period: 40 months

BIC, Bone-implant contact.
*Hayakawa T, Kiba H, Yasuda S et al: A histologic and histomorphometric evaluation of two types of retrieved human titanium implants, *Int J Periodontics Restorative Dent* 22:164-171, 2002.
†Sakakura CE, Nociti FH Jr, Mello GP et al: Histomorphometric evaluation of a threaded, sandblasted, acid-etched implant retrieved from a human lower jaw: a case report, *Implant Dent* 14:289-293, 2005.

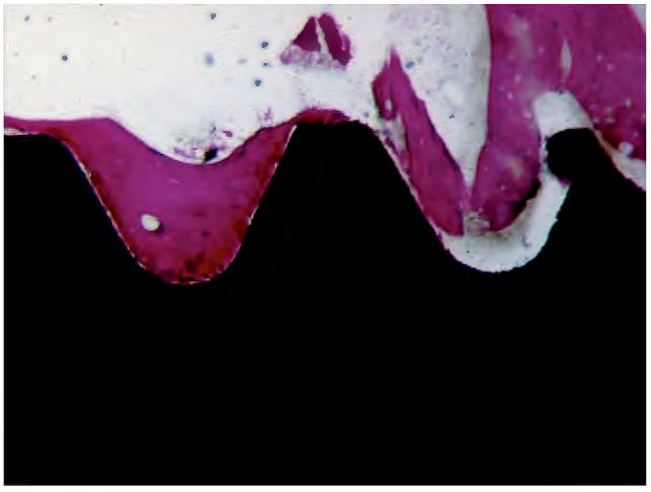


Figure 27-12 Resorbable blast media (RBM) surface. A high percentage of bone-implant contact (BIC) is present. (Acid fuchsin-toluidine blue; ×40.)

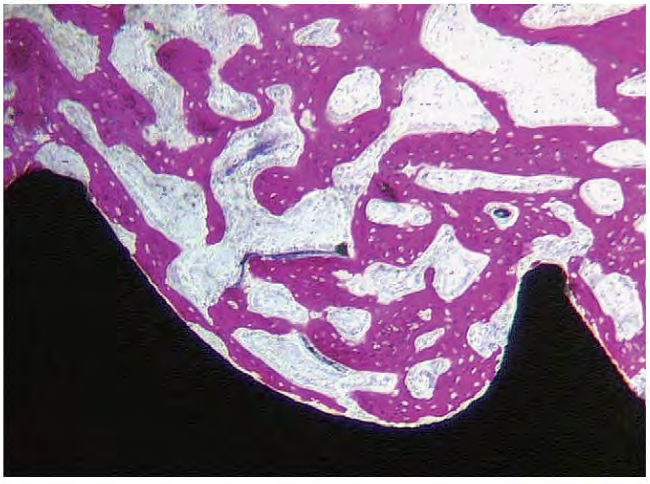


Figure 27-13 Anodized surface. Many newly formed bone trabeculae are present at the interface. (Acid fuchsin-toluidine blue; ×40.)

the surface without depositing grit particles. Anodized surfaces are prepared by applying a voltage on the titanium specimen immersed in an electrolyte. The resultant surface presents micropores of variable diameters and demonstrates lack of cytotoxicity; moreover, cell attachment and proliferation are enhanced as compared with turned surfaces.[94]

The removal torque of anodized surfaces with different oxide thicknesses (oxide thickness of approximately 200, 600, 800, or 1000 nm; S_a ranging from 0.96 to 1.03 µm) was also investigated in comparison with turned implants (oxide thickness of 17.4 nm; S_a = 0.83 µm). Six weeks after implant placement in rabbit tibiae, the values obtained (11.3, 12, and 12.9 N-cm, respectively) in anodized sites were significantly higher than those reported for the turned sites (removal torque value of 7.5 N-cm).[27] However, in rabbit tibiae, anodized, anodized and hydrothermally treated, and turned surfaced titanium implants were inserted. Six and 12 weeks after implantation, BIC values and removal torque strength were not statistically different among the groups.[95]

In humans an experiment was developed to compare anodized (S_a = 1.17 µm) and turned (S_a = 0.78 µm) surfaces. With this aim, microimplants (2.3 × 5 mm) were inserted in a suitable edentulous area in maxilla or mandible of 20 patients. Each patient had an oxidized and a turned implant inserted, and mean healing time

WWW.HIGHDENT.IR

was 6.6 months. BIC values were statistically higher in oxidized (34%) than in turned (13%) implants. The authors suggested that these results may be explained by the thicker oxide layer, increased surface roughness, different surface morphology in terms of porosity, or change in crystal structure.[96]

A comparison of the response of osteoblasts was evaluated using anodized (Nobel Biocare TiUnite, R_a = 0.76 µm), blasted and acid-etched (Dentsply Friadent DPS, R_a = 2.41 µm; Dentsply Friadent Plus, R_a = 2.75 µm; Straumann SLA, R_a = 2.93 µm), plasma-sprayed (Dentsply Friadent TPS, R_a = 3.5 µm), acid-etched (3i Osseotite, R_a = 0.86 µm), and turned (Nobel Biocare Mk III, R_a = 0.81 µm) surfaces. The rate of cellular spreading was significantly increased in surfaces combining blasting and acid etching. The authors observed that differentiation and calcification occurred on surfaces of both rough and smooth microstructure, and they speculated that a rough surface of porous microstructure may enhance the rate of cell spreading.[59] In Table 27-7, studies in which anodized implants were evaluated are presented.

HYDROXYAPATITE

Hydroxyapatite Coatings

Hydroxyapatite coatings have a similar roughness and increase in functional surface area as TPS (Figures 27-14, 27-15, and 27-16). A direct bone bond shown with HA coating and the strength of the HA-to-bone interface is greater than titanium to bone and even greater than TPS to bone. In addition, accelerated interfacial bone formation and maturation have been observed in dogs.[97] An initial implant-to-bone interface contract is essential for a predicable interface to form.[98] The space or "gap" between the implant and bone may affect the percentage of bone contact after healing. Gap healing may be enhanced by the HA coating.[99,100] The corrosion rate of metal is also reduced, which is more significant for cobalt chrome alloys.[102]

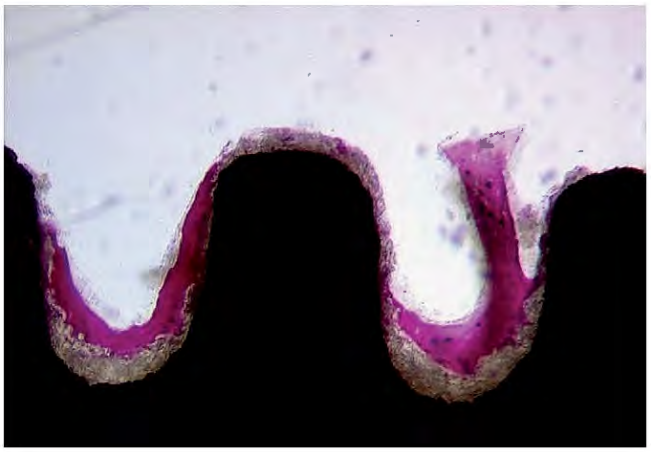


Figure 27-14 Hydroxyapatite-coated implant. A close contact of the bone with the coating is present. (Acid fuchsin-toluidine blue; ×40.)

Table 27-7 Comparative Studies That Used Anodized Implants

AUTHOR	SURFACE TREATMENT	RESULTS	FINDINGS	EXPERIMENTAL DESIGN
Sul et al.*	1. Anodized (oxide thickness approximately 200, 600, 800 or 1000 nm) 2. Turned (oxide thickness: 17.4 nm)	Removal torque: 1. 0.113 to 0.129 N-cm 2. 0.075 N-cm	Preliminary results of this study suggest that the oxide thickness influences the bone tissue formation	Implants were inserted in the tibiae of rabbits Healing period: 6 weeks
Son et al.†	1. Anodized 2. Turned	Removal torque: 1. 51.35 N-cm 2. 35.28 N-cm	Difference between groups was not statistically significant concerning removal torque and BIC values (data not shown)	Implants were inserted in the tibiae of rabbits Healing period: 12 weeks
Ivanoff et al.‡	1. Anodized 2. Turned	BIC values: 1. 34% 2. 13%	BIC values were statistically higher in oxidized than in turned sites	Histologic analysis involved implants inserted in the ridges of 20 patients Mean healing period: 6.6 months

BIC, Bone-implant contact.

*Sul YT, Johansson CB, Petronis S et al: Characteristics of the surface oxides on turned and electrochemically oxidized pure titanium implants up to dielectric breakdown: the oxide thickness, micropore configurations, surface roughness, crystal structure and chemical composition, *Biomaterials* 23:491-501, 2002.

†Data from the first healing period and an experimental group are not included; Son WW, Zhu X, Shin HI et al: In vivo histological response to anodized and anodized/hydrothermally treated titanium implants, *J Biomed Mater Res B Appl Biomater* 66:520-525, 2003.

‡Ivanoff CJ, Widmark G, Johansson C et al: Histologic evaluation of bone response to oxidized and turned titanium micro-implants in human jawbone, *Int J Oral Maxillofac Implants* 18:341-348, 2003.

WWW.HIGHDENT.IR
انجمن دندانسازان و دندانپزشکان

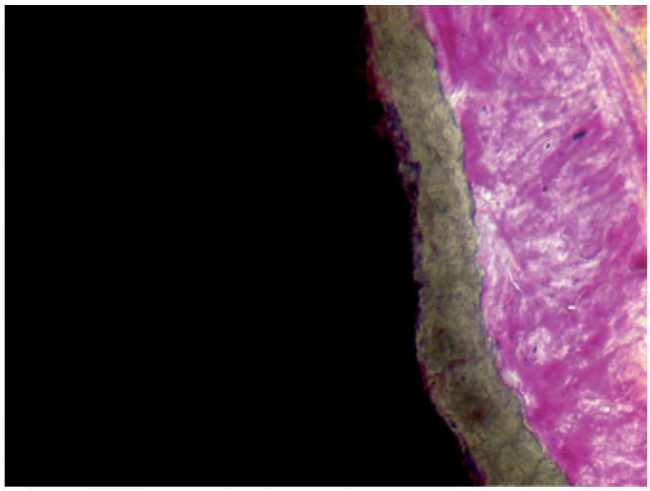


Figure 27-15 Hydroxyapatite (HA)-coated implant. A very tight connection is present between bone and HA coating with no intervening gaps at the interface. (Acid fuchsin-toluidine blue; ×100.)

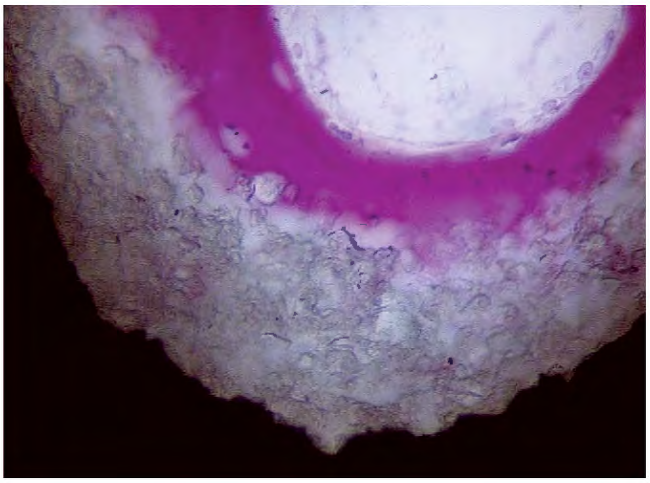


Figure 27-16 Hydroxyapatite (HA)-coated implant. At higher magnification it is possible to observe that bone is in close contact with the HA coating. (Acid fuchsin-toluidine blue; ×200.)

The clinical advantages of TPS or HA coatings may be summarized as the following:

1. Increased surface area
2. Increased roughness for initial stability
3. Stronger bone-implant interface

Additional advantages of HA over TPS include the following:

1. Faster-healing bone interface
2. Increased gap healing between bone and HA
3. Stronger interface than TPS
4. Less corrosion of metal

Disadvantages of Coatings

Although coatings on implant bodies have benefits, several disadvantages exist. The coating may flake, crack, or scale on insertion, especially when being inserted into dense bone. In addition, the increased surface roughness increases the risk of bacterial contamination when present above the bone. HA may not only increase plaque retention when exposed but also may act as a nidus for bacteria, and bacterial endotoxins may be more adherent because of the surface charge and characteristics.

Although a faster bone interface may develop,[30] it may be an unnecessary risk to reduce the healing time of the loaded implant interface. Coatings also increase the cost of the implant body, compared with uncoated implants. Therefore the disadvantages of coatings include the following:

1. Flaking, cracking, or scaling on insertion
2. Increased plaque retention when above bone
3. Increased bacteria and nidus for infection
4. Complication of treatment of failing implants
5. Increased cost

The HA or TPS coating should not be the only system of load transfer to the bone. This is especially important when bone loss occurs, and the coating must be removed for repair of the implant. However, a coating may enhance an implant body design.

As a consequence, the decision to use a coating may be based more on the bone density than nearly any other factor. Type 1 (D1) and type 2 (D2) bone have the greatest strength and bone contact. In addition, increased risk of material flaking from the implant occurs during insertion. The risk of implant failure as a result of implant design is reduced, as is the incidence of crestal bone loss. Rather than using a coating, D1 and D2 implants benefit from a roughened surface, sound biomechanical design, and a minimum implant length of 10 to 11 mm. Type 3 (D3) bone is approximately 50% weaker than D2 bone. As a result, the advantages of TPS outweigh its disadvantage. The increased initial fixation, increased bone contact, and greater strength of the interface all support its use. Care is taken to reduce crestal bone loss through sound design so that the TPS does not become exposed and the increased plaque retention is not a concern. Type 4 (D4) bone has proven to be the one most at risk. Therefore the benefits of HA are most required in the type of bone. Although it may have the greatest risk relative to bacteria, the benefits of gap healing, faster bone mineralization, and increased bone contact all favor HA. Higher success rates have been reported when HA-coated implants have been used in soft bone. The HA should be added to an implant body with the most macroscopic load-bearing surface area. To minimize crestal bone loss, larger diameter and an increased number of implants are also suggested in this very weak bone.

Synthetic HA is a ceramic material that was introduced as a surface modifier in the 1980s.[97,98] In nature

WWW.HIGHDENT.IR

this material is a component of bone; for this reason it can provide reliable, early, and strong fixation by chemical bonding with bone[99] on the so-called biointegration level.[100] HA surfaces have been used in dental implants to improve the bone-implant anchorage, and it was demonstrated that osteoblasts respond to this contact with immediate changes in gene expression associated with osteoblast adhesion, proliferation, extracellular matrix synthesis, and differentiation.[101]

Histological evidence of the clinical success of HA coating has been reported. Two dental implants, fractured because of an accident, were retrieved from a patient after an 18-month period of loading. Dense bone was found in close relation to the surface of the implants, and the interspaces of each thread were filled with mineralized bone. BIC values were 87.5% and 97.4%, and the authors reported that the connection between the HA coating and the metal was uniformly tight and constant (30 and 50 µm).[102] High survival rates for HA-coated implants have been reported in studies with 3, 8, and 12 years of follow-up.[103-105] Specifically in the 12-year follow-up study, 120 patients (156 titanium and 232 HA-coated implants) were evaluated. The survival rate of HA-coated implants was 93.2%, which was statistically higher than titanium implants (89%).[105]

Histological postmortem analyses of two implants removed 10 years after loading revealed 70.74% and 86.23% BIC values. Interestingly, HA-coating disappearance had occurred in 23.5% and 22.0% of the implant perimeter; however, in these areas bone was in direct apposition to the titanium surface.[106] Other authors also observed this phenomenon of resorption and degradability of HA coating when in contact with the biological environment.[107] For example, Buser et al.[71] demonstrated that this resorption can be followed by bone apposition, resulting in high BIC values.

In humans, in a postmortem evaluation, five titanium implants (85 months of loading) and two HA-coated implants (38 months of loading) were analyzed. Differences were observed concerning the arrangement and pattern of bone contact between the surfaces: HA coating was separated from the maxillary implants in some areas and was free within surrounding connective tissue or surrounded by invaginating sulcular epithelium.[108] Furthermore, in a case report by Piattelli et al.,[109] localized chronic suppurative bone infection was observed as a sequela of peri-implantitis in an HA-coated implant. The patient presented poor hygiene and signs of overloading caused by an imperfect prosthetic treatment plan. The authors observed that bacteria, which penetrated into the marrow spaces and destroyed the mineralized matrix of the bone, filled most of the surrounding bone. Despite the fact that the implant was clinically stable, the histologic analysis showed that the coating was almost completely detached from the titanium surface. Notwithstanding, this resorption must be higher with the presence of periodontal pathogens, which was demonstrated when experimental peri-implantitis was induced in dogs.[110,111] Thus the instability of the coating-substrate interface, the unstable duration of coating, and the lack of long-term observation provided a basis for some authors to question the clinical reliability of this material as a surface modifier.[112,113]

The contrasting results observed in the literature related to a HA surface in an implant body can be attributed to the quality of the different HA coatings, which include factors such as chemical composition and physical and mechanical properties. Different methods can be used to achieve HA coating: dip coating and sintering, electrophoretic deposition, immersion coating, hot isostatic pressing, solution deposition, sputter coating, and thermal spraying techniques (including plasma spraying, which is the method most frequently used).[114] Each of these processes leads to a difference in composition, physical and mechanical properties, thickness, and adhesion to the metal to which it the coating is applied.

HA plasma spraying was created to combine the chemical-bonding properties of HA and the bone-implant mechanical interlock achieved with the plasma-spraying technique. A comparison study between HA plasma-sprayed, plasma-sprayed, and turned cylindrical implants was performed in dogs 12 weeks after implantation (before loading) and 1 year after loading. After both time periods, significantly higher BIC values were observed for HA implants (77.8% and 88.9%, respectively) when compared with plasma-sprayed (58.6% and 64.9%, respectively) and turned (71.2% and 67.1%, respectively) implants. However, this result was not reflected in the pullout strength, in which plasma-sprayed and HA implants exhibited similar values. The authors attributed this phenomenon to the weak coating-substrate interfacial strength.[115]

HA plasma spraying can present problems, including variation in bond strength at the coating-metal interface, nonuniformity in coating density, and structural and chemical alterations of the coatings as a result of the process.[116] To solve the problems regarding the quality of coating, different techniques have been proposed, such as heat treatment[117] or the technique of ion beam–assisted deposition (IBAD).[118,119]

In a comparison of HA-coated implants using the IBAD process (R_a = 1.04 µm), implants blasted with Al_2O_3 particles (50 µm) (R_a = 1.13 µm), and turned surfaces (R_a = 1.13 µm), dental implants were inserted in the tibiae of rabbits. Twelve weeks later, BIC values were significantly higher in IBAD surfaces (62.5%) than in blasted (54.2%) and machined (38.2%) surfaces. The removal torque values of IBAD (48.5 N-cm) and blasted (47.3 N-cm) surfaces were statistically higher than machined (32.3 N-cm) surfaces. The authors mentioned that advantages of the HA-deposited implants in the early healing phase could be apparent, and the separation or fracture of the coating layer could be prevented. However, the control of resorption needs

WWW.HIGHDENT.IR

to be further investigated.[118] The best form of HA of HA coatings to obtain more favorable results using this technology is under evaluation.[120]

ZIRCONIA

Zirconia (ZrO_2) is a ceramic material used in implantology because of its biocompatibility, esthetics (because its color is similar to the teeth), and mechanical properties, which are better than alumina.[121] Implants produced with ZrO_2 are biocompatible, bioinert, and radiopaque, and they present a high resistance to corrosion, flexion, and fracture.[122-127]

This material is reported to present a contact with bone and soft tissue similar to that observed in titanium implants,[56,128] and it can be used to produce a entire implant or as a coating. The interface is composed by a proteoglycan layer, which is thicker than titanium (ranging from 300 to 500 Å and 200 to 400 Å, respectively).[129,130] However, the amount of bone formed 1 and 6 months after implant placement (in rabbits) did not differ in titanium and zirconium implants.[130]

Bone response to ZrO_2 implants was evaluated in a rabbit study. Four weeks after implantation, the BIC value was 68.4%, and the authors reported an absence of epithelial down growth, foreign bone reaction, gaps, or fibrous tissue between bone and implant.[55] The stability of osseointegration around ZrO_2 implants was also evaluated under different loading protocols in a monkey model. Dental implants were inserted, and 3 months later, prostheses were installed (single freestanding implant support, connected freestanding implant support, and a combination of implant and tooth support). Peri-implant tissues were observed by clinical, histologic, and histomorphometric examination 12 and 24 months after loading, and statistically significant differences were observed among groups (BIC values ranged from 66% to 81%).[131]

In humans, Kohal and Klaus[132] presented a case report in which a ZrO_2 implant was immediately inserted in substitution to a central incisor extracted because of a longitudinal fracture. After a 6-month healing period, a ZrO_2 abutment was cemented and the extracted tooth was modified and served as provisional restoration. One month later, a ZrO_2 single-crown framework was cemented onto the implant. The results obtained with ZrO_2 implants seem promising, and further studies are necessary to clarify the biological response of ZrO_2 implants, especially after longer time periods.

SUMMARY

An enormous quantity of studies is being published to investigate the viability of modified surfaces. Some results are promising; for example, the use of titanium nitride (TiN) surface, nanostructured surfaces, surfaces modified by laser, and the improvement of ceramic materials.[55,133] Nanostructured surfaces, with typical features in the range of 1 to 100 nm, may affect the early events of tissue interaction of the surface with the environment.[134,135] An in vitro study demonstrated that this modification of the surface supported a significantly larger extension of the fibrin clot formation when compared with two control surfaces.[136] In dogs it was shown that a higher percentage of new bone was formed on this type of surface compared with plasma-sprayed and turned surfaces.[137] Finally, in retrieved human implants, observed BIC values varying from 55% to 96%, 6 to 8 weeks after implantation of nanostructured implants, were observed.[138]

Covering titanium implants with TiN was proposed to obtain a surface less susceptible to the release of ions. This is possibly the result of a "physical vapor deposition" that produces a thin TiN layer (about 1 µm), which has a similar histological response to titanium. This layer is responsible for an increase in the corrosion resistance, reduces the bacterial adhesion, and confers a golden aspect to the implant, which can be useful in esthetic zones because the color of these implants is more easily covered by the peri-implant soft tissues.[139-144]

Laser ablation is a technique that can be used to produce a surface with predetermined reproducible characteristics. Implants were modified to produce a controlled, micron-sized surface, with topographical features on the flanks of the threads. These implants demonstrated a significantly greater BIC and greater peak removal torque values compared with a turned surface (BIC values of 40% and 32%, respectively).[145,146] Further studies are necessary to evaluate the responses to this treatment.

$CaPO_4$ coating is also being widely studied because of its chemical bonding property, similar to HA, which seems to result in an improvement in bone response during the healing phase.[147] Biphasic $CaPO_4$ and tricalcium phosphate (TCP) have been studied to evaluate their use in implant coating[148-150]; however, the optimal coating is not yet reported.[151,152]

It is important for the clinician using implants on patients that many studies investigating the biological responses to different surface topographies are performed under in vitro conditions. Although this kind of study is consistently important to clarify the mechanism of interaction between a surface and specific type of cells, the clinical relevance of these results is believed to be low, and the development of long-term clinical evaluations is fundamental. Ideally, those experiments should be designed in humans as randomized controlled trials (e.g., if each hemiarch of one patient is randomly designed to allocate an implant of a specific surface).[153] Different implant topographies seem to influence the outcome of dental implants, but the magnitude and clinical relevance of this influence are

WWW.HIGHDENT.IR

still being investigated. Based on randomized controlled trials available in current literature, Esposito et al.[154] concluded no evidence shows that any particular type of dental implant has superior long-term success. However, the authors report that those findings were based on "a few studies, often at high risk of bias, with few participants and relatively short follow-up periods" and suggest that "more randomized controlled trials should be conducted, with follow-up of at least 5 years including a sufficient number of patients to detect a true difference if any exists." Thus clinicians should consider that, even if several new surface treatments have been proposed, only long-term results can be helpful to evaluate the real biological response of each given surface on the market.

References

1. Steigenga J, Al-Shammari K, Misch C et al: Effects of implant thread geometry on percentage of osseointegration and resistance to reverse torque in the tibia of rabbits, *J Periodontol* 75:1233-1241, 2004.
2. Hermann JS, Cochran DL, Nummikoski PV et al: Crestal bone changes around titanium implants: a radiographic evaluation of unloaded nonsurbmerged and submerged implants in the canine mandible, *J Periodontol* 68:1117-1130, 1997.
3. Brånemark PI, Adell R, Breine U et al: Intra-osseous anchorage of dental prostheses. I. Experimental studies, *Scand J Plast Reconstr Surg* 3:81-100, 1969.
4. Schwartz-Arad D, Herzberg R, Levin L: Evaluation of long-term implant success, *J Periodontol* 76:1623-1628, 2005.
5. Davies JE: Understanding peri-implant endosseous healing, *J Dent Educ* 67:932-949, 2003.
6. Albrektsson T, Brånemark P-I, Hansson HA et al: Osseointegrated titanium implants, *Acta Orthop Scand* 52:155-170, 1981.
7. Albrektsson T, Johansson C: Quantified bone tissue reactions to various metallic materials with reference to the so-called osseointegration concept. In Davies JE, editor: *The bone-biomaterial interface*, Toronto, 2003, University of Toronto Press.
8. Klokkevold PR, Nishimura RD, Adachi M et al: Osseointegration enhanced by chemical etching of the titanium surface. A torque removal study in the rabbit, *Clin Oral Implants Res* 8:442-447, 1997.
9. Wennerberg A, Albrektsson T: Suggested guidelines for the topographic evaluation of implant surfaces, *Int J Oral Maxillofac Implants* 15:331-344, 2000.
10. Jalbout Z, Tabourian G, editors: *Glossary of implant dentistry*, Upper Montclair, NJ, 2004, ICOI.
11. Lautenschlager EP, Monaghan P: Titanium and titanium alloys as dental materials, *Int Dent J* 43:245-253, 1993.
12. Scarano A, Piattelli M: Superfici implantari. In Novello G: *Implantologia pratica*, Cordenons, Italy, 2005, New Service International.
13. Lincks J, Boyan BD, Blachard CR et al: Response of MG63 osteoblast-like cells to titanium and titanium alloy is dependent on surface roughness and composition, *Biomaterials* 19:2219-2232, 1998.
14. Eisenbarth P, Meyle J, Nachtigall W et al: Influence of the surface structure of titanium materials on the adhesion of fibroblasts, *Biomaterials* 17:1399-1403, 1996.
15. Thompson GJ, Puleo DA: Ti-6Al-4V ion solution inhibition of osteogenic cell phenotype as a function of differentiation time course in vitro, *Biomaterials* 17:1949-1954, 1996.
16. Rosa AL, Beloti MM: Rat bone marrow cell response to titanium and titanium alloy with different surface roughness, *Clin Oral Implants Res* 14:43-48, 2003.
17. Han C-H, Johansson CB, Wennerberg A et al: Quantitative and qualitative investigations of surface enlarged titanium and titanium alloy implants, *Clin Oral Implants Res* 9:1-10, 1988.
18. Johansson CB, Han CH, Wennerberg A et al: A quantitative comparison of machined commercially pure titanium and titanium-aluminum-vanadium implants in rabbit bone, *Int J Oral Maxillofac Implants* 13:315-321, 1998.
19. Carr AB, Larsen PE, Gerard DA: Histomorphometric comparison of implant anchorage for two types of dental implants after 3 and 6 months' healing in baboon jaws, *J Prosthet Dent* 85:276-280, 2001.
20. Lausmaa J, Mattson L, Rolander U et al: Chemical composition and morphology of titanium surface oxides, *Mater Res Soc Symp Proc Mater Res Soc* 55:351-359, 1986.
21. Arys A, Philippart C, Dourov N et al: Analysis of titanium dental implants after failure of osseointegration: combined histological, electron microscopy, and x-ray photoelectron spectroscopy approach, *J Biomed Mater Res* 43:300-312, 1998.
22. Zitter H, Plenk H Jr: The electrochemical behavior of metallic implant materials as an indicator of their biocompatibility, *J Biomed Mater Res* 21:881-896, 1987.
23. Ducheyne P: Titanium and calcium phosphate ceramic dental implants, surfaces, coatings and interfaces, *J Oral Implantol* 14:325-340, 1988.
24. Larsson C, Thomsen P, Aronsson BO et al: Bone response to surface-modified titanium implants: studies on the early tissue response to machined and electropolished implants with different oxide thicknesses, *Biomaterials* 17:605-610, 1996.
25. Olefjord I, Hansson S: Surface analysis of four dental implant systems, *Int J Oral Maxillofac Implants* 8:32-40, 1993.
26. Lim YJ, Oshida Y, Andres CJ et al: Surface characterizations of variously treated titanium materials, *Int J Oral Maxillofac Implants* 16:333-342, 2001.
27. Sul YT, Johansson CB, Petronis S et al: Characteristics of the surface oxides on turned and electrochemically oxidized pure titanium implants up to dielectric breakdown: the oxide thickness, micropore configurations, surface roughness, crystal structure and chemical composition, *Biomaterials* 23:491-501, 2002.
28. Sul YT, Johansson CB, Roser K et al: Qualitative and quantitative observations of bone tissue reactions to anodized implants, *Biomaterials* 23:1809-1817, 2002.
29. Kasemo B: Biocompatibility of titanium implants; surface science aspects, *J Prosthet Dent* 49:832-837, 1983.
30. Thomas KA, Kay JF, Cook SD et al: The effect of surface macrotexture and hydroxyapatite coating on the mechanical strengths and histologic profiles of titanium implant materials, *J Biomed Mater Res* 21:1395-1414, 1987.
31. Cheroudi B, Gould TRL, Brunette DM: Effects of grooved titanium-coated implant surface on epithelial cell behavior in vitro and in vivo, *J Biomed Mater Res* 23:1067-1085, 1987.

WWW.HIGHDENT.IR

32. Brunette DM: The effects of implant surface topography on the behavior of cells, *Int J Oral Maxillofac Implants* 3:231-246, 1988.
33. Bowers KT, Keller JC, Randolph BA et al: Optimization of surface micromorphology for enhanced osteoblast responses in vitro, *Int J Oral Maxillofac Implants* 7:302-310, 1992.
34. Boyan BD, Batzer R, Kieswetter K et al: Titanium surface roughness alters responsiveness of MG63 osteoblastic-like cells to 1a,25-(OH)2D3, *J Biomed Mater Res* 39:77-85, 1988.
35. Kieswetter K, Schwartz Z, Hummert TW et al: Surface roughness modulates the local production of growth factors and cytokines by osteoblast-like MG63 cells, *J Biomed Mater Res* 32:55-63, 1996.
36. Doillon CJ, Silver FH, Berg RA: Fibroblast growth on a porous collagen sponge containing hyaluronic acid and fibronectin, *Biomaterials* 8:195-200, 1987.
37. Martin JY, Schwartz Z, Hummert TW et al: Effect of titanium surface roughness on proliferation, differentiation, and protein synthesis of human osteoblast-like cells (MG63), *J Biomed Mater Res* 29:389-401, 1995.
38. Rupp F, Scheideler L, Olshanska N et al: Enhancing surface free energy and hydrophilicity through chemical modification of microstructured titanium implant surfaces, *J Biomed Mater Res A* 76:323-334, 2006.
39. Wennerberg A, Albrektsson T, Andersson B et al: A histomorphometric and removal torque study of screw-shaped titanium implants with three different surface topographies, *Clin Oral Implants Res* 6:24-30, 1995.
40. Wennerberg A, Albrektson T, Johansson C et al: Experimental study of turned and grit-blasted screw-shaped implants with special emphasis on effects of blasting material and surface topography, *Biomaterials* 17:15-22, 1996.
41. Szmukler-Moncler S, Testori T, Bernard JP: Etched implants: a comparative surface analysis of four implant systems, *J Biomed Mater Res B Appl Biomater* 69:46-57, 2004.
42. Schwartz Z, Martin JY Dean DD et al: Effect of titanium surface roughness on chondrocyte proliferation, matrix production, and differentiation depends on the state of cell maturation, *J Biomed Mater Res* 30:145-155, 1996.
43. Abron A, Hopfensperger M, Thompson J et al: Evaluation of a predictive model for implant surface topography effects on early osseointegration in the rat tibia model, *J Prosthet Dent* 85:40-46, 2001.
44. Blumenthal NC, Cosma V: Inhibition of apatite formation by titanium and vanadium ions, *J Biomed Mater Res B Appl Biomater* 23:13-22, 1989.
45. Piattelli A, Manzon L, Scarano A et al: Histologic and histomorphometric analysis of the bone response to machined and sandblasted titanium implants: an experimental study in rabbits, *Int J Oral Maxillofac Implants* 13:805-810, 1998.
46. Wennerberg A, Hallgren C, Johansson C et al: A histomorphometric evaluation of screw-shaped implants each prepared with two surface roughnesses, *Clin Oral Implants Res* 9:11-19, 1998.
47. Wennerberg A, Albrektson T, Andersson B: Bone tissue response to commercially pure titanium implants blasted with fine and coarse particles of aluminum oxide, *Int J Oral Maxillofac Implants* 11:38-45, 1996.
48. Savarino L, Cenni E, Stea S et al: X-ray diffraction of newly formed bone close to alumina or hydroxyapatite-coated femoral stem, *Biomaterials* 14:900-905, 1993.
49. Toni A, Lewis CG, Sudanese A et al: Bone demineralization induced by cementless alumina-coated femoral stems, *J Arthroplasty* 9:435-444, 1994.
50. Darwell BW, Samman N, Luk WK et al: Contamination of titanium castings by aluminum oxide blasting, *J Dent* 23:319-322, 1995.
51. Piattelli A, Degidi M, Paolantonio M et al: Residual aluminum oxide on the surface of titanium implants has no effect on osseointegration, *Biomaterials* 24:4081-4099, 2003.
52. Gotfredsen K, Nimb L, Hjorting-Hansen E et al: Histomorphometric and removal torque analysis for TiO2-blasted titanium implants. An experimental study on dogs, *Clin Oral Implants Res* 3:77-84, 1992.
53. Ivanoff CJ, Hallgren C, Widmark G et al: Histologic evaluation of the bone integration of TiO(2) blasted and turned titanium microimplants in humans, *Clin Oral Implants Res* 12:128-134, 2001.
54. Van Steenberghe D, De Mars G, Quirynen M et al: A prospective split-mouth comparative study of two screw-shaped self-tapping pure titanium implant systems, *Clin Oral Implant Res* 11:202-209, 2000.
55. Sennerby L, Dasmah A, Larsson B et al: Bone tissue responses to surface-modified zirconia implants: a histomorphometric and removal torque study in the rabbit, *Clin Implant Dent Relat Res* 7(suppl 1):S13-S20, 2005.
56. Kohal RJ, Weng D, Bachle M et al: Loaded custom-made zirconia and titanium implants show similar osseointegration: an animal experiment, *J Periodontol* 75:1262-1268, 2004.
57. Hahn H, Palich W: Preliminary evaluation of porous meta-surfaced titanium for orthopedic implants, *J Biomed Mater Res* 45:71-77, 1970.
58. Schroeder A, van der Zypen E, Stich H et al: The reactions of bone, connective tissue, and epithelium to endosteal implants with titanium-sprayed surfaces, *J Maxillofac Surg* 9:15-25, 1981.
59. Sammons RL, Lumbikanonda N, Gross M et al: Comparison of osteoblast spreading on microstructured dental implant surfaces and cell behaviour in an explant model of osseointegration: a scanning electron microscopic study, *Clin Oral Implants Res* 16:657-666, 2005.
60. Xue W, Liu X, Zheng X et al: In vivo evaluation of plasma-sprayed titanium coating after alkali modification, *Biomaterials* 26:3029-3037, 2005.
61. Carr AB, Gerard DA, Larsen PE: Histomorphometric analysis of implant anchorage for 3 types of dental implants following 6 months of healing in baboons, *Int J Oral Maxillofac Implants* 15:785-791, 2000.
62. Piattelli A, Scarano A, Corigliano M et al: Effects of alkaline phosphatase on bone healing around plasma-sprayed titanium implants: a pilot study in rabbits, *Biomaterials* 17:1443-1449, 1996.
63. Piattelli A, Corigliano M, Scarano A et al: Immediate loading of titanium plasma-sprayed implants: an histologic analysis in monkeys, *J Periodontol* 69:321-327, 1998.
64. Scarano A, Iezzi G, Petrone G et al: Immediate postextraction implants: a histologic and histometric analysis in monkeys, *J Oral Implantol* 26:163-169, 2000.
65. Piattelli A, Corigliano M, Scarano A: Microscopical observations of the osseous responses in early loaded human titanium implants: a report of two cases, *Biomaterials* 17:1333-1337, 1996.

WWW.HIGHDENT.IR

66. Piattelli A, Scarano A, Piattelli M et al: Histologic aspects of the bone and soft tissues surrounding three titanium non-submerged plasma-sprayed implants retrieved at autopsy: a case report, *J Periodontol* 68:694-700, 1997.
67. Piattelli A, Paolantonio M, Corigliano M et al: Immediate loading of titanium plasma-sprayed screw-shaped implants in man: a clinical and histological report of two cases, *J Periodontol* 68:591-597, 1997.
68. Piattelli A, Scarano A, Dalla Nora A et al: Microscopical features in retrieved human Brånemark implants: a report of 19 cases, *Biomaterials* 19:643-649, 1998.
69. Piattelli A, Corigliano M, Scarano A et al: Bone reactions to early occlusal loading of two-stage titanium plasma-sprayed implants: a pilot study in monkey, *Int J Periodontics Restorative Dent* 17:162-169, 1997.
70. Franchi M, Bacchelli B, Martini D et al: Early detachment of titanium particles from various different surfaces of endosseous dental implants, *Biomaterials* 25:2239-2246, 2004.
71. Buser D, Schenk R, Steinemann S et al: Influence of surface characteristics on bone integration to titanium implants: a histomorphometric study in miniature pigs, *J Biomed Mater Res* 25:889-902, 1991.
72. Weingart D, Steinemann S, Schilli W et al: Titanium deposition in regional lymph nodes after insertion of titanium screw implants in maxillofacial region, *Int J Oral Maxillofac Surg* 23:450-452, 1994.
73. Klokkevold PR, Johnson P, Dadgostari S et al: Early endosseous integration enhanced by dual acid etching of titanium: a torque removal study in the rabbit, *Clin Oral Implants Res* 12:350-357, 2001.
74. Park JY, Gemmell CH, Davies JE: Platelet interactions with titanium: modulation of platelet activity by surface topography, *Biomaterials* 22:2671-2682, 2001.
75. Ogawa T, Nishimura I: Different bone integration profiles of turned and acid-etched implants associated with modulated expression of extracellular matrix genes, *Int J Oral Maxillofac Implants* 18:200-210, 2003.
76. Weng D, Hoffmeyer M, Hurzeler MB et al: Osseotite vs. machined surface in poor bone quality: a study in dogs, *Clin Oral Implants Res* 14:703-708, 2003.
77. Degidi M, Petrone G, Iezzi G et al: Bone contact around acid-etched implants: a histological and histomorphometrical evaluation of two human-retrieved implants, *J Oral Implantol* 29:13-18, 2003.
78. Testori T, Szmukler-Moncler S, Francetti L et al: Immediate loading of Osseotite implants: a case report and histologic analysis after 4 months of occlusal loading, *Int J Periodontics Restorative Dent* 21:451-459, 2001.
79. Trisi P, Lazzara R, Rao W et al: Bone-implant contact and bone quality: evaluation of expected and actual bone contact on machined and Osseotite implant surfaces, *Int J Periodontics Restorative Dent* 22:535-545, 2002.
80. Cho SA, Park KT: The removal torque of titanium screw inserted in rabbit tibia treated by dual acid etching, *Biomaterials* 24:3611-3617, 2003.
81. Galli C, Guizzardi S, Passeri G et al: Comparison of human mandibular osteoblasts grown on two commercially available titanium implant surfaces, *J Periodontol* 76: 364-372, 2005.
82. Buser D, Nydegger T, Hirt HP et al: Removal torque values of titanium implants in the maxillae of miniature pigs, *Int J Oral Maxillofac Implants* 13:611-619, 1998.
83. Abrahamsson I, Berglundh T, Linder E et al: Early bone formation adjacent to rough and turned endosseous implant surfaces: an experimental study in the dog, *Clin Oral Implants Res* 15:381-392, 2004.
84. Cochran DL, Schenk RK, Lussi A et al: Bone response to unloaded and loaded titanium implants with a sandblasted and acid-etched surface: a histometric study in the canine mandible, *J Biomed Mater Res* 40:1-11, 1998.
85. Hayakawa T, Kiba H, Yasuda S et al: A histologic and histomorphometric evaluation of two types of retrieved human titanium implants, *Int J Periodontics Restorative Dent* 22:164-171, 2002.
86. Buser D, Nydegger T, Oxland T et al: Interface shear strength of titanium implants with a sandblasted and acid-etched surface: a biomechanical study in the maxilla of miniature pigs, *J Biomed Mater Res* 45:75-83, 1999.
87. Sakakura CE, Nociti FH Jr, Mello GP et al: Histomorphometric evaluation of a threaded, sandblasted, acid-etched implant retrieved from a human lower jaw: a case report, *Implant Dent* 14:289-293, 2005.
88. Cochran DL, Buser D, ten Bruggenkate CM et al: The use of reduced healing times on ITI implants with a sandblasted and acid-etched (SLA) surface: early results from clinical trials on ITI SLA implants, *Clin Oral Implants Res* 13:144-153, 2002.
89. Franchi M, Orsini E, Trire A et al: Osteogenesis and morphology of the peri-implant bone facing dental implants, *Scientific World J* 4:1083-1095, 2004.
90. Guizzardi S, Galli C, Martini D et al: Different titanium surface treatment influences human mandibular osteoblast response, *J Periodontol* 75:273-282, 2004.
91. Sanz A, Oyarzun A, Farias D et al: Experimental study of bone response to new surface treatment of endosseous titanium implants, *Implant Dent* 10:126-131, 2001.
92. Piattelli M, Scarano A, Paoloantonio M et al: Bone response to machined and resorbable blast material titanium implants: an experimental study in rabbits, *J Oral Implantol* 28:2-8, 2002.
93. Gonshor A, Goveia G, Sotirakis E: A prospective, multicenter, 4-year study of the ACE surgical resorbable media implant, *J Oral Implantol* 29:174-180, 2003.
94. Zhu X, Chen J, Scheideler L et al: Effects of topography and composition of titanium surface oxides on osteoblast responses, *Biomaterials* 25:4087-4103, 2004.
95. Son WW, Zhu X, Shin HI et al: In vivo histological response to anodized and anodized/hydrothermally treated titanium implants, *J Biomed Mater Res B Appl Biomater* 66:520-525, 2003.
96. Ivanoff CJ, Widmark G, Johansson C et al: Histologic evaluation of bone response to oxidized and turned titanium micro-implants in human jawbone, *Int J Oral Maxillofac Implants* 18:341-348, 2003.
97. Geesink RG, de Groot K, Klein CP: Bonding of bone to apatite-coated implants, *J Bone Joint Surg Br* 70:17-22, 1988.
98. de Groot K, Geesink R, Klein CP et al: Plasma sprayed coatings of hydroxyapatite, *J Biomed Mater Res* 21: 1375-1381, 1987.
99. Oonishi H, Yamamoto M, Ishimaru H et al: The effect of hydroxyapatite coating on bone growth into porous titanium alloy implants, *J Bone Joint Surg Br* 71:213-216, 1989.
100. Meffert RM: Ceramic-coated implant system, *Adv Dent Res* 13:170-172, 1999.

WWW.HIGHDENT.IR

101. Xie J, Baumann MJ, McCabe LR: Osteoblasts respond to hydroxyapatite surfaces with immediate changes in gene expression, *J Biomed Mater Res A* 71:108-17, 2004.
102. Uehara T, Takaoka K, Ito K: Histological evidence of osseointegration in human retrieved fractured hydroxyapatite-coated screw-type implants: a case report, *Clin Oral Implants Res* 15:540-545, 2004.
103. Morris HF, Ochi S: Hydroxyapatite-coated implants: a case for their use, *J Oral Maxillofac Surg* 56:1303-1311, 1998.
104. Lee JJ, Rouhfar L, Beirne OR: Survival of hydroxyapatite-coated implants: a meta-analytic review, *J Oral Maxillofac Surg* 58:1372-1379, 2000.
105. Schwartz-Arad D, Mardinger O, Levin L et al: Marginal bone loss pattern around hydroxyapatite-coated versus commercially pure titanium implants after up to 12 years of follow-up, *Int J Oral Maxillofac Implants* 20:238-244, 2005.
106. Trisi P, Keith DJ, Rocco S: Human histologic and histomorphometric analyses of hydroxyapatite-coated implants after 10 years of function: a case report, *Int J Oral Maxillofac Implants* 20:124-130, 2005.
107. Collier JP, Surprenant VA, Mayor MB et al: Loss of hydroxyapatite coating on retrieved, total hip components, *J Arthroplasty* 8:389-393, 1993.
108. Rohrer MD, Sobczak RR, Prasad HS et al: Postmortem histologic evaluation of mandibular titanium and maxillary hydroxyapatite-coated implants from 1 patient, *Int J Oral Maxillofac Implants* 14:579-586, 1999.
109. Piattelli A, Cosci F, Scarano A et al: Localized chronic suppurative bone infection as a sequel of peri-implantitis in a hydroxyapatite-coated dental implant, *Biomaterials* 16:917-920, 1995.
110. Jovanovic SA, Kenney EB, Carranza FA Jr et al: The regenerative potential of plaque-induced peri-implant bone defects treated by a submerged membrane technique: an experimental study, *Int J Oral Maxillofac Implants* 8:13-18, 1993.
111. Martins MC, Shibli JA, Abi-Rached RS et al: Progression of experimental chronic peri-implantitis in dogs: clinical and radiographic evaluation, *J Periodontol* 76:1367-1373, 2005.
112. Albrektsson T: Hydroxyapatite-coated implants: a case against their use, *J Oral Maxillofac Surg* 56:1312-1326, 1998.
113. Lu Y-P, Li M-S, Li S-T et al: Plasma-sprayed hydroxyapatite + titania composite bond coat for hydroxyapatite coating on titanium substrate, *Biomaterials* 25:4393-4403, 2004.
114. Sun L, Berndt CC, Gross KA et al: Material fundamentals and clinical perfomance of plasma-sprayed hydroxyapatite coatings: a review, *J Biomed Mater Res* 58:570-592, 2001.
115. Ong JL, Carnes DL, Bessho K: Evaluation of titanium plasma-sprayed and plasma-sprayed hydroxyapatite implants in vivo, *Biomaterials* 25:4601-4606, 2004.
116. Filiaggi MJ, Coombs NA, Pilliar RM: Characterization of the interface in the plasma-sprayed HA coating/Ti-6Al-4V implant system, *J Biomed Mater Res* 25:1211-1230, 1991.
117. Filiaggi MJ, Pilliar RM, Coombs NA: Post-plasma-spraying heat treatment of the HA coating/Ti-6A1-4V implant system, *J Biomed Mater Res* 27:191-198, 1993.
118. Jung YC, Han CH, Lee IS et al: Effects of ion beam-assisted deposition of hydroxyapatite on the osseointegration of endosseous implants in rabbit tibiae, *Int J Oral Maxillofac Implants* 16:809-818, 2001.
119. Lee IS, Kim DH, Kim HE et al: Biological performance of calcium phosphate films formed on commercially pure Ti by electron-beam evaporization, *Biomaterials* 23:609-615, 2002.
120. Park YS, Yi KY, Lee IS et al: The effects of ion beam-assisted deposition of hydroxyapatite on the grit-blasted surface of endosseous implants in rabbit tibiae, *Int J Oral Maxillofac Implants* 20:31-84, 2005.
121. Piconi C, Maccauro G: Zirconia as a ceramic biomaterial, *Biomaterials* 20:1-25, 1999.
122. Ichikawa Y, Akagawa Y, Nikai H et al: Tissue compatibility and stability of a new zirconia ceramic in vivo, *J Prosthet Dent* 68:322-326, 1992.
123. Akagawa Y, Ichikawa Y, Nikai H et al: Interface histology of unloaded and early loaded partially stabilized zirconia endosseous implant in initial bone healing, *J Prosthet Dent* 69:599-604, 1993.
124. Covacci, Bruzzese N, Maccauro G et al: An in vitro evaluation of the mutagenic and carcinogenic power of high purity zirconia ceramic, *Biomaterials* 20:371-376, 1999.
125. Schultze-Mosgau S, Schkiephake H, Radespiel-Troger M et al: Osseointegration of endosseous cones: zirconium oxide vs titanium, *Oral Surg Oral Med Oral Pathol Radiol Endod* 89:91-98, 2000.
126. Josset Y, Oum'Hamed Z, Zarrinpour A et al: In vitro reactions of human osteoblasts in culture with zirconia and alumina ceramics, *J Biomed Mater Res* 47:481-493, 1999.
127. Scarano A, Di Carlo F, Quaranta M et al: Bone response to zirconia ceramic implants: an experimental study in rabbits, *J Oral Implantol* 29:8-12, 2003.
128. Dubruille JH, Viguier E, Le Naour G et al: Evaluation of combinations of titanium, zirconia, and alumina implants with 2 bone fillers in the dog, *Int J Oral Maxillofac Implants* 14:271-277, 1999.
129. Albrektsson T, Hansson HA, Ivarsson B: Interface analysis of titanium and zirconium bone implants, *Biomaterials* 6:97-101, 1985.
130. Thomsen P, Larsson C, Ericson LE et al: Structure of the interface between rabbit cortical bone and implants of gold, zirconium and titanium, *J Mater Sci Mater Med* 8:653-665, 1997.
131. Akagawa Y, Hosokawa R, Sato Y et al: Comparison between freestanding and tooth-connected partially stabilized zirconia implants after two years' function in monkeys: a clinical and histologic study, *J Prosthet Dent* 80:551-558, 1998.
132. Kohal RJ, Klaus G: A zirconia implant-crown system: a case report, *Int J Periodontics Restorative Dent* 24:147-153, 2004.
133. Lee TM, Yang CY, Chang E et al: Comparison of plasma-sprayed hydroxyapatite coatings and zirconium-reinforced hydroxyapatite composite coatings: in vivo study, *J Biomed Mater Res A* 71:652-660, 2004.
134. de Oliveira PT, Nanci A: Nanotexturing of titanium-based surfaces upregulates expression of bone sialoprotein and osteopontin by cultured osteogenic cells, *Biomaterials* 25:403-413, 2004.
135. Webster TJ, Ejiofor JU: Increased osteoblast adhesion on nanophase metals: Ti, Ti6Al4V, and CoCrMo, *Biomaterials* 25:4731-4739, 2004.
136. Di Iorio D, Traini T, Degidi M et al: Quantitative evaluation of the fibrin clot extension on different implant surfaces: an in vitro study, *J Biomed Mater Res B Appl Biomater* 74:636-642, 2005.

WWW.HIGHDENT.IR

137. Papalexiou V, Novaes AB Jr, Grisi MF et al: Influence of implant microstructure on the dynamics of bone healing around immediate implants placed into periodontally infected sites. A confocal laser scanning microscopic study, *Clin Oral Implants Res* 15:44-53, 2004.
138. Iezzi G, Degidi M, Scarano A et al: Bone response to submerged, unloaded implants inserted in poor bone sites: a histological and histomorphometrical study of 8 titanium implants retrieved from man, *J Oral Implantol* 31:225-233, 2005.
139. Wisbery A, Gregson PJ, Tuke M: Application of PVD TiN coating to Co-Cr-Mo based surgical implants, *Biomaterial* 8:477-480, 1987.
140. Scarano A, Piattelli M, Vrespa G et al: Bone healing around and titanium and titanium nitride (TiN) coated dental implants with 3 different surfaces: an experimental study in rats, *Clin Implant Dent Relat Res* 5:103-111, 2003.
141. Scarano A, Piattelli M, Vrespa G et al: Bacterial adhesion on titanium nitride-coated and uncoated implants: an in vivo human study, *J Oral Implantol* 29:80-85, 2003.
142. Grossner-Schreiber B, Griepentrog M, Haustein I et al: Plaque formation on surface modified dental implants. An in vitro study, *Clin Oral Implants Res* 12:543-551, 2001.
143. Groessner-Schreiber B, Neubert A, Muller WD et al: Fibroblast growth on surface-modified dental implants: an in vitro study, *J Biomed Mater Res A* 64:591-599, 2003.
144. Groessner-Schreiber B, Hannig M, Duck A et al: Do different implant surfaces exposed in the oral cavity of humans show different biofilm compositions and activities? *Eur J Oral Sci* 112:516-522, 2004.
145. Hallgren C, Reimers H, Chakarov D et al: An in vivo study of bone response to implants topographically modified by laser micromachining, *Biomaterials* 24:701-710, 2003.
146. Park CY, Kim SG, Kim MD et al: Surface properties of endosseous dental implants after NdYAG and CO2 laser treatment at various energies, *J Oral Maxillofac Surg* 63:1522-1577, 2005.
147. Hayakawa T, Yoshinari M, Kiba H et al: Trabecular bone response to surface roughened and calcium phosphate (Ca-P) coated titanium implants, *Biomaterials* 23:1025-1031, 2003.
148. Citeau A, Guicheux J, Vinatier C et al: In vitro biological effects of titanium rough surface obtained by calcium phosphate grid blasting, *Biomaterials* 26:157-165, 2005.
149. Schopper C, Ziya-Ghazvini F, Goriwoda W et al: HA/TCP compounding of a porous CaP biomaterial improves bone formation and scaffold degradation—a long-term histological study, *J Biomed Mater Res B Appl Biomater* 74:458-467, 2005.
150. Schopper C, Moser D, Goriwoda W et al: The effect of three different calcium phosphate implant coatings on bone deposition and coating resorption: a long-term histological study in sheep, *Clin Oral Implants Res* 16:357-368, 2005.
151. Wen HB, de Wijn JR, Cui FZ et al: Preparation of calcium phosphate coatings on titanium implant materials by simple chemistry, *J Biomed Mater Res* 41:227-236, 1998.
152. Yang Y, Kim K-H, Ong JL: A review on calcium phosphate coatings produced using a sputtering process—an alternative to plasma spraying, *Biomaterials* 26:327-337, 2005.
153. Needleman I, Moles DR, Worthington H: Evidence-based periodontology, systematic reviews and research quality, *Periodontol 2000* 37:12-28, 2005.
154. Esposito M, Coulthard P, Thomsen P et al: Interventions for replacing missing teeth: different types of dental implants, *Cochrane Database Syst Rev* 25:CD003815, 2005.
155. Shalabi MM, Wolke JG, Jansen JA et al: The effects of implant surface roughness and surgical technique on implant fixation in an in vitro model, *Clin Oral Implants Res* 17:172-178, 2006.
156. O'Sullivan D, Sennerby L, Meredith N: Influence of implant taper on the primary and secondary stability of osseointegrated titanium implants, *Clin Oral Implants Res* 15:474-480, 2004.
157. Degidi M, Scarano A, Iezzi G et al: Periimplant bone in immediately loaded titanium implants: histologic and histomorphometric evaluation in human. A report of two cases, *Clin Implant Dent Relat Res* 5:170-175, 2003.
158. Degidi M, Scarano A, Petrone G et al: Histologic analysis of clinically retrieved immediately loaded titanium implants: a report of 11 cases, *Clin Implant Dent Relat Res* 5:89-94, 2003.
159. Jeffcoat MK, McGlumphy EA, Reddy MS et al: A comparison of hydroxyapatite (HA)-coated threaded, HA-coated cylindric, and titanium threaded endosseous dental implants, *Int J Oral Maxillofac Implants* 18:406-410, 2003.
160. Testori T, Szmukler-Moncler S, Francetti L et al: Immediate loading of Osseotite implants: a case report and histologic analysis after 4 months of occlusal loading, *Int J Periodontics Restorative Dent* 21:451-459, 2001.
161. Orsini G, Assenza B, Scarano A et al: Surface analysis of machined versus sandblasted and acid-etched titanium implants, *Int J Oral Maxillofac Implants* 15:779-784, 2000.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 28

Bone Response to Mechanical Loads

Girish Ramaswamy, Martha Warren Bidez, Carl E. Misch

In an effort to optimize the interaction between bone and dental or skeletal implants, many researchers have focused their attention on the implant-tissue interface.[1-19] The importance of the events that take place at the interfacial "zone" between an implant and the host tissue cannot be overstated. This complex interaction involves not only biomaterial and biocompatibility issues but also the alteration of the mechanical environment that occurs when placement of an implant disturbs the normal physiological distribution of forces, fluids, and cell communication. The purpose of this chapter is to describe the current state of understanding regarding the biological and biomechanical response of bone to mechanical loads, with particular emphasis on the dental implant–bone interface.

BIOLOGICAL RESPONSE

The implant-tissue interface is an extremely dynamic region of interaction. This interface completely changes character as it goes from its genesis (placement of the implant into the prepared bony site) to its maturity (healed condition). The biomechanical environment plays an immediate role in the quality and compositional outcome of the new interface. For example, extensive research shows that if the implant is stable in the bone at the time of placement, then the interface is more likely to result in osteointegration.[15,20] Relative movement (or micromotion) between the implant and the bone at the time of placement is more likely to favor the development of a fibro-osseous interface.[11,18] The healing stage of the interface, however, is only the beginning of its dynamic nature. Functional loading of the implant brings additional biomechanical influences that greatly affect the composition of this junction.

A topic of intense research for many years is the transduction of loading-induced strain at the interface into a signal that can direct the interfacial tissues to respond or remodel. It has been proven that bone responds to both hormonal and biomechanical (functional loading) regulation. These two regulating mechanisms are often in opposition to each other. Research has shown that even in instances in which a large demand exists for calcium (the primary objective for hormonal regulation), functional loading can compete and maintain bone mass.[21] Researchers have theorized that the actual strain (see Chapter 23) that is perceived by the bone tissue initiates a chain of events that results in a biological response. For tissue strain to influence bone adaptation at the bone-implant interface, it must elicit some sort of a chemical or biological response in a strain-sensitive population. The current hypothesis is that bone cells in conjunction with the extracellular matrix comprise the strain-sensitive population and that each plays a vital role in the mediation of the interface. Based on this rationale, the objective of a good implant design would be to establish and maintain a strain environment within the host bone tissue and at the interface that favors osteointegration of the implant.

MECHANOTRANSDUCTION

Mechanotransduction is a multistep process that includes (1) mechanocoupling (transduction of mechanical forces into signals sensed by sensor cells), (2) biochemical coupling (conversion of mechanical signal into a biochemical signal to elicit a cellular response such as gene activation), (3) transfer of a signal from sensor to effector cells, and (4) the effector cell response.[22] Recent studies have led to the current consensus that osteocytes embedded within lacunae in bone matrix act as mechanosensors and help translate mechanical loads into biochemical signals.[23-25] Osteocytes are the most abundant cells and inhabit an extensive lacunocanalicular network that enables them to communicate with other osteocytes, as well as with periosteal and endosteal osteoblasts.[24-26] Furthermore, osteocytes have higher sensitivity to mechanical stimulation than osteoblasts.[27-29]

The strains experienced at the tissue level in vivo during normal activities (0.04% to 0.3%) are much less than the strain levels (1% to 10%) required to elicit a cellular response.[30,31] Initially, shear stress caused by

WWW.HIGHDENT.IR

fluid flow in the canalicular spaces was believed to be the primary driver of biochemical coupling in bone, with strain being the dominant stimulus.[23,32] Later, You et al.[33] proposed a model for the amplification of physiologically induced strains to levels that would initiate intracellular biochemical responses. The model suggested that drag forces on the transverse tethering fibers[34,35] during fluid flow through the pericellular space (filled with matrix between osteocyte cell membrane and canalicular wall) will produce a tensile stress, which will in turn create a hoop strain in the intracellular actin cytoskeleton that is two orders of magnitude higher than the strains at the whole-bone level. This model was further modified using updated ultrastructural data on the cytoskeleton, transverse tethering fibers, and their structural rigidity.[36] Cowin[37] compared and reviewed the two models by focusing on the relationship between the bone microstructure and the mechanism by which osteocytes sense the fluid flow as the result of mechanical loading. Possible mechanoreceptors, mechanisms by which osteocytes sense mechanical stimuli, and intracellular signaling pathways are discussed in several research groups.[38-40] Regulation of osteoblastic activity by osteocytes has been proposed to occur through gap junctions,[41-44] with the stimulation of gap junctions mediated by prostaglandin E_2 (PGE_2).[45-49]

Loading of bone decreases osteocyte apoptosis, whereas disuse and supraphysiological strains increases it followed by haversian remodeling.[50-52] Bone disuse, even for short durations, may rapidly induce a hypoxic state of stress in osteocytes, which when extended may lead to apoptosis. This hypoxia can be reversed by short-term physiological loading, which suggests that mechanical loading at such magnitudes plays a key role in osteocyte viability.[53] This may adversely affect bone strength independent of bone loss.[54] Hypoxic osteocytes may also mediate disuse-induced bone resorption by increasing osteopontin expression.[55]

Biomechanically Based Bone-Remodeling Theories

Biomechanically based bone-remodeling theories largely fostered the desire to optimize the effects of strain at the bone-implant interface to encourage osteointegration. In 1887, Meier[56] described the systematic structure of trabecular bone in the femoral head. In 1892, Wolff[57] described these events as a law of nature and stated that the trabecular bone will place or displace itself in relationship to the functional pressures. In 1895, Roux[58] suggested that the tissue changes to loading were a result of a cellular-regulation process. Frost[59] proposed the theory of the mechanostat. He postulated that bone mass is a direct result of the mechanical use of the skeleton. This agrees with Wolff's law[57] that essentially states, "form follows function." Frost established a mechanical-adaptation chart relating trivial loading, physiological loading, overloading, and pathologic loading zones to ranges of microstrain (Figure 28-1). His studies showed that strains in the range of 50 to 1500 microstrain (με) stimulated increases in cortical bone mass until the strains were reduced to the threshold range (or minimum effective strain). This process of the mechanostat would effectively switch the bone modeling *on* and *off*. This phenomenon led him to the flexure drift hypothesis in which he proposed that long bones (e.g., femur) were geometrically curved to minimize the strain distribution down the long axis of the bone.[60] Frost suggested that the curvature of the long bones canceled the bending moment caused by the eccentric pull of the muscles.

Bone may reduce strains by bone apposition or reduction, by bone formation or resorption, and by changing modulus of elasticity or stiffness by changing mineral content.[61-63] Necrosis of bone cells appears to determine the upper equilibrium level. Cell destruction can be observed when stresses exceed 6.9×10 N/mm^2, whereas a stress of 2.48×10 N/mm^2 will cause an increase in bone growth.[64]

Turner et al. [23] and Turner[65] summarized the rules governing bone adaptation as (1) dynamic (not static) loading drives bone adaptation; (2) short-term loading has an anabolic effect, whereas increased duration degrades bone adaptation; and (3) abnormal strains evoke bone adaptation, whereas bone becomes accustomed to routine strains and remodeling ceases.

Dynamic loading has consistently been found to have more osteogenic potential than static loading.[66] Dynamic axial loading for short durations, which produced strains within the physiological range that were added to normal activity, led to adaptive straightening of growing rat

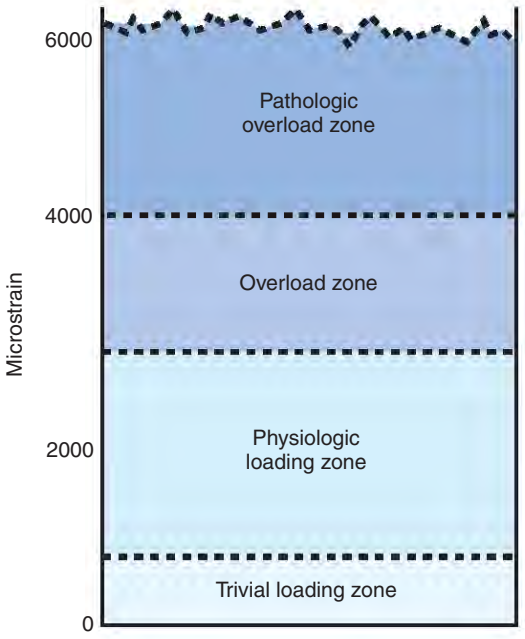


Figure 28-1 Mechanostat chart. *(Adapted from Frost HM: Bone "mass" and the "mechanostat": a proposal,* Anat Rec *219:1-9, 1987.)*

WWW.HIGHDENT.IR

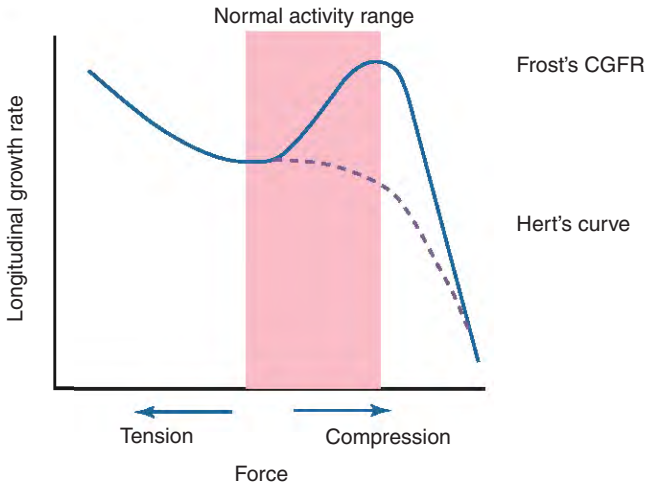


Figure 28-2 Comparison between Frost's chondral growth force response (CGFR) curve and Hert's curve. Shaded region shows the forces experienced during normal activities. *(Adapted from Ohashi N, Robling AG, Burr DB et al: The effects of dynamic axial loading on the rat growth plate,* J Bone Miner Res *17:284-292, 2002.)*

ulnae. Reduced and increased periosteal bone formation were observed at moderate and higher peak strains, respectively.[67] Similar studies observed adaptive osteogenic response to be proportional to the strain rate and local surface strain.[68-69]

Contrary to the previously stated anabolic responses by rat ulna, both static and dynamic axial loading have been found to cause a reduction in longitudinal bone growth.[70,71] Growing male rats[70] receiving 10-minute bouts of static loading at 17 N, static loading at 8.5 N, or dynamic loading at 17 N exhibited shorter bone growth than the controls. The suppression was mainly visible in the hypertrophic zone and was proportional to the load magnitude. A later study[71] investigated the growth plate biology after the application of three different compressive loads (4 N, 8.5 N, and 17 N) for 10 min/day for 8 days on rat ulnae. The longitudinal mineralization rate was completely suppressed and never recovered in the 17-N rats, whereas other groups showed significant suppression that recovered within 1 week after loading. From their results, the authors suggested that even low-magnitude compressive loads suppress growth rate (Figure 28-2), which supported Hert's proposal[72] and deviates from Frost's chondral growth force response curve.[73]

Studies show that adaptive osteogenic response attains saturation after the initial few cycles during continuous cyclic loading, despite further increases in magnitude of load and cycle number.[74] Dynamic loading in short bouts (i.e., with rest periods inserted between loading) have been found to induce an increase in the number and activity of osteoblasts[75,76] and enhance osteogenesis in normal and aged skeletons[76-79] during normal activities such as walking. This consequently improves the biomechanical integrity of the bone despite only slight increments in bone mineral density and content.[80] Rest-

Figure 28-3 Summary of recent studies on anabolic effects of rest insertions between mechanical loading cycles. *(Adapted from Gross TS, Poliachik SL, Ausk BJ et al: Why rest stimulates bone formation: a hypothesis based on complex adaptive phenomenon,* Exerc Sport Sci Rev *32:9-13, 2004.)*

inserted loading decreased the threshold for lamellar bone formation,[81] reduced the number of cycles required to stimulate bone formation, and promoted an increased osteogenic response at any load magnitude (Figure 28-3).[82] Recently, Gross et al.[82] hypothesized that rest periods between each cycle in cyclic loading enhances fluid flow through the canalicular network, thereby extending the communication range of osteocytes by improving transport of signaling molecules between them. Furthermore, rest-inserted loading may also turn on synchronized activity among osteocytes.

Osteogenic response has been found to vary with respect to anatomical site and even at different regions within the same bone. Axial compressive dynamic loading of adult female rat ulnae led to greater periosteal lamellar bone formation distally and lower bone formation proximally when compared with middiaphysis. Strain thresholds and periosteal lamellar bone formation correlated with the peak strains experienced in rat ulnae being higher distally than proximally with intermediate values at the diaphysis.[83] Results from a previous study showed that treadmill exercise increased cancellous bone at the distal tibia more markedly than the proximal tibia, whereas vertebra showed no change.[84] A more recent study in mice revealed that the metaphyseal region in

WWW.HIGHDENT.IR

the distal femur showed an increased osteogenic response as compared with the cortical bone at the middiaphysis.[85] Loading of the knee increased bone formation and mineral apposition on the medial side of the tibial diaphysis compared with the lateral and posterior sides.[86]

Small increases in such mechanical signals to which bones are exposed during regular activities such as standing produce a local, rather than systemic, adaptive anabolic response in cancellous bone without any effect on cortical bone.[87,88] Anabolic effects on trabecular bone were attributed to an increase in bone mineral content and trabecular number, whereas decreases in trabecular spacing indicated the creation of new trabeculae and thickening of existing ones. In addition, researchers observed the adaptation of trabecular bone from rod shaped to plate shaped, primarily in the weight-bearing direction, consequently exhibiting increased strength and stiffness when measured longitudinally.[89] Short bursts of such stimuli inhibit bone resorption by reducing osteoclastic activity and concurrently increase bone formation, maintaining the bone matrix properties.[90] This anabolic response is not controlled by matrix strain magnitude but by applied frequency.[91] Controlled compressive load producing a peak pressure of 1 MPa was applied for 10, 25, or 50 cycles/day at a frequency of 0.5 Hz for 1 month on the distal femoral condyles of rabbits. The loaded limbs showed increased bone volume fraction, trabecular thickness, mean intercept length, and mineral apposition rate when compared with unloaded contralateral limbs.[92] Such low-magnitude, high-frequency mechanical signals (whole-body vibrations) have high potential in the treatment of osteoporosis.[93]

Mechanical stimulation has also been used to accelerate bone formation during fracture and trauma. In vivo dynamic axial compression loading of low magnitude after a short delay increased the strength of fracture callus, whereas immediate loading and shear movement inhibited and delayed the healing process.[94,95] Alternate axial compression and distraction, called *dynamization*, was found to be more effective than either technique applied alone; this combination stimulated callus at both central (influenced by distraction) and peripheral (influenced by compression) regions in a closed transverse fracture in rat tibiae.[96]

Indicators of the Biological Response

The characterization of the biological responses resulting from cellular deformation is equally as diverse as the deformation methodologies. These include changes in the concentration of intracellular mediators and cellular proliferation.

Changes in Concentration of Intracellular Mediators

Numerous investigators have reported fluctuation in the concentrations of intracellular second-messenger molecules.[97-112] In general, cell surface receptors relay information by activating a chain of events that alters the concentration of one or more small intracellular-signaling molecules often referred to as *second messengers* or *intracellular mediators*. In turn, these messenger molecules pass the signal on by altering the behavior of selected cellular proteins. Some of the most widely used intracellular mediators are cyclic AMP (cAMP), Ca^{2+}, and cyclic GMP (cGMP).[113] PGE_2 and prostacyclin are paracrines that are released by osteoblasts in response to mechanical strain.[97,104-108] They are essential for bone formation by mechanical loading[114-119] and are also increased by fluid shear stress[120-125] in a dose-dependent manner. The anabolic effect of mechanical stimulation in vivo has been shown to be greatly depressed by the addition of indomethacin, a chemical that blocks the production of these prostaglandins (PGs).[109] Increase in messenger ribonucleic acid (mRNA) of c-fos and insulin-like growth factor 1 (IGF-1) in osteocytes immediately after mechanical stimulation led to the study involving compressive dynamic loading of the eighth caudal vertebrae in rats. When indomethacin and N^G-monomethyl-L-arginine (L-NMMA), inhibitors of PG and nitric oxide (NO), respectively, were administered individually, c-fos (a marker for mechanical responsiveness in osteocytes) was suppressed partially, whereas combined administration resulted in drastic suppression.[126] This suggested that PG might be produced by NO-dependent and NO-independent mechanisms. Rats injected with NO donors showed increased osteogenic response only when loaded, which suggested that NO requires other molecules such as PG induced by mechanical loading for bone.[117] Human bone cells from osteoporotic patients subjected to pulsating fluid flow showed reduced long-term release of PGE_2, suggesting that long-term adaptive response of these bone cells to mechanical stimuli may have been affected.[127]

Harrell and Binderman[99] observed that isolated osteoblasts, grown on a polystyrene plate that had an orthodontic jackscrew glued to its bottom, responded to continuous strain by increasing PGE_2 concentrations followed in minutes by an increase in cAMP release. Rodan et al.[100] agreed that mechanical strain affected the second-messenger cAMP and also reported changes in cGMP and calcium ions. Yeh and Rodan[97] suggested that PGs might be involved in the transduction of mechanical strain but did not apply physiological levels of strain to their samples. Fluid shear experiments by Reich and Frangos[101] and cyclic biaxial strain studies by Brighton et al.[98] have demonstrated that osteoblasts respond with an increase in cellular levels of inositol triphosphate.

Osteoblasts form bone by secreting many extracellular matrix proteins, including type I collagen, osteopontin, osteocalcin, osteonectin, biglycan, and decorin. Osteopontin was first purified from rat bone matrix and is considered to play an important role in the

WWW.HIGHDENT.IR

cascade of events required for the formation of bone matrix.[110] In vitro studies have revealed osteoblasts to be more responsive to fluid forces than mechanical strain, associating increased osteopontin expression with increases in force magnitude without any dependence on strain magnitude or rate.[128] Recently, experiments on the femoral epiphyses of rabbits have shown that cyclic loading can influence endochondral bone formation by accelerating formation of secondary ossification centers, increase expression of RUNX2 (an important transcription factor of osteoblasts) and extracellular proteins including osteopontin, type X collagen, and decorin.[129] Osteocalcin, also known as *bone Gla protein*, is widely used as a marker for bone metabolism. Studies have shown that the production of osteocalcin can be stimulated by mechanical stress both in vivo[112] and in vitro.[113] Experiments on bone marrow stromal cells have revealed that shear caused by fluid flow enhances maturation of osteoblasts by stimulating the expression of osteocalcin,[130] osteopontin, and bone sialoprotein[131] but not proliferation of stromal cells.

Parathyroid hormone (PTH) has been found to play a vital role in bone adaptation to mechanical stimuli. Rats with thyroparathyroidectomy did not show any osteogenic response caused by mechanical loading of vertebrae,[132] but the response could be restored by a single PTH injection before loading. However, the restoration did not occur when PTH was injected 3 days after mechanical stimulation. Expression of c-fos was observed only in loaded rats injected with PTH,[132] further highlighting the importance of PTH in mechanical adaptation of bone. In vitro studies on mouse osteoblasts provided further insights into the interactive effects of PTH and pulsating fluid flow on PGE_2 and NO production. Although fluid flow stimulated a twofold rise in PGE_2 and NO production, PTH induced a similar effect on PGE_2 but reduced NO production by degrading the enzyme activity of NO synthase. When applied together, the stimulatory effects of fluid flow were nullified. According to the authors, the results suggested that PTH enhances NO-independent PGE_2 production but inhibits stress-induced NO production by degrading NO synthase, in turn reducing NO-dependent PGE_2 production.[133] PTH may also regulate mechanotransduction by influencing the influx of extracellular calcium in hypotonic osteocytes.[134]

Changes in Cellular Proliferation

As previously discussed, the response of osteoblast-like cells to mechanical strain has been shown to be variable. Many studies have reported increases in cell proliferation,[98,102,135-137] total protein production, and DNA synthesis[135,136,138,139] in response to mechanical strain. A review by Burger and Veldhuijzen[31] suggested that at high magnitudes of strain, osteoblasts proliferate and decrease their production of osteoblast phenotypic markers, such as alkaline phosphatase and bone matrix proteins. At lower magnitudes of strain, osteoblasts exhibit a more differentiated state, with an increase in alkaline phosphatase and matrix protein production and a decrease in proliferation. Strains of physiological magnitude (1000 με) applied by cyclic dynamic stretching on human osteoblast cultures increased cell proliferation and osteoblast activities related to matrix production but decreased alkaline phosphatase and osteocalcin release.[140-142] Frequency and cycle number affect proliferation of bone cells and expression of various osteoblast genes in a different manner.[143,144] Applying uniaxial strain at a constant frequency, the cell number increased up to 1800 cycles. At a constant cycle rate, frequency variation produced only slight differences. Frequencies of 1 Hz and 300 cycles were optimum, having the maximum positive effect of cell proliferation.[144]

In addition to experiments correlating increased proliferation with increased or altered strain levels, several investigators have focused their attention on the timing of the proliferative response. Studies conducted by Lanyon[145] have shown that cellular metabolism is activated within the first few minutes of loading. Raab-Cullen et al.[146] investigated the pattern of gene expression in the tibial periosteum shortly after in vivo controlled external load application. They documented that mRNA expression was altered within 2 hours after loading and that the pattern of specific mRNA expression first reflected proliferation and subsequently differentiation. Cyclic equibiaxial stretching[147] of 7-day osteoblast cultures increased apoptosis independent of the strain range (0.4% to 2.5%), whereas more mature cell culture (2 weeks) increased proliferation. This study revealed the importance of the differentiation stage of osteoblasts in their response to mechanical stimulation.

Chondrogenesis at the periosteum of long bones possesses both osteogenic and chondrogenic potential and holds clinical significance in the repair of articular cartilage and in fracture healing.[148-151] Dynamic fluid pressure was found to increase proliferation of periosteal chondrocytes from immature rabbits in vitro.[152] The possible chondrogenic effects stimulated by continuous passive motion of joints after periosteal arthroplasty via dynamic fluid pressure were investigated using periosteal explants suspended in agarose gel. Low-level pressure application increased chondrogenesis and type II collagen in a dose-dependent manner, whereas high-pressure completely inhibited these activities.[153]

Changes in Cellular Morphology and Organization

Ives et al.,[154] using human and bovine endothelial cells, found that the cells responded differently to various types of strain. The cells oriented themselves parallel to the direction of shear strain induced by fluid flow but perpendicular to the axis of mechanical deformation on a cyclically stretched polyurethane membrane. Investigations by Buckley et al.,[136] using osteoblast-like cells stimulated by cyclic mechanical strain, also resulted

WWW.HIGHDENT.IR

in the alignment of the cells perpendicular to the strain vector. This perpendicular alignment was noted at 4 hours after loading and was significant by 12 hours. They suggested that the preferred orientation might have resulted from a mechanical effect on the osteoblast, wherein cell attachments were broken in the maximum strain direction, leaving only those attachments already present in the least strained conformation. A second hypothesis suggested that the cells may have resolved their focal contacts and migrated in an attempt to minimize the strain to which they were subjected.

Another study involving osteoblast-like cells was reported by Carvalho et al.[155] They investigated cytoskeletal organization in mechanically strained alveolar bone cells isolated from the alveolar processes of Sprague-Dawley rats. The earliest change in cytoskeletal organization was noted at 30 minutes after the initiation of strain. They observed that the cells oriented themselves perpendicular to the long axis of the applied mechanical strain.

In vitro studies on osteoblastic and osteocytic cell lines subjected to unidirectional and oscillatory fluid shear stresses showed that stress fibers formed and aligned in osteoblasts within 1 hour of unidirectional stress but were delayed in the latter type of stress. Osteocytes show alignment for unidirectional stress and dendritic morphology for oscillatory stress only after 24 hours.[156]

Altered Expression and Reorganization of Osteoblast Integrins

Although changes in the distribution of the cytoskeleton in mechanically strained cells have been reported, the exact mechanism for the initial detection and transduction of mechanical force into a biological signal has yet to be determined. One possible transduction pathway is the extracellular matrix integrin cytoskeletal axis.[22,155,157] To understand how the cells interact with the extracellular matrix, attention must be given to the nature of the attachment.

Integrins are the primary receptors used by animal cells to attach to the extracellular matrix,[158] and they function as transmembrane linkers that mediate bidirectional interactions between the extracellular matrix and the actin cytoskeleton. Integrins are composed of two noncovalently associated transmembrane glycoprotein subunits called α and β, both of which contribute to the binding of the matrix protein. Electron micrographs of isolated integrins suggest that the molecule has approximately the shape shown in Figure 28-3, with the globular head projecting more than 20 nm from the lipid bilayer. After the binding of a typical integrin to its ligand in the matrix, the cytoplasmic tail of the β chain binds to both talin and α-actinin and thereby initiates the assembly of a complex of intracellular attachment proteins that link the integrin to actin filaments in the cell cortex[37,158] (Figure 28-4). This process is thought to be how local

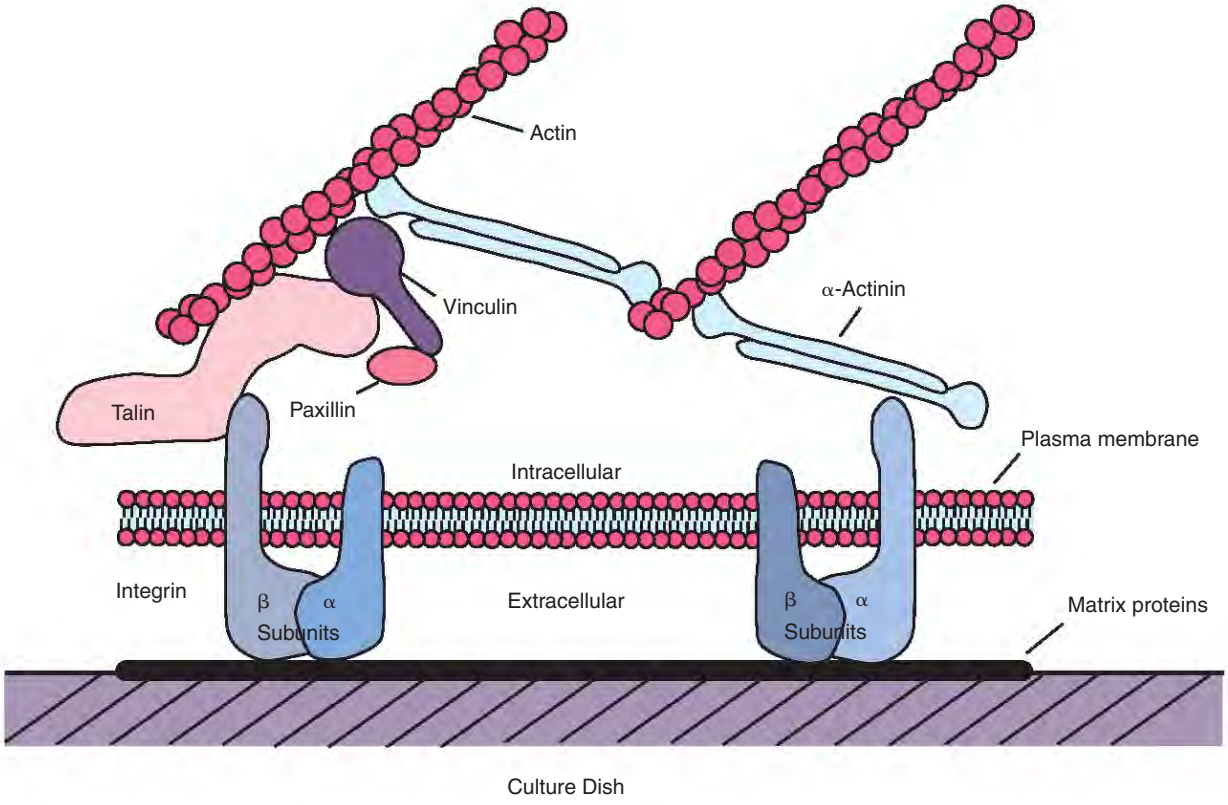


Figure 28-4 Diagram illustrating cytoskeletal components at point of attachment with extracellular matrix in vitro. *(Adapted from Duncan RL, Turner CH: Mechanotransduction and the functional response of bone to mechanical strain,* Calcif Tissue Int *57:344-358, 1995.)*

WWW.HIGHDENT.IR
انجمن دندانسازان و دندانپزشکان

contacts form between cells and the extracellular matrix. If the cytoplasmic tail of the β chain is removed or mutated using recombinant DNA techniques, then the integrins can still bind to the matrix, but the strength of the bond is decreased and the integrins no longer cluster at the focal contacts.[158]

The connection between integrins and the actin cytoskeleton is considered to be a possible pathway for sensing mechanical signals and producing a response in bone.[159-161] The interactions that integrins mediate between the extracellular matrix and the cytoskeleton play an important part in regulating the shape, orientation, and movement of the cells.[162] Schwartz and Ingber[163] suggested a direct link between mechanical strain and cellular response. Integrins of endothelial cells subjected to shear stress were shown to realign with the direction of flow, suggesting that cell adhesion is a dynamic process responding to mechanical strain.[164] Wang et al.[165] demonstrated that a physical strain applied directly to integrins using a magnetic twisting device was shown to be resisted by the cytoskeleton. Pavalko et al.[161] performed in vitro studies on MC3T3-E1 osteoblasts to analyze the role of actin and actin-membrane interactions in altering gene expression because of mechanical loading. Observations of reorganization of actin filaments into contractile stress fibers, formation of focal adhesions, and recruitment of β_1-integrins and α-actinin to focal adhesions revealed a critical role played by actin cytoskeleton in altering gene expression (upregulation of COX-2 and c-fos) in osteoblasts as a response to fluid shear stress. Increase in the number and size of stress fibers and focal adhesion complexes associated with mechanical strain indicated a combined change in both cytoskeleton and extracellular matrix favoring tighter adhesion of osteoblasts to the latter.[160] Both cell adhesion and mechanical stimulation induce expression of integrin-binding proteins—osteopontin, fibronectin, and bone sialoprotein—by osteoblasts but via different mechanisms at different time frames after stimulation. Although strain-induced (dynamics biaxial strain of 1.3% at 0.25 Hz) osteopontin expression was dependent on cytoskeletal integrity, cell adhesion was not.[166-168] These observations indicate that the extracellular matrix integrin cytoskeletal system may be part of the cascade responsible for the transduction of mechanical strain into a biological response.

Other studies have shown that several intracellular signaling pathways are activated coincident with a clustering of integrins at the focal contacts between the cells and the matrix. These clustered integrins may generate intracellular signals by initiating the assembly of a signaling complex just inside the plasma membrane, similar to that of growth factor receptors. Many cells in culture will not respond to growth factors unless the cells are attached via integrins to the extracellular matrix molecules.[158,169] Recent investigations have related the extracellular signal-regulated kinase (ERK) pathway (one of the mitogen-activated protein [MAP] kinases identified) to growth and differentiation of osteoblasts,[170] differentiation of mesenchymal stem cells toward osteogenic lineage,[171] and also mechanotransduction.[172-179] Fluid flow applying physiological strain levels on human osteoblasts rapidly induced ERK phosphorylation and clustering of $\alpha v\beta_3$ integrins in vitro,[176] whereas the mechanism behind the regulation of osteocyte apoptosis by mechanical stimulation involves and requires the activation of an integrin/cytoskeleton/Src/ERK pathway.[179] These results have led researchers to suggest that both mechanical (e.g., fluid flow, cyclic stretching) and chemical (e.g., hormones, growth factors) stimuli may act through the same intracellular signaling pathways.[176,179]

Numerous subunits have been characterized, and different combinations of α and β subunits function as receptors for a variety of extracellular proteins.[180,181] The β_1 integrin subunit is often expressed in bone cells both in vitro and in vivo.[181] Carvalho et al.[155] demonstrated that changes in the organization of the β_1 subunit were induced by the application of strain as early as 4 hours from its onset. They compared the expression of the β_1 integrin subunit mRNA from strained cultures with unstrained controls.

CHANGES IN GENE EXPRESSION

To characterize the biological response of osteoblast-like cells to external mechanical loading, many researchers are investigating strain-induced alterations in patterns of osteoblast gene expression. Several authors have reported that the initial response to strain is a rapid increase in c-fos mRNA expression, indicative of increased proliferation, paired with a rapid decline in levels of mRNA encoding bone matrix proteins, such as type I collagen, osteopontin, and osteocalcin.[146,182] A "rebound" effect or reversal of this trend is usually seen with time as the proliferation tapers off, accompanied by an increase in expression of the matrix proteins.[146,182-184]

The term *matrix proteins* refers to both collagenous and noncollagenous proteins. Type I collagen is the most abundant protein in the organic matrix of bone. This molecule is composed of one α_2 and two α_1 chains. These three chains are initially assembled into a triple helical structure within the cell and are subsequently bundled into fibrils once secreted from the cell. These extracellular fibrils are arranged in a specific, repeating orientation that produces the typical banded appearance common to type I collagen. Intermolecular cross-links stabilize this pattern and produce a porous, repeating, three-dimensional structure.[21] Active osteogenesis involves the expression of genes that result in the production of collagen type I protein.[184] This trait makes the type I collagen molecule a valuable indicator of differentiated osteoblastic activity. Cyclic pressure increases mRNA

WWW.HIGHDENT.IR

expression for type 1 collagen and accumulation of calcium by improving osteoblast function without affecting the cell number.[185] Cyclic stretching of rat calvarial osteoblasts increased collagen production at lower strains (500 με) and inhibited production at higher strain levels (1500 με).[186]

In the last 20 years, noncollagenous proteins have received increased attention. Researchers have suggested that these minor components of organic bone matrix may play a role in regulating bone function, expression, and turnover.

Osteocalcin (bone Gla protein) is a noncollagenous protein that binds calcium and has been isolated from bone, dentin, and other mineralized tissues. It is specifically synthesized by differentiated osteoblasts and, like type I collagen, is an ideal marker for osteoblast phenotypic expression.[187]

Another noncollagenous protein that is generating great interest is osteopontin. This bone sialoprotein is synthesized by primary osteoblasts and has been shown to play a role in cell attachment and spreading. Osteopontin contains a binding sequence that appears to be recognized by an integrin cell surface receptor related to the vitronectin receptor.[187]

Both osteocalcin and osteopontin are regulated by a number of hormones and growth factors. The most common promoter of osteocalcin and osteopontin expression and secretion is 1,25-dihydroxyvitamin D_3 ($1,25[OH]_2D_3$), which directly influences the genes of both proteins. This is possible because the genes for both osteopontin and osteocalcin contain regions that recognize vitamin D.[183] A study by Harter et al.[183] analyzed the expression and production of bone matrix proteins in human osteoblast-like osteosarcoma cells in response to 1 to 4 days of chronic, intermittent, mechanical strain. Northern analysis for type I collagen detected an increase in collagen message after 48 hours of strain. Immunofluorescent labeling of type I collagen indicated that secretion was also enhanced. In the absence of vitamin D, osteopontin message levels were increased severalfold by the application of mechanical load. This increase in osteopontin expression was doubled when the cells were subjected to mechanical load in the presence of vitamin D.

Osteocalcin secretion was also increased with cyclic strain. Osteocalcin levels were not detectable in vitamin D–untreated control cells; however, after 4 days of induced load, significant levels of osteocalcin were observed in the medium. With vitamin D present, osteocalcin levels were four times higher in the medium of strained cells compared with unstrained controls. This study demonstrates that mechanical strain of osteoblast-like cells is sufficient to increase the transcription and secretion of matrix proteins via mechanotransduction without hormonal induction.[183] Osteoblasts in mechanically loaded mouse periodontium showed increased expressions of osteocalcin, type I collagen, and alkaline phosphatase.[188] The first two were more responsive and were found to be stimulated within a short time after loading. The authors suggested that mechanical stimulation drives rapid differentiation of committed osteoblast precursors to produce an anabolic skeletal adaptation.[189] Weight loading of chicks at the prepubertal stage led to development of shorter bones with narrower growth plates but with increased mineralization and vascular penetration. Increased osteopontin and matrix metalloproteinases (MMP9 and MMP13) led to the speculation by the authors that MMPs allowed greater penetration of blood vessels carrying osteoblasts and osteoclasts, whereas osteopontin increased osteoclast numbers, thereby increasing resorption at the growth plate region.[190]

Disruption of genes can alter the normal bone formation response to mechanical stimulation as observed in mice lacking thrombospondin 2[191] that showed contrasting behavior as compared with wild-type mice with increase in endocortical bone formation despite higher strains at the periosteal surface.

Frost[59] has reported that the mechanism for the biomechanical response of osteoblasts is not discrete. Osteoblastic products such as interleukin-1 (IL-1) can stimulate osteoblasts. He groups these cells as basic multicellular units (BMUs). These BMUs are most prevalent on periosteal and endosteal surfaces, and the periosteal BMUs are most sensitive to biomechanical stimuli.

Limitations of Previous Studies

Although these cell culture studies generate promise for the quantitative delineation of the mechanically induced cellular response of bone, the enthusiasm for all of these studies must be tempered in light of the experimental models that were used. Virtually all of these models used some form of polyurethane membrane, collagen ribbon, or silastic plate as the substrate on which the cells were grown and mechanically stimulated. Given the complex host-biomaterial interactions within the human body, the cellular response of isolated bone cells on polyurethane membranes or collagen ribbons may be significantly different from bone cells in intimate contact with a contemporary implant biomaterial, such as titanium or titanium alloy. Investigations are in progress to confirm the effects of mechanical strain on the cells of the bone-implant interface in an experimental system that allows growth of osteoblastic-like cells on the surface of an actual implant material.[192]

Additional limitations can be found in the methodologies mentioned previously. In many of the experiments, the imposed strains were not quantified. Some of the other studies that did report strain magnitudes used levels of strain that were either supraphysiological (>7000 με)[22] or subphysiological (<1500 με)[22] in nature.

WWW.HIGHDENT.IR
مرجع دندانسازان و دندانپزشکان

The experimental techniques that have been discussed so far, such as strain gauges being applied to living bone, organ cultures, and cell culture, can provide illuminating data, but all the techniques have drawbacks and sources of error. Strain gauges are technique sensitive and are difficult to use with biological tissues because of moisture, heat, irregularity of surface, and sometimes poor access to the application site. Their application to animal models can introduce further complications caused by the unpredictability of the behavior of the animal. Movements and loads artificially created when the animal is anesthetized may not give accurate data concerning physiological loads, but the animal may pull wires loose when it is awake.

Organ culture analysis may retain some of the spatial accuracy needed to test strain in the matrix; however, perfusion is necessary to maintain the organ tissue, and not much working time exists before the organ culture dies. Isolated cell culture models can give very useful information related to the release of certain biological mediators in response to the cells' environment. However, again, organization has been lost, and this departure from the in vivo situation must be kept in mind when the results of such experiments are analyzed.

All of these experimental techniques are very valuable despite their individual drawbacks. When they are used in combination and their limitations are understood, helpful insight can be obtained and used to further understand functional loading and its biological ramifications.

BIOMECHANICAL RESPONSE

A compelling argument has been presented for strain-induced biological response of bone to mechanical load. The question remains: What controls the magnitude of strain imparted to the dental implant–bone interface?

Strain has been generically defined (see Chapter 21) in relation to deformation and applied stress. The discussion of strain must be necessarily extended for biological structures. The mechanical properties of the trabecular and cortical bone found within the oral environment exhibit a high degree of variation as a function of load direction, rate, and duration. In addition, the structural density of the bone has a significant influence on its stiffness (modulus of elasticity) and ultimate strength. As such, the mechanical strain exhibited in bone is ultimately a function of the bone density.

Dependence on Direction of Loading

The degree to which the mechanical properties of cortical bone are dependent on its structure is referred to as *anisotropy*. This concept is illustrated in Figure 28-5, which illustrates how a material may exhibit directionally dependent mechanical properties (e.g., modulus of elasticity). A material is said to be *orthotropic* if it exhibits different properties in all three directions and *isotropic* if the properties are the same in all three directions. *Transversely isotropic* describes a material in which two of the three directions exhibit the same mechanical properties.

Reilly and Burstein[193] and Yoon and Katz[194] have reported bone to be transversely isotropic (referring to Figure 28-5, E_1 and E_2 are the same). Knets and Malmeister[195] and Ashman et al.[196] have described bone as *orthotropic* (i.e., $E_1 = E_2 = E_3$). The mandible has been reported as transversely isotropic, with the stiffest direction oriented around the arch of the mandible[197] (see Figure 28-5). These authors suggest that cortical bone of the mandible functions as a long bone that has been molded into a curved-beam geometry. The stiffest direction (around the arch) thus corresponds to the long axis of the tibia or femur.

Early studies on the mandibular and supraorbital bone reported elastic constants of cortical bone in all three orthogonal directions at both locations to be different, suggesting the anisotropy of craniofacial bone. Comparing properties from both locations, mandibular bone along a longitudinal direction was stiffer than bone from a supraorbital region, which may be the result of a difference in function.[198] Cancellous bone in human mandible exhibited transverse isotropy by compression tests and symmetry along inferosuperior directions. Elastic modulus was greatest in the mesiodistal direction (907 MPa), lowest in the inferosuperior direction (114 MPa), and intermediate in the buccolingual direction (511 MPa).[199] Finite element (FE) analysis of mandibular bone around implants indicated an increase in stresses and strains because of anisotropy. A compressive and shear anisotropy of 3% and 1% in cortical bone and 40% and 38% for cancellous bone, respectively, increased stresses by 20% to 30% in the cortical crest. Although tensile and radial-hoop shear stress increased by threefold to fourfold in the cancellous bone along

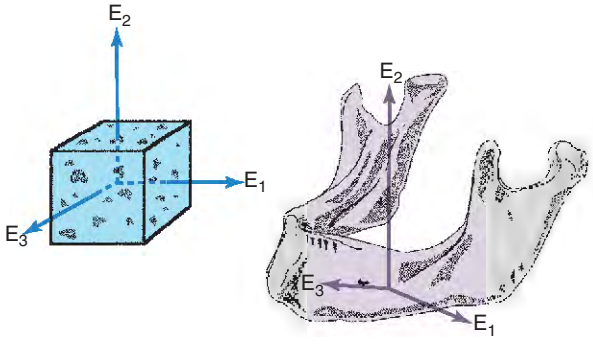


Figure 28-5 Cortical bone of the human mandible has been reported as transversely isotropic, with the stiffest direction oriented around the arch of the mandible (E_3). *(From Ashman RB, Van Buskirk WC: The elastic properties of a human mandible,* Adv Dent Res *1:64-67, 1987.)*

WWW.HIGHDENT.IR

the lingual side, anisotropy decreased radial-vertical interface shear stress by 40% on the buccal side near the apex in cancellous bone.[200]

Such data raise interesting questions regarding the primary loads that the mandible experiences: occlusal loads or flexural loads imposed during opening and closing of the mouth. Clinical experience has qualitatively revealed that the actual mandible has more compact bone at the inferior border, less compact bone on the superior aspect, and greater quality of trabecular bone, especially between the mental foraminae. In addition, the presence of teeth and/or implants significantly increases the trabecular bone amount and density within the residual alveolar bone. Several models have analyzed stress distributions around implants and supporting bone in mandible as an effect of differences in load directions.[201,202] Experiments and models have suggested that off-axial loads produced during occlusal loading produce higher strains in the cervical region and cause significant concern regarding crestal bone loss, cervical tooth loss, and failure of osseointegration.[203-205] Off-axial loads also induce adaptive bone remodeling around oral implants as shown by experimental and FE analysis in dog mandibles.[206,207] The experimental study revealed a significant remodeling difference between axial and nonaxial loading. Although axial loads produced a uniform and mild remodeling response that decreased from the coronal aspect to the apex of the implant, nonaxial loads induced more dynamic remodeling in the surrounding cortical bone and more severely in trabecular bone.[206] The FE analysis (incorporating vertical and horizontal loads and a moment) attributed this difference in response to the horizontal component of the stress experienced by the loads. Horizontal compressive stresses were found to induce more intense remodeling as compared with tensile stresses in the same direction. In addition, stress distributions revealed that stresses decreased from periosteum to endosteum in the cortical bone and then increased along trabecular bone toward the apex of the implant.[207]

Nanoindentation is a new method used to measure the material properties (hardness and indentation modulus) of bone at a microstructural level.[208-210] Cortical bone shows elastic anisotropy at a lamellar level as shown by nanoindentation experiments on human tibial cortical bone.[211] Indentation experiments in 12 different directions in three principal planes for osteonal and interstitial lamellae revealed variation in indentation modulus along different directions in each plane.[211]

At small strains, trabecular bone elicits a nonlinear response that varies with respect to the anatomical site and type of loading. The nonlinearity measured by the reduction in tangent modulus was found to differ based on mode of loading (tension or compression) and was positively correlated with density in tension. Yield strains are higher in compression than tension for trabecular

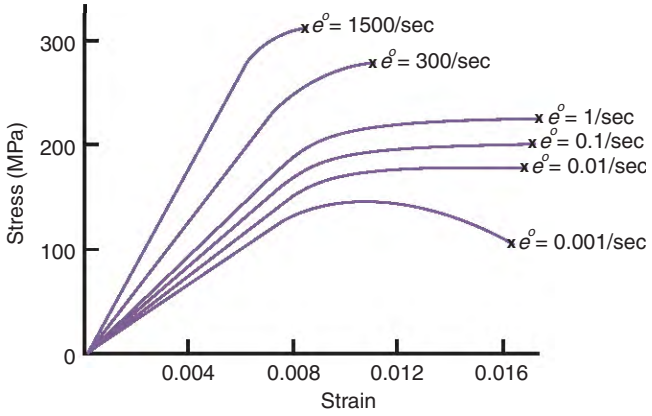


Figure 28-6 Strain rate dependence of bone. *(From McElhaney JH: Dynamic response of bone and muscle tissue,* J Appl Physiol *21:1231, 1966. Used by permission.)*

bone[212,213] in long bones. Microdamage is reported to occur before apparent yield in trabecular bone. Yield is said to occur at 88 to 121 MPa in compression and 35 to 43 MPa in tension at local principal strains of 0.46% to 0.63% for the former and 0.18% to 0.24% in the latter.[213] Comparison of apparent and tissue level yield strains in trabecular bone from femoral neck specimens by FE models revealed that apparent and tissue level yield strains were equivalent in tension but not in compression, and the equivalence was attributed to the highly oriented structure.[214] In compression, yield strains at tissue level were found to be 17% higher than at apparent level. This could lead to residual strains, local tissue yielding, and damage accumulation, degrading the apparent mechanical properties of trabecular bone.[215] Increase in compressive or shear strain could increase the number of microcracks but not the mean length. Any change in the mode of loading can cause cracks to propagate beyond microstructural barriers.[216]

Dependence on Rate of Loading

A material is said to be *viscoelastic* if its mechanical behavior is dependent on the rate of load application. McElhaney[217] investigated the strain rate dependence of bone (graphically illustrated in Figure 28-6). A significant difference can be noted in both ultimate tensile strength and modulus of elasticity over a wide range of strain rates, with bone acting both stiffer and stronger at higher strain rates. Restated, bone fails at a higher load but with less allowable elongation (deformation) at higher as compared with lower strain rates. Thus bone behaves in a more brittle fashion at higher strain rates. Bovine cortical bone has been found to be three to four times more brittle under dynamic load than under a quasistatic load. This brittleness was attributed to a possible shear stress on the fibers in the bone at a high-velocity loading.[218] A similar idea of a change in failure mode because of increasing brittleness at higher strain rates was observed in a study on a galloping horse.[219]

WWW.HIGHDENT.IR
مرجع تخصصی دندانسازان و دندانپزشکان

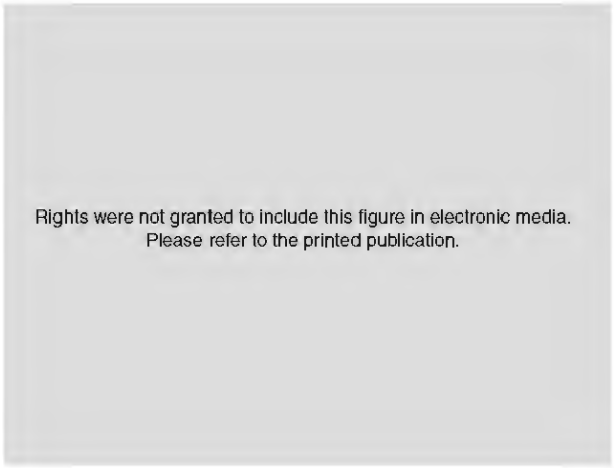


Figure 28-7 Creep curve for adult human cortical bone at constant stress of 60 MPa. *(From Carter DR, Caler WE: Cycle dependent and time dependent bone fracture with repeated loading,* J Biomech Eng *105:166, 1983. Used by permission.)*

Variations in properties such as stiffness, strength, and ultimate strain of human trabecular bone from proximal tibiae[220] and vertebrae[221] were explained using linear and power function relations to strain rate. When compared with compression, strain rate was found to have a higher effect on the change in trabecular bone properties in shear.[222]

Carter and Hayes[223] have reported both strength and elastic modulus of human bone to be proportional to strain rate raised to the 0.06th power. Strain rate to which bone is normally exposed varies from 0.001 sec^{-1} for slow walking to 0.01 sec^{-1} for higher levels of activity. Although closure speeds of the human mouth have been reported by one author,[224] no data are available regarding human mandibular or maxillary bone strain rates in vivo.

At a microstructural level, modulus measured by nanoindentation of human cortical bone (osteons and interstitial bone tissue) was found to increase with increase in loading rates.[225] The difference in modulus at different loading rates was observed to be higher that than predicted by early uniaxial tensile and compression studies at the continuum level.[220,226] However, a previous study investigating the viscoelasticity of cortical bone found modulus to be a function of strain rate raised to the 0.06th power,[227] which compared well with the early macroscopic studies.[226] Similarly, an increase in mechanical properties with increasing strain rates was found in cortical bone from lateral and medial aspects of human femur.[228]

Dependence on Duration of Loading

Carter and Caler[229] have described bone damage or fracture caused by mechanical stress as the sum of both the damage caused by creep or time-dependent loading and cyclic or fatigue loading and the relative interaction of these two types of damage.

Creep refers to the phenomenon whereby a material continues to exhibit increasing deformation as a function of time when subjected to a constant load. Carter and Caler[230] have reported the creep-fracture curve for adult human bone at a constant stress of 60 MPa (Figure 28-7). Over approximately 6 hours, a threefold increase in strain was observed. Such data raise the question of whether resorption and/or failure in the dental bruxer or "clencher" patient may be partially (or wholly) the result of an accumulation of creep damage.

Mixed results have been reported of the effects of creep on fatigue damage of trabecular bone. Although Moore et al.[231] concluded that creep does not contribute to fatigue failure in bovine trabecular bone except for possible effects on low-density osteoporotic bone, recent study on cadaveric vertebrae revealed that trabecular bone does not fully recover from residual strains[232] (time for complete recovery is twentyfold greater than duration of applied loads) caused by creep (static or cyclic loading) and may lead to nontraumatic fractures. Both static and cyclic loading led to similar residual strains of the order of magnitude of initially applied elastic strain. [232]

Fatigue strength of a material refers to an ultimate strength below which the material may be repetitively subjected for an infinite number of cycles without failure. Carter et al.[233,234] have investigated the fatigue properties of human cortical bone. Fatigue failure has been reported for in vivo bone at relatively low cycles (10^4 to 10^8 cycles).[235-237] Given the high magnitude of cycles encountered in oral function, the relatively low in vivo fatigue life reported in bone (i.e., accumulated fatigue damage) is likely accommodated in vivo through the normal process of bone remodeling.

Excessive cyclic loading of bones is known to cause microcrack growth and increase fracture risk.[238,239] Fatigue failure of cortical and trabecular bone has been characterized by a continuous reduction in modulus, with increasing number of cycles with a drastic drop closer to failure and increasing plastic strain. [229,230,240-242] Cortical bone has been observed to behave in an increasingly nonlinear form, with the cyclic energy dissipation increasing with number of cycles in both tensile and compressive cyclic loading.[240,243-245] Thresholds levels of 2500 με and 4000 με in compression were noticed, below which bone exhibited viscoelastic behavior and above which a microdamage accumulation started.[240] Resistance to fracture is a combination of resistance against initiation and propagation of cracks. Discontinuities in osteons and other features such as haversian canals or Volkmann's canals have been suggested to act as microstructural barriers and slow or stop the propagation of cracks.[245,246-252] Osteonal lamellae in cortical bone are hypothesized to play a key role in preventing propagation of smaller cracks, whereas they may be areas of weakness in cases of

WWW.HIGHDENT.IR

longer cracks.[253,254] Cortical bone typically exhibits cyclic softening during cyclic loading, which refers to the nonelastic strain amplitude greater than zero after the initial few cycles, along with a simultaneous rise in stress amplitudes (Figure 28-8). Because of viscoelastic behavior and crack formation, this value stabilizes after the initial few cycles but undergoes a drastic increase before the final failure owing to macroscopic crack growth.[245]

Models relating modulus reduction to microcrack growth have also been proposed.[255-257] Tests have supported a strain-based failure criterion in bovine trabecular bone, with maximum strain attained during the cyclic loading being a better indicator of normalized modulus and linearly related to secant modulus and residual strain.[258] A threshold of –0.5% was observed to attain plasticity in strain, change mechanical properties, and begin the accumulation of microdamage in cyclic and uniaxial compression loading.[259,260]

Rats subjected to external loads by orthodontic tooth movement at different times of the day were found to exhibit increased bone formation on the side experiencing tension and increase in osteoclast formation on the compression side during light periods than dark periods. Inhibition of proliferation and differentiation of chondrocytes by mandibular retractive force was also higher in light periods. Based on these results, the authors suggested that bones and cartilage are more metabolically active during rest periods than during activity; furthermore, orthodontic treatment by force application during rest periods may be more effective than when subjects are active.[261-263]

Dependence on Species and Anatomical Location

Large variations have been noted in experimental measurements of elastic modulus and ultimate compressive strength of trabecular bone. The strength of human mandibular trabecular bone[264] is lower than the proximal femoral trabecular bone reported in previous studies.[265] In the proximal part of the femur, the thickness of the cortical bone wall gradually reduces from the shaft to the metaphyseal region. At the femoral head, the cortical bone represents only a thin shell. Three sets of lamellae arrangement of cancellous bone can be observed in this region, and the cancellous network shows a sheet and strut architecture lined up along the compression and tension lamellae. The trabecular bone in this region is thus the primary structure to dissipate and transfer loads.

Trabecular architecture being either "rodlike" or "platelike"—depending on the anatomical site—could be responsible for the intersite differences.[266] Although vertebrae have the former architecture, femoral neck and proximal tibiae have the latter.[267] Experimental data suggest that rodlike structure is more susceptible to large

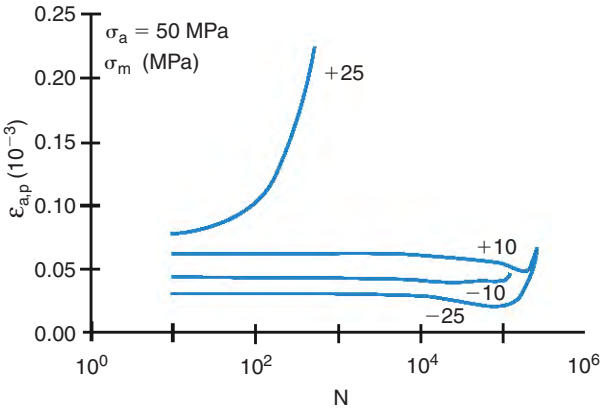


Figure 28-8 Increase in nonelastic strain amplitude with increase in mean stress and cycle number. *(Adapted from Fleck C, Eifler D: Deformation behaviour and damage accumulation of cortical bone specimens from the equine tibia under cyclic loading,* J Biomech *36: 179-189, 2003.)*

deformations by bending and rotation of trabeculae than platelike structures. A comparative study of compressive and tensile yield strains in trabecular bone from vertebra, the proximal tibia, the femoral neck, and the femoral greater trochanter revealed that compressive and tensile yield strains were higher at the femoral neck and vertebra, respectively, whereas yield strains within an anatomical site were found to vary less[266,268] despite huge variations in elastic modulus and yield stress.[266,269-271] Volume fraction (density) and architecture had very little effect in the variations of apparent-level yield strains, which was predominantly influenced by tissue yield strains.[213]

Nonlinearity of trabecular bone, measured as percent reductions in tangent modulus at 0.2% and 0.4% strains, was found to differ at four different sites—vertebra, proximal tibia, and proximal femora from human and bovine proximal tibia. The percent reductions were found to be higher in tension than compression at all sites at 0.4% strain and only in bovine proximal tibiae at 0.2% strain.[272] Occasional overloading of trabecular bone (up to 3% strain) degrades its mechanical properties and increases the risk of fracture.[273] This damage behavior may apply to lumbar and lower thoracic vertebral bodies that are predominantly occupied by trabecular bone and play an important role in causing vertebral fractures.[274] A study of different sites showed femoral neck to possess the highest resistance to fracture initiation for both tension and shear loading in a comparison among femoral neck, femoral shafts, and tibial shafts, whereas femoral shaft possessed the least.[275]

In the edentulous mandible, trabecular bone is continuous with the inner surface of the cortical shell. In the dentate mandible, trabecular bone is surrounded by a thick cortical shell and dense alveolar bone under the teeth. Finite element models of the human mandible[276,277] have shown that cortical bone plays a major role in the dissipation of occlusal loads. Thus load patterns on trabecular bone and microstructure

WWW.HIGHDENT.IR

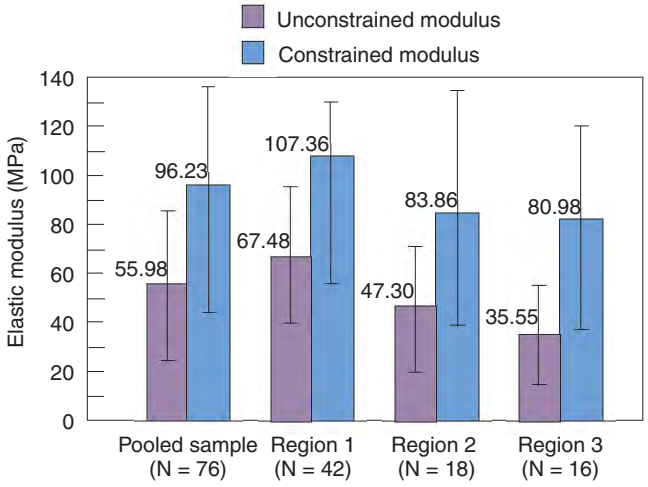


Figure 28-9 Ultimate compressive strength of human mandibular trabecular bone.

of trabecular bone may contribute to differences in the mechanical behavior of the mandible as compared with other anatomical regions. Given that implants do not routinely engage apical cortical bone, attention to trabecular bone mechanical properties is paramount.

Mechanical loads in the mandible are different from those typically experienced by long bones. In the long bones, such as the femur and tibia, loads are primarily axial. In contrast, muscle loads in the mandible may be large and include dorsoventral shear, twisting about the long axis of the mandible, and transverse, increasing in magnitude from posterior to anterior in the mandible.[278] The regional differences observed in the mechanical properties within the human mandible likely reflect the difference in load carried by the different regions of the mandible. With muscle attachments located posteriorly on the mandible, the anterior mandible experiences a large moment load, even in the absence of occlusal loads, caused by the buccolingual flexure of the mandible. Thus significantly higher densities are to be expected in the anterior as compared with posterior mandible. Study on the material properties of human dentate mandible and maxilla revealed regional and directional variations in the properties in both anatomical sites.[279,280] In mandible,[279] direction of maximum stiffness varied at different regions being parallel to occlusal plane at the corpus to vertical orientation at the ramus. Among the corpus, symphysis, and ramus—although symphysis had the thicker cortex, lesser density, anisotropicity, and stiffness—the ramus showed the opposite properties; the material properties of the corpus were between the two others. Such regional variations left questions about relationships between the material properties and mandibular function as suggested by the authors. Edentulation produces a change in all these material properties in the mandible.[281] In the maxilla,[280] alveolar regions were thicker, less dense, and less stiff, whereas cortical bone from the body of the maxilla was thinner, denser, and stiffer. Elastic properties, specifically principal stiffness direction, were more variable in the maxilla than the mandible. Stress and strain distributions along different orientations and between working and balancing sides of the mandible to gain a better understanding of its function augmented the previous studies. The results from the balancing side suggested bending and twisting of the mandible during mastication and transducer biting, whereas the working side was found to undergo torsion. Lingual aspect was stiffer than the buccal side.[282]

Although twofold to threefold higher bite (occlusal) forces are present in the posterior as compared with the anterior mandible, both apparent density and ultimate compressive strength of trabecular bone are lowest in the posterior mandible.[283] These data suggest that the large, multiple-root structure of molar teeth serves to dissipate such posterior occlusal loads as opposed to concomitantly higher ultimate strengths in the bone itself. Current clinical practice routinely places the same-size dental implant diameter and geometry in the posterior and anterior mandible. This practice appears contraindicated given the inherent strength variations within human mandibular bone.

Dependence on Side Constraint

The biomechanical response of trabecular bone in the mandible is highly dependent on the presence or absence of cortical plates as a "side constraint." Qu[264] showed a 65% higher stiffness (elastic modulus) for trabecular bone of the mandible when constrained by cortical plates as compared with unconstrained test values (Figure 28-9). In these tests, fluid was allowed to escape circumferentially so that stiffness trends were lower compared with additional hydrostatic stiffening effects afforded by a constraining test mode. These results are supported by the work of Linde and Hvid,[284] who reported a 19% greater stiffness of trabecular bone specimens (from the proximal tibia) tested when constrained by the surrounding trabecular bone compared with comparable unconstrained tests.

Dental implant patients exhibit variation in the integrity of the buccal and lingual cortical plates. In some instances, one or both plates are completely absent. Treatment planning for such patients should incorporate consideration of the significantly compromised mechanical stiffness (and likely, strength) of the trabecular bone in such anatomical sites.

Dependence on Structural Density

Trabecular bone is a porous, structurally anisotropic, inhomogeneous material. A 25-year literature base documents the work of numerous investigators,[285-296] who have reported in vitro data used in the development of mathematical relationships between elastic modulus and structural density, as well as ultimate strength and

WWW.HIGHDENT.IR

Table 28-1 Relationship between Compressive Strength and Apparent Density of Trabecular Bone in the Human Mandible

REGION	COMPRESSIVE STRENGTH
Pooled sample	$S = 153.4–401.6\rho + 340\rho^2–90.9\rho^3$ ($r = 0.88$, $p<0.0001$)
Region 1	$S = 139.0–366.6\rho + 135.7\rho^2–85.9\rho^3$ ($r = 0.91$, $p<0.0001$)
Region 2	$S = –129.6 + 390.3\rho + 392.7\rho^2$ ($r = 0.90$, $p<0.0001$)
Region 3	No correlation

S, Compressive strength; *r*, relations; *ρ*, density.

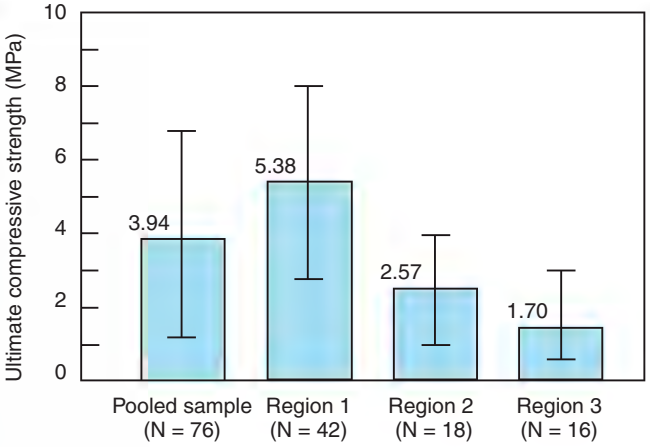


Figure 28-10 Elastic modulus for constrained and unconstrained test conditions in human mandibular trabecular bone.

structural density. Vertebral trabecular bone was found to be highly anisotropic and stiffer in the superoinferior direction,[297] suggesting that trabecular bone should not be considered transversely isotropic. The anisotropy was found to increase with decrease in apparent density to maintain stiffness in the load-bearing direction as suggested by the authors. In addition, structural Young's modulus in all three directions was found to have good correlations explained by power-law models with apparent density. Compressive yield strains of human vertebral trabecular bone depend on apparent density, whereas tensile yield strains do not.[298-300] Buckling of individual trabeculae dominates as the on-axis compressive failure mode at lower densities, whereas axial yielding takes over as the major failure mode at higher densities.[298]

Qu[264] specifically reported on the mechanical properties of mandibular trabecular bone. The study design used cylindric trabecular bone specimens (5 mm in diameter and 5 mm high) from the human mandible. These tests were mechanically tested in compression in the occlusal-apical direction and were performed at a constant strain rate of 0.01 sec^{-1} under both nondestructive and destructive testing conditions. In the nondestructive tests, the trabecular bone specimens were constrained by cortical plates in the buccal and lingual directions and by trabecular bone in the mesial and distal directions. Before the destructive test, the cylindric specimens were measured and weighed to determine the apparent (structural) density.

Regional differences were noted in the human mandibular trabecular bone elastic modulus and ultimate compressive strength, exhibiting up to 47% to 68% higher mean values in the anterior (region 1) compared with the posterior region of the mandible (Table 28-1). No differences were observed in elastic modulus and ultimate compressive strength in the region between the premolars and molars (regions 2 and 3) (Figure 28-10). The compressive strength was correlated at a high level of significance ($r = 0.88$, $p < 0.0001$) with the trabecular apparent density for a best-fit cubic relationship.

Based on clinical experience with varying densities of available trabecular bone, Misch[285] defined two types of trabecular bone in his clinical classification scheme for the mandible and maxilla: (1) *coarse* (Division 2 [D2]) in the anterior mandible and (2) *fine* trabecular bone in the posterior mandible (Division 3 [D3]). Qu[264] found a significant difference between apparent density in region 1 (anterior mandible) and in regions 2 and 3 (posterior mandible). No significant difference was noted between region 2 and region 3. The results of the study by Qu thus provide quantitative validation of Misch's classification scheme for trabecular bone in the oral environment.

References

1. Dattilo DJ, Misch CM, Arena S: Interface analysis of hydroxylapatite-coated implants in a human vascularized iliac bone graft, *Int J Oral Maxillofac Implants* 10: 405-409, 1995.
2. Ericsson I, Johansson CB, Bystedt H et al: A histomorphometric evaluation of bone-to-implant contact on machine-prepared and roughened titanium dental implants: a pilot study in the dog, *Clin Oral Implants Res* 5:202-206, 1994.
3. Cook SD Salkeld SL, Gaisser DM et al: The effect of surface macrotexture on the mechanical and histologic characteristics of hydroxylapatite-coated dental implants, *J Oral Implantol* 19:288-294, 1993.
4. Clift SE, Fisher J, Watson CJ: Stress and strain distribution in the bone surrounding a new design of dental implant: a comparison with a threaded Brånemark type implant. Proceedings of the Institute of Mechanical Engineers, *J Med Eng Technol* 207:133-138, 1993.
5. De Lange G, De Putter C: Structure of the bone interface to dental implants in vivo, *J Oral Implantol* 19:123-135, 1993.
6. Weber HP, Fiorellini JP: The biology and morphology of the implant-tissue interface, *Alpha Omegan* 85:61-64, 1992.
7. Sisk AL, Steflik DE, Parr GR et al: A light and electron microscopic comparison of osseointegration of six implant types, *J Oral Maxillofac Surg* 50:709-716, 1992.

WWW.HIGHDENT.IR
مرجع دندانسازان و دندانپزشکان

8. Pilliar RM, Lee JM, Davies JE: Interface zone—factors influencing its structure for cementless implants. In Morrey BF, editor: *Biological, material, and mechanical considerations of joint replacement*, New York, 1993, Raven Press.
9. Clemow AJ, Weinstein AM, Klawitter JJ et al: Interface mechanics of porous titanium implants, *J Biomed Mater Res* 15:73-82, 1981.
10. Sadegh AM, Luo GM, Cowin SC: Bone ingrowth: an application of the boundary element method to bone remodeling at the implant interface, *J Biomech* 26:167-182, 1992.
11. Soballe K, Hansen ES, B-Rasmussen H et al: Tissue ingrowth into titanium and hydroxyapatite-coated implants during stable and unstable mechanical conditions, *J Orthop Res* 10:285-299, 1992.
12. Schwartz Z, Boyan BD: Underlying mechanisms at the bone-biomaterial interface, *J Cell Biochem* 56:340-347, 1994.
13. Linder L, Albrektsson T, Brånemark PI et al: Electron microscopic analysis of the bone-titanium interface, *Acta Orthop Scand* 54:45-52, 1983.
14. Ravaglioli A, Krajewski A, Biasini V et al: Interface between hydroxyapatite and mandibular human bone tissue, *Biomaterials* 13:162-167, 1992.
15. Brunski JB, Moccia AF Jr, Pollack SR et al: The influence of functional use of endosseous dental implants on the tissue-implant interface. II. Clinical aspects, *J Dent Res* 58:1970-1980, 1979.
16. Brunski JB: The influence of force, motion, and related quantities on the response of bone to implants. In Fitzgerald JR, editor: *Non-cemented total hip arthroplasty*, New York, 1988, Raven Press.
17. Albrektsson T: Direct bone anchorage of dental implants, *J Prosthet Dent* 50:255-261, 1983.
18. Boss JH, Shajrawi I, Mendes DG: The nature of the bone-implant interface, *Med Prog Technol* 20:119-142, 1994.
19. Albrektsson T, Brånemark PI, Hansson H-A: The interface zone of inorganic implants in vivo: titanium implants in bone, *Ann Biomed Eng* 11:1-27, 1983.
20. Pilliar RM, Lee JM, Maniatopoulos C: Observations on the effect of movement on bone ingrowth into porous-surfaced implants, *Clin Orthop Relat Res* 208:108, 1986.
21. Marks SC, Popoff SN: Bone cell biology: the regulation of development, structure and function in the skeleton, *Am J Anat* 183:1-44, 1988.
22. Duncan RL, Turner CH: Mechanotransduction and the functional response of bone to mechanical strain, *Calcif Tissue Int* 57:344-358, 1995.
23. Turner CH, Pavalko FM: Mechanotransduction and functional response of the skeleton to physical stress: the mechanisms and mechanics of bone adaptation, *J Orthop Sci* 3:346-355, 1998.
24. Cowin SC, Moss-Salentijn L, Moss ML: Candidates for the mechanosensory system in bone, *J Biomech Eng* 113:191-197, 1991.
25. Burger EH, Klein-Nulend J: Mechanotransduction in bone—role of the lacuno-canalicular network, *FASEB J* 13:S101-S112, 1999.
26. Burger EH, Klein-Nulend J, van der Plas A et al: Function of osteocytes in bone—their role in mechanotransduction, *J Nutr* 125(suppl 7):2020S-2023S, 1995.
27. Klein-Nulend J, van der Plas A, Semeins CM et al: Sensitivity of osteocytes to biomechanical stress in vitro, *FASEB J* 9:441-445, 1995.
28. Westbroek I, Ajubi NE, Alblas MJ et al: Differential stimulation of prostaglandin G/H synthase-2 in osteocytes and other osteogenic cells by pulsating fluid flow, *Biochem Biophys Res Commun* 268:414-419, 2000.
29. Vezeridis PS, Semeins CM, Chen Q et al: Osteocytes subjected to pulsating fluid flow regulate osteoblast proliferation and differentiation, *Biochem Biophys Res Commun* 348:1082-1088, 2006.
30. You J, Yellowley CE, Donahue HJ et al: Substrate deformation levels associated with routine physical activity are less stimulatory to bone cells relative to loading-induced oscillatory fluid flow, *J Biomech Eng* 122:387-393, 2000.
31. Burger EH, Veldhuijzen JP: Influence of mechanical factors on bone formation, resorption and growth in vitro. In Hall K, editor: *Bone growth*, Melbourne, 1993, CRC Press.
32. Weinbaum S, Cowin SC, Zeng Y: A model for the excitation of osteocytes by mechanical loading-induced bone fluid shear stresses, *J Biomech* 27:339-360, 1994.
33. You L, Cowin SC, Schaffler MB et al: A model for strain amplification in the actin cytoskeleton of osteocytes due to fluid drag on pericellular matrix, *J Biomech* 34:1375-1386, 2001.
34. Shapiro F, Cahill C, Malatantis G et al: Transmission electron microscopic demonstration of vimentin in rat osteoblast and osteocyte cell bodies and processes using the immunogold technique, *Anat Rec* 241:39-48, 1995.
35. You LD, Weinbaum S, Cowin SC et al: Ultrastructure of the osteocyte process and its pericellular matrix, *Anat Rec A Discov Mol Cell Evol Biol* 278A:505-513, 2004.
36. Han Y, Cowin SC, Schaffler MB et al: Mechanotransduction and strain amplification in osteocyte cell processes, *Proc Natl Acad Sci U S A* 101:16689-16694, 2004.
37. Cowin SC: The significance of bone microstructure in mechanotransduction, *J Biomech*, April 11, 2007 (Epub ahead of print).
38. Rubin J, Rubin C, Jacobs CR: Molecular pathways mediating mechanical signaling in bone, *Gene* 367:1-16, 2006.
39. Liedert A, Kaspar D, Blakytny R et al: Signal transduction pathways involved in mechanotransduction in bone cells, *Biochem Biophys Res Commun* 349:1-5, 2006.
40. Bonewald LF: Mechanosensation and transduction in osteocytes, *Bonekey Osteovision* 3:7-15, 2006.
41. Yellowley CE, Li Z, Zhou Z et al: Functional gap junctions between osteocytic and osteoblastic cells, *J Bone Miner Res* 15:209-217, 2000.
42. Cheng B, Zhao S, Luo J et al: Expression of functional gap junctions and regulation by fluid flow in osteocyte-like MLO-Y4 cells, *J Bone Miner Res* 16:249-259, 2001.
43. Alford AI, Jacobs CR, Donahue HJ: Oscillating fluid flow regulates gap junction communication in osteocytic MLO-Y4 cells by an ERK1/2 MAP kinase-dependent mechanism, *Bone* 33:64-70, 2003.
44. Taylor AF, Saunders MM, Shingle DL et al: Mechanically stimulated osteocytes regulate osteoblastic activity via gap junctions, *Am J Physiol Cell Physiol* 292:C545-C552, 2007.
45. Hakeda Y, Arakawa T, Ogasawara A et al: Recent progress in studies on osteocytes—osteocytes and mechanical stress, *Kaibogaku Zasshi* 75:451-456, 2000.
46. Jiang JX, Cheng B: Mechanical stimulation of gap junctions in bone osteocytes is mediated by prostaglandin E_2, *Cell Commun Adhes* 8:283-288, 2001.
47. Cheng B, Kato Y, Zhao S et al: PGE(2) is essential for gap junction-mediated intercellular communication between osteocyte-like MLO-Y4 cells in response to mechanical strain, *Endocrinology* 142:3464-3473, 2001.

WWW.HIGHDENT.IR

48. Saunders MM, You J, Zhou Z et al: Fluid flow-induced prostaglandin E_2 response of osteoblastic ROS 17/2.8 cells is gap junction-mediated and independent of cytosolic calcium, *Bone* 32:350-356, 2003.
49. Cherian PP, Cheng B, Gu S et al: Effects of mechanical strain on the function of gap junctions in osteocytes are mediated through the prostaglandin EP_2 receptor, *J Biol Chem* 278:43146-43156, 2003.
50. Noble BS, Peet N, Stevens HY et al: Mechanical loading: biphasic osteocyte survival and targeting of osteoclasts for bone destruction in rat cortical bone, *Am J Physiol Cell Physiol* 284:C934-C943, 2003.
51. Bakker A, Klein-Nulend J, Burger E: Shear stress inhibits while disuse promotes osteocyte apoptosis, *Biochem Biophys Res Commun* 320:1163-1168, 2004.
52. Tan SD, Kuijpers-Jagtman AM, Semeins CM et al: Fluid shear stress inhibits TNF alpha-induced osteocyte apoptosis, *J Dent Res* 85:905-909, 2006.
53. Dodd JS, Raleigh JA, Gross TS: Osteocyte hypoxia: a novel mechanotransduction pathway, *Am J Physiol* 277 (3 pt 1):C598-C602, 1999.
54. O'Brien CA, Jia D, Plotkin LI et al: Glucocorticoids act directly on osteoblasts and osteocytes to induce their apoptosis and reduce bone formation and strength, *Endocrinology* 145:1835-1841, 2004.
55. Gross TS, King KA, Rabaia NA et al: Upregulation of osteopontin by osteocytes deprived of mechanical loading or oxygen, *J Bone Miner Res* 20:250-256, 2005.
56. Meier GH: Die architekture der spongiosa, *Arch Anat Physiol Wiss Med* 34:615-628, 1887.
57. Wolff J: *Das gesetz der transformation der knochen*, Berlin, 1892, August Hirschwald.
58. Roux W: *Gesammelte abhand lungen uber die entwick lungsmechanik der organismen*, 1895.
59. Frost HM: Bone "mass" and the "mechanostat": a proposal, *Anat Rec* 219:1-9, 1987.
60. Lanyon LE: Biomechanical properties and response in bone matrix and bone specific products. In Hall BK, editor: *Bone*, vol 3, Boca Raton, Fla, 1991, CRC Press.
61. Cowin SC, Hegedus DH: Bone remodeling I: theory of adaptive elasticity, *J Elast* 6:313-326, 1976.
62. Cowin SC, Hegedus DH: Bone remodeling II: small strain adaptive elasticity, *J Elast* 6:337-352, 1976.
63. Cowin SC, Nachlinger RR: Bone remodeling II: uniqueness and stability in adaptive elasticity theory, *J Elast* 8:285-295, 1978.
64. Hassler CR, Rylicky EF, Cummings KD et al: Quantification of bone stress during remodeling, *J Biomech* 13:185-190, 1980.
65. Turner CH: Three rules for bone adaptation to mechanical stimuli, *Bone* 23:399-407, 1998.
66. Akuz E, Braun JT, Brown NA et al: Static versus dynamic loading in the mechanical modulation of vertebral growth, *Spine* 31:E952-E958, 2006.
67. Mosley JR, March BM, Lynch J et al: Strain magnitude related changes in whole bone architecture in growing rats, *Bone* 20:191-198, 1997.
68. Torrance AG, Mosley JR, Suswillo RF et al: Noninvasive loading of the rat ulna in vivo induces a strain-related modeling response uncomplicated by trauma or periostal pressure, *Calcif Tissue Int* 54:241-247, 1994.
69. Mosley JR, Lanyon LE: Strain rate as a controlling influence on adaptive modeling in response to dynamic loading of the ulna in growing male rats, *Bone* 23: 313-318, 1998.
70. Robling AG, Duijvelaar KM, Geevers JV et al: Modulation of appositional and longitudinal bone growth in the rat ulna by applied static and dynamic force, *Bone* 29: 105-113, 2001.
71. Ohashi N, Robling AG, Burr DB et al: The effects of dynamic axial loading on the rat growth plate, *J Bone Miner Res* 17:284-292, 2002.
72. Hert J: Acceleration of the growth after decrease of load on epiphyseal plates by means of spring distractors, *Folia Morphol (Praha)* 17:194-203, 1969.
73. Frost HM: Skeletal structural adaptations to mechanical usage (SATMU): 3. The hyaline cartilage modeling problem, *Anat Rec* 226:423-432, 1990.
74. Rubin CT, Lanyon LE: Regulation of bone formation by applied dynamic loads, *J Bone Joint Surg Am* 66:397-402, 1984.
75. Robling AG, Burr DB, Turner CH: Partitioning a daily mechanical stimulus into discrete loading bouts improves the osteogenic response to loading, *J Bone Miner Res* 15:1596-1602, 2000.
76. Srinivasan S, Weimer DA, Agans SC et al: Low-magnitude mechanical loading becomes osteogenic when rest is inserted between each load cycle, *J Bone Miner Res* 17:1613-1620, 2002.
77. Srinivasan S, Agans SC, King KA et al: Enabling bone formation in the aged skeleton via rest-inserted mechanical loading, *Bone* 33:946-955, 2003.
78. LaMothe JM, Zernicke RF: Rest insertion combined with high-frequency loading enhances osteogenesis, *J Appl Physiol* 96:1788-1793, 2004.
79. Srinivasan S, Ausk BJ, Poliachik SL et al: Rest-inserted loading rapidly amplifies the response of bone to small increases in strain and load cycles, *J Appl Physiol* 102: 1945-1952, 2007.
80. Robling AG, Hinant FM, Burr DB et al: Improved bone structure and strength after long-term mechanical loading is greatest if loading is separated into short bouts, *J Bone Miner Res* 17:1545-1554, 2002.
81. Turner CH, Forwood MR, Rho JY et al: Mechanical loading thresholds for lamellar and woven bone formation, *J Bone Miner Res* 9:87-97, 1994.
82. Gross TS, Poliachik SL, Ausk BJ et al: Why rest stimulates bone formation: a hypothesis based on complex adaptive phenomenon, *Exerc Sport Sci Rev* 32:9-13, 2004.
83. Hsieh Y-F, Robling A, Ambrosius W et al: Mechanical loading of diaphyseal bone in vivo: the strain threshold for an osteogenic response varies with location, *J Bone Miner Res* 16:2291-2297, 2001.
84. Iwamoto J, Yeh J, Aloia J: Differential effect of treadmill exercise on three cancellous bone sites in the young growing rat, *Bone* 24:163-169, 1999.
85. Hamrick MW, Skedros JG, Pennington C et al: Increased osteogenic response to exercise in metaphyseal versus diaphyseal cortical bone, *J Musculoskelet Neuronal Interact* 6:258-263, 2006.
86. Zhang P, Tanaka SM, Jiang H et al: Diaphyseal bone formation in murine tibiae in response to knee loading, *J Appl Physiol* 100:1452-1459, 2006.
87. Rubin C, Turner AS, Mallinckrodt C et al: Mechanical strain, induced noninvasively in the high-frequency domain, is anabolic to cancellous bone, but not cortical bone, *Bone* 30:445-452, 2002.
88. Judex S, Boyd S, Qin YX et al: Adaptations of trabecular bone to low magnitude vibrations result in more uniform stress and strain under load, *Ann Biomed Eng* 31:12-20, 2003.

WWW.HIGHDENT.IR

انجمن دندانسازان و دندانپزشکان

89. Rubin C, Turner AS, Muller R et al: Quantity and quality of trabecular bone in the femur are enhanced by a strongly anabolic, noninvasive mechanical intervention, *J Bone Miner Res* 17:349-357, 2002.
90. Xie L, Jacobson JM, Choi ES et al: Low-level mechanical vibrations can influence bone resorption and bone formation in the growing skeleton, *Bone* 39:1059-1066, 2006.
91. Judex S, Lei X, Han D et al: Low-magnitude mechanical signals that stimulate bone formation in the ovariectomized rat are dependent on the applied frequency but not on the strain magnitude, *J Biomech* 40:1333-1339, 2007.
92. van der Meulen MC, Morgan TG, Yang X et al: Cancellous bone adaptation to in vivo loading in a rabbit model, *Bone* 38:871-877, 2006.
93. Rubin C, Judex S, Qin YX: Low-level mechanical signals and their potential as a non-pharmacological intervention for osteoporosis, *Age Ageing* 35(suppl 2): ii32-ii36, 2006.
94. Gardner MJ, van der Meulen MC, Demetrakopoulos D et al: In vivo cyclic axial compression affects bone healing in the mouse tibia, *J Orthop Res* 24:1679-1686, 2006.
95. Augat P, Burger J, Schorlemmer S et al: Shear movement at the fracture site delays healing in a diaphyseal fracture model, *J Orthop Res* 21:1011-1017, 2003.
96. Takeda T, Narita T, Ito H: Experimental study on the effect of mechanical stimulation on the early stage of fracture healing, *J Nippon Med Sch* 71:252-262, 2004.
97. Yeh CK, Rodan GA: Tensile forces enhance prostaglandin synthesis in osteoblastic cells grown on collagen ribbons, *Calcif Tissue Int* 36:67-71, 1984.
98. Brighton CT, Sennett BJ, Farmer JC et al: The inositol phosphate pathway as a mediator in the proliferative response of rat calvarial bone cells to cyclical biaxial mechanical strain, *J Orthop Res* 10:385-393, 1992.
99. Harrell A, Binderman DS: Biochemical effect of mechanical stress on cultured bone cells, *Calcif Tissue Res* 22(suppl): 202-209, 1977.
100. Rodan GA, Bourret LA, Harvey A et al: Cyclic AMP and cyclic GMP mediators of the mechanical effects in bone remodeling, *Science* 198:467-469, 1975.
101. Reich KM, Frangos JA: Effect of flow on prostaglandin E_2 and inositol triphosphate levels in osteoblasts, *Am J Physiol* 261:C428-C432, 1991.
102. Jones DB, Nolte H, Scholubbers JG et al: *Biochemical signal transduction of mechanical strain in osteoblast-like cells, Biointeractions,* Oxford, 1990, Oxford University Press.
103. Alberts B, Bray D, Lewis J et al: Cell signaling. In Alberts B, Bray D, Lewis J et al, editors: *Molecular biology of the cell,* New York, 1994, Garland.
104. Somjen D, Binderman I, Berger E et al: Bone remodeling induced by physical stress is prostaglandin E2 mediated, *Biochem Biophys Acta* 627:91-100, 1980.
105. Ozawa H, Imamura K, Abe E et al: Effect of a continuously applied compressive pressure on mouse osteoblast-like-cells (MC3T3-E1) in vitro, *J Cell Physiol* 142:177-185, 1990.
106. Murray DW, Rushton N: The effect of strain on bone cell prostaglandin E_2 release: a new experimental method, *Calcif Tissue Int* 47:35-39, 1990.
107. Rawlinson SCF, el-Haj AJ, Minter SL et al: Loading-related increases in prostaglandin production in cores of adult canine cancellous bone in vitro: a role for prostacyclin in adaptive bone remodeling? *J Bone Miner Res* 6:1345-1351, 1991.
108. Rawlinson SC, Mohan S, Baylink DJ et al: Exogenous prostacyclin, but not prostaglandin E2 produces similar responses in both G6PD activity and RNA production as mechanical loading, and increases IGF-II release, in adult cancellous bone in culture, *Calcif Tissue Int* 53:324-329, 1993.
109. Chow JWM, Chambers TJ: Indomethacin has distinct early and late actions on bone formation induced by mechanical stimulation, *Am J Physiol* 267:E287-E292, 1994.
110. Butler WT: The nature and significance of osteopontin, *Connect Tissue Res* 23:123-136, 1989.
111. Sodek J, Chen J, Nagata T et al: Regulation of osteopontin expression in osteoblasts, *Ann N Y Acad Sci* 760:223-241, 1995.
112. Patterson-Buckendahl P, Globus RK, Bikle DD et al: Effects of simulated weightlessness on rat osteocalcin and bone calcium, *Am J Physiol* 257:R1103-R1109, 1989.
113. Miyajima K, Suzuki S, Iwata T et al: Mechanical stress as a stimulant to the production of osteocalcin in osteoblast-like cells, *Aichi Gakuin Dent Sci* 4:1-5, 1991.
114. Forwood MR: Inducible cyclo-oxygenase (COX-2) mediates the induction of bone formation by mechanical loading in vivo, *J Bone Miner Res* 11:1688-1693, 1996.
115. Fox SW, Chambers TJ, Chow JWM: Nitric oxide is an early mediator of the increase in bone formation by mechanical stimulation, *Am J Physiol* 270:E955-E960, 1996.
116. Turner CH, Takano Y, Owan I et al: Nitric oxide inhibitor L-NAME suppresses mechanically induced bone formation in rats, *Am J Physiol* 270:E634-E639, 1996.
117. Chow JW, Fox SW, Lean JM et al: Role of nitric oxide and prostaglandins in mechanically induced bone formation, *J Bone Miner Res* 13:1039-1044, 1998.
118. Chambers TJ, Fox S, Jagger CJ et al: The role of prostaglandins and nitric oxide in the response of bone to mechanical forces, *Osteoarthritis Cartilage* 7:422-433, 1999.
119. Chow JW: Role of nitric oxide and prostaglandins in the bone formation response to mechanical loading, *Exerc Sport Sci Rev* 28:185-188, 2000.
120. Klein-Nulend J, Semeins CM, Ajubi NE et al: Pulsating fluid flow increases nitric oxide (NO) synthesis by osteocytes but not periosteal fibroblasts—correlation with prostaglandin upregulation, *Biochem Biophys Res Commun* 14:217:640-648, 1995.
121. Ajubi NE, Klein-Nulend J, Nijweide PJ et al: Pulsating fluid flow increases prostaglandin production by cultured chicken osteocytes—a cytoskeleton-dependent process, *Biochem Biophys Res Commun* 225:62-68, 1996.
122. Klein-Nulend J, Burger EH, Semeins CM et al: Pulsating fluid flow stimulates prostaglandin release and inducible prostaglandin G/H synthase mRNA expression in primary mouse bone cells, *J Bone Miner Res* 12:45-51, 1997.
123. Smalt R, Mitchell FT, Howard RL et al: Induction of NO and prostaglandin E_2 in osteoblasts by wall-shear stress but not mechanical strain, *Am J Physiol* 273(4 pt 1): E751-E758, 1997.
124. Bakker AD, Soejima K, Klein-Nulend J et al: The production of nitric oxide and prostaglandin E(2) by primary bone cells is shear stress dependent, *J Biomech* 34:671-677, 2001.
125. Bacabac RG, Smit TH, Mullender MG et al: Nitric oxide production by bone cells is fluid shear stress rate dependent, *Biochem Biophys Res Commun* 315:823-829, 2004.
126. Fox SW, Chambers TJ, Chow JW: Nitric oxide is an early mediator of the increase in bone formation by

WWW.HIGHDENT.IR

mechanical stimulation, *Am J Physiol* 270(6 pt 1): E955-E960, 1996.
127. Sterck JG, Klein-Nulend J, Lips P et al: Response of normal and osteoporotic human bone cells to mechanical stress in vitro, *Am J Physiol* 274(6 pt 1):E1113-E1120, 1998.
128. Owan I, Burr DB, Turner CH et al: Mechanotransduction in bone: osteoblasts are more responsive to fluid forces than mechanical strain, *Am J Physiol* 273(3 pt 1):C810-C815, 1997.
129. Sundaramurthy S, Mao JJ: Modulation of endochondral development of the distal femoral condyle by mechanical loading, *J Orthop Res* 24:229-241, 2006.
130. Kreke MR, Goldstein AS: Hydrodynamic shear stimulates osteocalcin expression but not proliferation of bone marrow stromal cells, *Tissue Eng* 10:780-788, 2004.
131. Kreke MR, Huckle WR, Goldstein AS: Fluid flow stimulates expression of osteopontin and bone sialoprotein by bone marrow stromal cells in a temporally dependent manner, *Bone* 36:1047-1055, 2005.
132. Chow JW, Fox S, Jagger CJ et al: Role for parathyroid hormone in mechanical responsiveness of rat bone, *Am J Physiol* 274(1 pt 1):E146-E154, 1998.
133. Bakker AD, Joldersma M, Klein-Nulend J et al: Interactive effects of PTH and mechanical stress on nitric oxide and PGE_2 production by primary mouse osteoblastic cells, *Am J Physiol Endocrinol Metab* 285:E608-E613, 2003.
134. Miyauchi A, Notoya K, Mikuni-Takagaki Y et al: Parathyroid hormone-activated volume-sensitive calcium influx pathways in mechanically loaded osteocytes, *J Biol Chem* 275:3335-3342, 2000.
135. Hasegawa S, Sato S, Saito S et al: Mechanical stretching increases the number of cultured bone cells synthesizing DNA and alters their pattern of protein synthesis, *Calcif Tissue Int* 37:431-436, 1985.
136. Buckley MJ, Banes AJ, Levin LG et al: Osteoblasts increase their rate of division and align in response to cyclic, mechanical tension in vitro, *Bone Miner* 4:225-236, 1988.
137. Meyer U et al: Influence of mechanical strain on osteoblast behavior, *J Dent Res* 75:29, 1996.
138. Binderman I, Zor U, Kaye AM et al: The transduction of mechanical force into biochemical events in bone cells may involve activation of phospholipase A2, *Calcif Tissue Int* 42:261-266, 1988.
139. Rodan GA, Mensi T, Harvey A: A quantitative method for the application of compressive forces to bone in tissue culture, *Calcif Tissue Res* 18:125, 1975.
140. Kaspar D, Seidl W, Neidlinger-Wilke C et al: Dynamic cell stretching increases human osteoblast proliferation and CICP synthesis but decreases osteocalcin synthesis and alkaline phosphatase activity, *J Biomech* 33:45-51, 2000.
141. Kaspar D, Seidl W, Ignatius A et al: In vitro cell behavior of human osteoblasts after physiological dynamic stretching, *Orthopade* 29:85-90, 2000.
142. Kaspar D, Seidl W, Neidlinger-Wilke C et al: In vitro effects of dynamic strain on the proliferative and metabolic activity of human osteoblasts, *J Musculoskelet Neuronal Interact* 1:161-164, 2000.
143. Nagatomi J, Arulanandam BP, Metzger DW et al: Frequency- and duration-dependent effects of cyclic pressure on select bone cell functions, *Tissue Eng* 7:717-728, 2001.
144. Kaspar D, Seidl W, Neidlinger-Wilke C et al: Proliferation of human-derived osteoblast-like cells depends on the cycle number and frequency of uniaxial strain, *J Biomech* 35:873-880, 2002.
145. Lanyon L: Control of bone architecture by functional load bearing, *J Bone Miner Res* 7:369-375, 1992.
146. Raab-Cullen DM, Thiede MA, Petersen DN et al: Mechanical loading stimulates rapid changes in periosteal gene expression, *Calcif Tissue Int* 55:473-478, 1994.
147. Weyts FA, Bosmans B, Niesing R et al: Mechanical control of human osteoblast apoptosis and proliferation in relation to differentiation, *Calcif Tissue Int* 72:505-512, 2003.
148. O'Driscoll SW: Articular cartilage regeneration using periosteum, *Clin Orthop Relat Res* (suppl 367):S186-S203, 1999.
149. Ito Y, Sanyal A, Fitzsimmons JS et al: Histomorphological and proliferative characterization of developing periosteal neochondrocytes in vitro, *J Orthop Res* 19:405-413, 2001.
150. O'Driscoll SW, Saris DB, Ito Y et al: The chondrogenic potential of periosteum decreases with age, *J Orthop Res* 19:95-103, 2001.
151. O'Driscoll SW, Fitzsimmons JS: The role of periosteum in cartilage repair, *Clin Orthop Relat Res* (suppl 391): S190-S207, 2001.
152. Saris DB, Sanyal A, An KN et al: Periosteum responds to dynamic fluid pressure by proliferating in vitro, *J Orthop Res* 17:668-677, 1999.
153. Mukherjee N, Saris DB, Schultz FM et al: The enhancement of periosteal chondrogenesis in organ culture by dynamic fluid pressure, *J Orthop Res* 19:524-530, 2001.
154. Ives D, Eskin S, McIntire C: Mechanical effects on endothelial cell morphology: in vitro assessment, *In Vitro Cell Dev Biol* 22:500, 1986.
155. Carvalho RS, Scott JE, Suga DM et al: Stimulation of signal transduction pathways in osteoblasts by mechanical strain potentiated by parathyroid hormone, *J Bone Miner Res* 9:999-1011, 1994.
156. Ponik SM, Triplett JW, Pavalko FM: Osteoblasts and osteocytes respond differently to oscillatory and unidirectional fluid flow profiles, *J Cell Biochem* 100:794-807, 2007.
157. Ingber D: Integrins as mechanochemical transducers, *Curr Opin Cell Biol* 3:841-848, 1991.
158. Alberts B, Bray D, Lewis J et al: Extracellular matrix receptors on animals cells: the integrins. In Alberts B, Bray D, Lewis J et al, editors: *Molecular biology of the cell*, New York, 1994, Garland.
159. Salter DM, Robb JE, Wright MO: Electrophysiological responses of human bone cells to mechanical stimulation: evidence for specific integrin function in mechanotransduction, *J Bone Miner Res* 12:1133-1141, 1997.
160. Meazzini MC, Toma CD, Schaffer JL et al: Osteoblast cytoskeletal modulation in response to mechanical strain in vitro, *J Orthop Res* 16:170-180, 1998.
161. Pavalko FM, Chen NX, Turner CH et al: Fluid shear-induced mechanical signaling in MC3T3-E1 osteoblasts requires cytoskeleton-integrin interactions, *Am J Physiol* 275(6 pt 1):C1591-C1601, 1998.
162. Sastry SK, Horwitz AF: Integrin cytoplasmic domains: mediators of cytoskeletal linkages and extra- and intercellular initiated transmembrane signaling, *Curr Opin Cell Biol* 5:831-853, 1993.
163. Schwartz MA, Ingber DE: Integrating with integrins, *Mol Biol Cell* 5:389-393, 1994.
164. Davies PF, Robotewskyj A, Griem ML: Quantitative studies of endothelial cell adhesion: directional remodeling of focal adhesion sites in response to flow forces, *J Clin Invest* 93:2031-2038, 1994.

WWW.HIGHDENT.IR
انجمن علمی مهندسان دندانسازان و دندانپزشکان

165. Wang N, Butler JP, Ingber DE: Mechanotransduction across the cell surface and through the cytoskeleton, *Science* 260:1124-1127, 1993.
166. Toma CD, Ashkar S, Gray ML et al: Signal transduction of mechanical stimuli is dependent on microfilament integrity: identification of osteopontin as a mechanically induced gene in osteoblasts, *J Bone Miner Res* 12:1626-1636, 1997.
167. Carvalho RS, Schaffer JL, Gerstenfeld LC: Osteoblasts induce osteopontin expression in response to attachment on fibronectin: demonstration of a common role for integrin receptors in the signal transduction processes of cell attachment and mechanical stimulation, *J Cell Biochem* 70:376-390, 1998.
168. Carvalho RS, Bumann A, Schaffer JL et al: Predominant integrin ligands expressed by osteoblasts show preferential regulation in response to both cell adhesion and mechanical perturbation, *J Cell Biochem* 84:497-508, 2002.
169. Damsky CH, Werb Z: Signal transduction by integrin receptors for extracellular matrix: cooperative processing of extracellular information, *Curr Opin Cell Biol* 5:772-781, 1992.
170. Jaiswal RK, Jaiswal N, Bruder SP et al: Adult human mesenchymal stem cell differentiation to the osteogenic or adipogenic lineage is regulated by mitogen-activated protein kinase, *J Biol Chem* 275:9645-9652, 2000.
171. Lai CF, Chaudhary L, Fausto A et al: ERK is essential for growth, differentiation, integrin expression, and cell function in human osteoblastic cells, *J Biol Chem* 276:14443-14450, 2001.
172. Ogata T: Fluid flow-induced tyrosine phosphorylation and participation of growth factor signaling pathway in osteoblast-like cells, *J Cell Biochem* 76:529-538, 2000.
173. You J, Reilly GC, Zhen X et al: Osteopontin gene regulation by oscillatory fluid flow via intracellular calcium mobilization and activation of mitogen-activated protein kinase in MC3T3-E1 osteoblasts, *J Biol Chem* 276:13365-13671, 2001.
174. Wadhwa S, Godwin SL, Peterson DR et al: Fluid flow induction of cyclo-oxygenase 2 gene expression in osteoblasts is dependent on an extracellular signal-regulated kinase signaling pathway, *J Bone Miner Res* 17:266-274, 2002.
175. Ziros PG, Gil AP, Georgakopoulos T et al: The bone-specific transcriptional regulator Cbfa1 is a target of mechanical signals in osteoblastic cells, *J Biol Chem* 277:23934-23941, 2002.
176. Weyts FA, Li YS, van Leeuwen J et al: ERK activation and alpha v beta 3 integrin signaling through Shc recruitment in response to mechanical stimulation in human osteoblasts, *J Cell Biochem* 87:85-92, 2002.
177. Kapur S, Baylink DJ, Lau KH: Fluid flow shear stress stimulates human osteoblast proliferation and differentiation through multiple interacting and competing signal transduction pathways, *Bone* 32:241-251, 2003.
178. Boutahar N, Guignandon A, Vico L et al: Mechanical strain on osteoblasts activates autophosphorylation of focal adhesion kinase and proline-rich tyrosine kinase 2 tyrosine sites involved in ERK activation, *J Biol Chem* 279:30588-30599, 2004.
179. Plotkin LI, Mathov I, Aguirre JI et al: Mechanical stimulation prevents osteocyte apoptosis: requirement of integrins, Src kinases, and ERKs, *Am J Physiol Cell Physiol* 289:C633-C643, 2005.
180. Hynes RO: Integrins: versatility, modulation and signaling in cell adhesion, *Cell* 69:11-25, 1992.
181. Clover J, Dodds RA, Gowen M: Integrin subunit expression by human osteoblasts and osteoclasts in situ and in culture, *J Cell Sci* 103:267-271, 1992.
182. Buckley MJ, Banes AJ, Levin LG et al: Osteoblasts increase their rate of division and align in response to cyclic, mechanical tension in vitro, *Bone Miner* 4:225-236, 1988.
183. Harter LV, Hruska KA, Duncan RL: Human osteoblast-like cells respond to mechanical strain with increased bone matrix protein production independent of hormonal regulation, *Endocrinology* 136:528-535, 1995.
184. Zaman G, Dallas SL, Lanyon LE: Cultured embryonic bone shafts show osteogenic responses to mechanical loading, *Calcif Tissue Int* 51:132-136, 1992.
185. Nagatomi J, Arulanandam BP, Metzger DW et al: Cyclic pressure affects osteoblast functions pertinent to osteogenesis, *Ann Biomed Eng* 31:917-923, 2003.
186. Tang LL, Wang YL, Pan J et al: The effect of step-wise increased stretching on rat calvarial osteoblast collagen production, *J Biomech* 37:157-161, 2004.
187. Stanford CM, Keller JC: The concept of osseointegration and bone matrix expression, *Crit Rev Oral Biol Med* 2:83-101, 1991.
188. Pavlin D, Dove SB, Zadro R et al: Mechanical loading stimulates differentiation of periodontal osteoblasts in a mouse osteoinduction model: effect on type I collagen and alkaline phosphatase genes, *Calcif Tissue Int* 67:163-172, 2000.
189. Pavlin D, Zadro R, Gluhak-Heinrich J: Temporal pattern of stimulation of osteoblast-associated genes during mechanically induced osteogenesis in vivo: early responses of osteocalcin and type I collagen, *Connect Tissue Res* 42:135-148, 2001.
190. Reich A, Jaffe N, Tong A et al: Weight loading young chicks inhibits bone elongation and promotes growth plate ossification and vascularization, *J Appl Physiol* 98:2381-2389, 2005.
191. Hankenson KD, Ausk BJ, Bain SD et al: Mice lacking thrombospondin 2 show an atypical pattern of endocortical and periosteal bone formation in response to mechanical loading, *Bone* 38:310-316, 2006.
192. Rigsby DF: *Analysis of the metabolic and morphologic response of osteoblasts cultured on Ti-6A1-4V to dynamic, uniaxial stress* [doctoral thesis], Birmingham, Ala, 1997, University of Alabama.
193. Reilly DT, Burstein AH: The elastic and ultimate properties of compact bone tissue, *J Biomech* 8:393, 1975.
194. Yoon HS, Katz JL: Ultrasonic wave propagation in human cortical bone II: measurements of elastic properties and micro-hardness, *J Biomech* 9:459, 1976.
195. Knets I, Malmeister A: Deformability and strength of human compact bone tissue. In Brankov G, editor: *Mechanics of biological solids 1977*. Proceedings of the Euromechanic Colloquium 68, Sofia, 1977, Bulgarian Academy of Sciences.
196. Ashman RB, Cowin SC, Van Buskirk WC et al: A continuous wave technique for the measurement of the elastic properties of bone, *J Biomech* 17:349-361, 1984.
197. Ashman RB, Van Buskirk WC: The elastic properties of a human mandible, *Adv Dent Res* 1:64-67, 1987.
198. Dechow PC, Nail GA, Schwartz-Dabney CL et al: Elastic properties of human supraorbital and mandibular bone, *Am J Phys Anthropol* 90:291-306, 1993.
199. O'Mahony AM, Williams JL, Katz JO et al: Anisotropic elastic properties of cancellous bone from a human

WWW.HIGHDENT.IR

edentulous mandible, *Clin Oral Implants Res* 11:415-421, 2000.
200. O'Mahony AM, Williams JL, Spencer P: Anisotropic elasticity of cortical and cancellous bone in the posterior mandible increases peri-implant stress and strain under oblique loading, *Clin Oral Implants Res* 12:648-657, 2001.
201. Simsek B, Erkmen E, Yilmaz D et al: Effects of different inter-implant distances on the stress distribution around endosseous implants in posterior mandible: a 3D finite element analysis, *Med Phys Eng* 28:199-213, 2006.
202. Holmgren EP, Seckinger RJ, Kilgren LM et al: Evaluating parameters of osseointegrated dental implants using finite element analysis—a two-dimensional comparative study examining the effects of implant diameter, implant shape, and load direction, *J Oral Implantol* 24: 80-88, 1998.
203. Rees JS: The effect of variation in occlusal loading on the development of abfraction lesions: a finite element study, *J Oral Rehabil* 29:188-193, 2002.
204. Nohl FS, McCabe JF, Walls AWG: *The effect of load angle on strains induced in maxillary premolars in vitro* [abstract], Leeds, England, 1999, British Society of Dental Research Meeting.
205. O'Mahony A, Bowles Q, Woolsey G et al: Stress distribution in the single-unit osseointegrated dental implant: finite element analyses of axial and off-axial loading, *Implant Dent* 9:207-218, 2000.
206. Barbier L, Schepers E: Adaptive bone remodeling around oral implants under axial and nonaxial loading conditions in the dog mandible, *Int J Oral Maxillofac Implants* 12:215-223, 1997.
207. Barbier L, Vander Sloten J, Krzesinski G et al: Finite element analysis of non-axial versus axial loading of oral implants in the mandible of the dog, *J Oral Rehabil* 25:847-858, 1998.
208. Rho, JY, Roy ME II, Tsui TY et al: Elastic Properties of microstructural components of human bone tissue as measured by nanoindentation, *J Biomed Mater Res* 45: 48-54, 1999.
209. Rho JY, Zioupos P, Currey JD et al: Variations in the individual thick lamellar properties within osteons by nanoindentation, *Bone* 25:295-300, 1999.
210. Zysset KP, Guo XD, Hoffler CE et al: Elastic modulus and hardness of cortical and trabecular bone lamellae measured by nanoindentation in the human femur, *J Biomech* 32:1005-1012, 1999.
211. Fan Z, Swadener JG, Rho JY et al: Anisotropic properties of human tibial cortical bone as measured by nanoindentation, *J Orthop Res* 20:806-810, 3002.
212. Morgan EF, Keaveny TM: Dependence of yield strain of human trabecular bone on anatomic site, *J Biomech* 34:569-577, 2001.
213. Nagaraja S, Couse TL, Guldberg RE: Trabecular bone microdamage and microstructural stresses under uniaxial compression, *J Biomech* 38:707-716, 2005.
214. Bayraktar HH, Keaveny TM: Mechanisms of uniformity of yield strains for trabecular bone, *J Biomech* 37: 1671-1678, 2004.
215. Morgan EF, Yeh OC, Keaveny TM: Damage in trabecular bone at small strains, *Eur J Morphol* 42:13-21, 2005.
216. Wang X, Niebur GL: Microdamage propagation in trabecular bone due to changes in loading mode, *J Biomech* 39:781-790, 2006.
217. McElhaney JH: Dynamic response of bone and muscle tissue, *J Appl Physiol* 21:1231, 1966.
218. Evans GP, Behiri JC, Vaughan LC et al: The response of equine cortical bone to loading at strain rates experienced in vivo by galloping horse, *Equine Vet J* 24:125-128, 1992.
219. Pithioux M, Subit D, Chabrand P: Comparison of compact bone failure under two different loading rates: experimental and modeling approaches, *Med Eng Phys* 26:647-653, 2004.
220. Linde F, Norgaard P, Hvid I et al: Mechanical properties of trabecular bone: dependency on strain rate, *J Biomech* 24:803-809, 1991.
221. Ouyang J, Yang GT, Wu WZ et al: Biomechanical characteristics of human trabecular bone, *Clin Biomech (Bristol, Avon)* 12:522-524, 1997.
222. Kasra M, Grynpas MD: On shear properties of trabecular bone under torsional loading: effects of bone marrow and strain rate, *J Biomech*, April 18, 2007 (Epub ahead of print).
223. Carter DR, Hayes WC: The compressive behavior of bone as a two-phase porous structure, *J Bone Joint Surg* 59A:954, 1977.
224. Harrison A, Lewis TT: The development of an abrasion testing machine, *J Biomed Mater Res* 9:341, 1975.
225. Hoffler CE, Guo XE, Zysset PK et al: An application of nanoindentation technique to measure bone tissue lamellae properties, *J Biomech Eng* 127:1047-1053, 2005.
226. Carter DR, Hayes WC: Bones compressive strength: the influence of density and strain rate, *Science* 194:1174, 1976.
227. Fan Z, Rho JY: Effects of viscoelasticity and time-dependent plasticity on nanoindentation measurements of human cortical bone, *J Biomed Mater Res* 67A:208-214, 2003.
228. Vanleele M, Mazeran PE, Ho Ba Tho MC: Influence of strain rate on the mechanical behavior of cortical interstitial lamellae at the micrometer scale, *J Mater Res* 21:2093-2097, 2006.
229. Carter DR, Caler WE: A cumulative damage model for bone fracture, *J Orthop Res* 3:84, 1985.
230. Carter DR, Caler WE: Cycle dependent and time dependent bone fracture with repeated loading, *J Biomech Eng* 105:166, 1983.
231. Moore TLA, O'Brien FJ, Gibson LJ: Creep does not contribute to fatigue in bovine trabecular bone, *J Biomech Eng* 126:321-329, 2004.
232. Yamamoto E, Paul Crawford R, Chan DD et al: Development of residual strains in human vertebral trabecular bone after prolonged static and cyclic loading at low load levels, *J Biomech* 39:1812-1818, 2006.
233. Carter DR, Caler WE, Spengler DM et al: Fatigue behavior of adult cortical bone—the influence of mean strain and strain range, *Acta Orthop Scand* 52:481-490, 1981.
234. Carter DR, Caler WE, Spengler DM et al: Uniaxial fatigue of human cortical bone—the influence of tissue physical characteristics, *J Biomech* 14:461-470, 1981.
235. Gray RJ, Korbacher GK: Compressive fatigue behavior of bovine compact bone, *J Biomech* 14:461, 1981.
236. Swanson SAV, Freeman MAR, Day WH: The fatigue properties of human cortical bone, *Med Biol Eng* 101:112, 1979.
237. Lafferty JF, Raju PVV: The influence of stress frequency on fatigue strength of cortical bone, *J Biomed Eng* 101:112, 1979.
238. Schaffler MB, Choi K, Milgrom C: Aging and matrix microdamage accumulation in human compact bone, *Bone* 17:521-525, 1995.

WWW.HIGHDENT.IR
مرجع دندانسازان و دندانپزشکان

239. Burr DB, Forwood MR, Fyhrie DP et al: Bone microdamage and skeletal fragility in osteoporotic and stress fractures, *J Bone Miner Res* 12:6-15, 1997.
240. Pattin CA, Caler WE, Carter DR: Cyclic mechanical property degradation during fatigue loading of cortical bone, *J Biomech* 29:69-79, 1996.
241. Zioupos P, Wang XT, Currey JD: Experimental and theoretical quantification of the development of damage in bone and antler, *J Biomech* 29:989-1002, 1996.
242. O'Brien F J: *Microcracks and the fatigue behavior of compact bone* [doctoral thesis], Dublin, 2001, Trinity College and Royal College of Surgeons in Ireland.
243. Fleck C, Eifler D: Microstructure and fatigue behaviour of cortical bone. In *Proceedings of the Second World Congress of Biomechanics*, vol 2, Amsterdam, 1994.
244. Pattin CA, Carter DR, Caler WE: Cortical bone modulus reduction in tensile and compressive fatigue. In *Transactions of the 36th Annual Meeting of the Orthopaedic Research Society*, New Orleans, 1990.
245. Fleck C, Eifler D: Deformation behaviour and damage accumulation of cortical bone specimens from the equine tibia under cyclic loading, *J Biomech* 36:179-189, 2003.
246. Zioupos P, Currey JD: The extent of microcracking and the morphology of microcracks in damaged bone, *J Mater Sci* 29:978-986, 1994.
247. Schaffler MB, Choi K, Milgrom C: Microcracks and aging in human femoral compact bone, *J Orthop Res* 19:190, 1994.
248. Boyce TM, Fyhrie DP, Glotkowski MC et al: Damage type and strain mode associations in human compact bone bending fatigue, *J Orthop Res* 16:322-329, 1998.
249. Jepsen KJ, Davy DT, Krzypow DJ: The role of the lamellar interface during torsional yielding of human cortical bone, *J Biomech* 32:303-310, 1999.
250. O'Brien FJ, Taylor D, Lee TC: Microcrack accumulation at different intervals during fatigue testing of compact bone, *J Biomech* 36:973-980, 2003.
251. Hazenberg JG, Taylor D, Clive Lee T: Mechanisms of short crack growth at constant stress in bone, *Biomaterials* 27:2114-2122, 2006.
252. Mohsin S, O'Brien FJ, Lee TC: Osteonal crack barriers in ovine compact bone, *J Anat* 208:81-89, 2006.
253. Forwood MR, Parker AW: Microdamage in response to repetitive torsional loading in the rat tibia, *Calcif Tissue Int* 45:47-53, 1989.
254. O'Brien FJ, Taylor D, Clive Lee T: The effect of bone microstructure on the initiation and growth of microcracks, *J Orthop Res* 23:475-480, 2005.
255. Guo XE, McMahon TA, Keaveny TM et al: Finite element modeling of damage accumulation in trabecular bone under cyclic loading, *J Biomech* 27:145-155, 1994.
256. Schaffner G, Guo XE, Silva MJ et al: Modeling fatigue damage accumulation in two-dimensional Voronoi honeycombs, *Int J Med Sci* 42:645-656, 2000.
257. Makiyama AM, Vajjala S, Gibson LJ: Analysis of crack growth in a 3D Voronoi structure: a model for fatigue in low density trabecular bone, *J Biomech Eng* 124:512-520, 2002.
258. Moore TL, Gibson LJ: Fatigue of bovine trabecular bone, *J Biomech Eng* 125:761-768, 2003.
259. Moore TL, Gibson LJ: Fatigue microdamage in bovine trabecular bone, *J Biomech Eng* 125:769-776, 2003.
260. Moore TL, Gibson LJ: Microdamage accumulation in bovine trabecular bone in uniaxial compression, *J Biomech Eng* 124:63-71, 2002.
261. Igarashi K, Miyoshi K, Shinoda H et al: Diurnal variation in tooth movement in response to orthodontic force in rats, *Am J Orthod Dentofacial Orthop* 114:8-14, 1998.
262. Miyoshi K, Igarashi K, Saeki S et al: Tooth movement and changes in periodontal tissue in response to orthodontic force in rats vary depending on the time of day the force is applied, *Eur J Orthod* 23:329-338, 2001.
263. Yamada S, Saeki S, Takahashi I et al: Diurnal variation in the response of the mandible to orthopedic force, *J Dent Res* 81:711-715, 2002.
264. Qu Z: *Mechanical properties of trabecular bone in the human mandible* [master's thesis], Birmingham, Ala, 1994, University of Alabama.
265. Martens M, Van Audekercke R, Delport P et al: The mechanical characteristics of cancellous bone at the upper femoral region, *J Biomech* 16:971-983, 1983.
266. Morgan EF, Keaveny TM: Dependence of yield strain of human trabecular bone on anatomic site, *J Biomech* 34:569-577, 2001.
267. Hildebrand T, Laib A, Muller R et al: Direct three-dimensional morphometric analysis of human cancellous bone: microstructural data from spine, femur, iliac crest, and calcaneus, *J Bone Miner Res* 14:1167-1174, 1999.
268. Kopperdahl DL, Keaveny TM: Yield strain behavior of trabecular bone, *J Biomech* 31:601-608, 1998.
269. Linde F, Hvid I, Pongsoipetch B: Energy absorptive properties of human trabecular bone specimens during axial compression, *J Orthop Res* 7:432-439, 1989.
270. Lotz JC, Gerhart TN, Hayes WC: Mechanical properties of trabecular bone from the proximal femur: a quantitative CT study, *J Comput Assist Tomogr* 14:107-114, 1990.
271. Mosekilde L, Mosekilde L, Danielsen CC: Biomechanical competence of vertebral trabecular bone in relation to ash density and age in normal individuals, *Bone* 8:79-85, 1987.
272. Morgan EF, Yeh OC, Chang WC et al: Nonlinear behavior of trabecular bone at small strains, *J Biomech Eng* 123: 1-9, 2001.
273. Keaveny TM, Wachtel EF, Kopperdahl DL: Mechanical behavior of human trabecular bone after overloading, *J Orthop Res* 17:346-353, 1999.
274. Kopperdahl DL, Pearlman JL, Keaveny TM: Biomechanical consequences of an isolated overload on the human vertebral body, *J Orthop Res* 18:685-690, 2000.
275. Brown CU, Yeni YN, Norman TL: Fracture toughness is dependent on bone location—a study of the femoral neck, femoral shaft, and the tibial shaft, *J Biomed Mater Res* 49:380-389, 2000.
276. Bidez MW et al: A finite element model of an edentulous human mandible. In *Proceedings of the American Society of Mechanical Engineers Winter Annual Meeting*, Dallas, 1990.
277. Hart RT, Hennebel VV, Thongpreda N et al: Modeling the biomechanics of the mandible: a three dimensional finite element study, *J Biomech* 3:261-286, 1992.
278. Hylander WL: In vivo bone strain as a predictor of masticatory bite force in *Macaca fascicularis*, *Arch Oral Biol* 31:149-157, 1986.
279. Schwartz-Dabney CL, Dechow PC: Variations in cortical material properties throughout the human dentate mandible, *Am J Phys Anthropol* 120:252-277, 2003.
280. Peterson J, Wang Q, Dechow PC: Material properties of the dentate maxilla, *Anat Rec A Discov Mol Cell Evol Biol* 288:962-972, 2006.
281. Schwartz-Dabney CL, Dechow PC: Edentulation alters material properties of cortical bone in the human mandible, *J Dent Res* 81:613-617, 2002.

WWW.HIGHDENT.IR

282. Dechow PC, Hylander WL: Elastic properties and masticatory bone stress in the macaque mandible, *Am J Phys Anthropol* 112:553-574, 2000.
283. Bidez MW, Misch CE: Issues in bone mechanics related to oral implants, *Implant Dent* 1:289-294, 1992.
284. Linde F, Hvid I: The effect of constraint on the mechanical behavior of trabecular bone specimens, *J Biomech* 22: 485-490, 1989.
285. Misch CE: Bone density: its effect on treatment planning—surgical approach, healing and progressive bone loading, *Int J Oral Implantol* 6:23-31, 1990.
286. Runkle JC, Pugh JW: The micromechanics of cancellous bone. II. Determination of the elastic modulus of individual trabeculae by a buckling analysis, *Bull Hosp Joint Dis Orthop Inst* 36:2, 1975.
287. Wright TM, Hayes WC: Tensile testing of bone over a wide range of strain rates: effects of strain rate, microstructure and density, *Med Biol Eng* 14:671, 1976.
288. Brown TD, Ferguson AB: Mechanical property distributions in the cancellous bone of the human proximal femur, *Acta Orthop Scand* 51:429, 1980.
289. Carter DR, Schwab GH, Spengler DM: Tensile fracture of cancellous bone, *Acta Orthop Scand* 5:733, 1980.
290. Williams JL, Lewis JL: Properties and an anisotropic model of cancellous bone from the proximal tibial epiphysis, *J Biomech Eng* 104:50, 1982.
291. Stone JL, Beaupre GS, Hayes WC: Multiaxial strength characteristics of trabecular bone, *J Biomech* 16:743, 1983.
292. Goldstein SA, Wilson DL, Sonstegard DA et al: The mechanical properties of human tibial trabecular bone as a function of metaphyseal location, *J Biomech* 16:965, 1983.
293. Ryand SD, Williams JL: *Tensile testing of individual bovine trabeculae.* Proceedings of the Twelfth NE Bio-Engineering Conference, New Haven, Conn, March 13-14, 1986.
294. Kuhn JL, Goldstein SA, Choi K: The mechanical properties of single trabeculae, *Trans Orthop Res Soc* 12:48, 1987.
295. Mete PL, Lewis JL: Young's modulus of trabecular bone tissue, *J Orthop Res* 12:49, 1987.
296. Rice JC, Cowin SC, Bowan JA: On the dependence of the elasticity and strength of cancellous bone on apparent density, *J Biomech* 21:155, 1988.
297. Nicholson PH, Cheng XG, Lowet G et al: Structural and material mechanical properties of human vertebral cancellous bone, *Med Eng Phys* 19:729-737, 1997.
298. Kopperdahl DL, Keaveny TM: Yield strain behavior of trabecular bone, *J Biomech* 31:601-608, 1998.
299. Keaveny TM, Wachtel EF, Ford CM et al: Differences between the tensile and compressive strengths of bovine tibial trabecular bone depend on modulus, *J Biomech* 27:1137-1146, 1994.
300. Rohl L, Larsen E, Linde F et al: Tensile and compressive properties of cancellous bone, *J Biomech* 24:1143-1149, 1991.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 29

Density of Bone: Effect on Surgical Approach and Healing

Carl E. Misch

The density of available bone in an edentulous site has a primary influence on treatment planning, implant design, surgical approach, healing time, and initial progressive bone loading during prosthetic reconstruction.[1,2] A direct bone-implant interface has been demonstrated with a variety of endosteal implant designs and materials. Three requirements necessary for initial rigid fixation—atraumatic bone preparation,[3] close approximation of living bone to the biocompatible implant surface,[4] and absence of movement at the interface during healing[5]—are all closely dependent on the bone density at the implant site. The quality of the recipient bone directly influences the amount of trauma generated during osteotomy preparation. This in turn provokes a cascade of reactions at the bone-implant interface that affect the quality of the load-bearing surface.

Once the implant is initially integrated with the bone, the bone-loading process from occlusal forces becomes a critical factor in long-term implant survival. The bone density under load is directly related to the bone strength and is therefore a critical parameter for long-term survival.[6,7] The occlusal stresses applied through the implant to the bone must remain within the physiologic to mild overload zone; otherwise, pathologic overload with associated bone loss and microfracture, and implant mobility may occur. The treatment planning and scientific rationale of strength, modulus of elasticity, bone-implant contact percent, and stress transfer difference related to bone density has been addressed in a previous chapter. This chapter addresses the modifications of the surgical and healing aspects related to each bone density in the oral environment.

LITERATURE REVIEW

Lekholm and Zarb[8] listed four bone qualities found in the anterior regions of the jawbone: quality 1 comprises homogeneous compact bone; quality 2, a thick layer of compact bone surrounding a core of dense trabecular bone; quality 3, a thin layer of cortical bone surrounding dense trabecular bone of favorable strength; and quality 4, a thin layer of cortical bone surrounding a core of low-density trabecular bone. Irrespective of the different bone qualities, all bone was treated with the same implant design and standard surgical and prosthetic protocols.

Following the proposed protocols of Brånemark et al., it was found that implant survival in initial surgical success was related to the quality of bone.[4] A higher surgical failure was observed in softer bone types, especially in the maxilla. For example, Engquist[9] reported the surgical loss of 38 of 191 implants in the maxilla in D4 bone (20% loss), compared with 8 of 148 mandibular implants (5% loss) before stage II surgery. Jaffin and Berman reported an overall 8.3% surgical and initial healing loss in 444 maxillary implants with softer bone.[10] Friberg et al. reported a 4.8% implant failure at stage II uncovery for 732 maxillary posterior implants, which was greater than mandibular failure.[11] Quirynen et al. also reported a 4.1% implant loss at stage II uncovery out of 269 implants in the maxilla.[12] Fugazzotto et al. reported 22 failures out of 34 implants placed in quality 4 bone.[13] Hutton et al. identified poor bone quantity and quality 4 as the highest risk of implant failure in a study of 510 implants with an overall failure rate in the maxilla nine times greater than in the mandible.[14] Sullivan et al. indicated a 6.4% stage II failure rate in the maxilla (12/188) and a 3.2% failure in the mandible (7/216).[15] Snauwaert et al. reported more frequent early failures in poor density maxillae.[16] Herrmann et al. correlated failure factors such as poor bone quality and volume.[17] A number of reports in the literature demonstrated the greatest risk of surgical failure was observed in the softest bone type (D4), especially when found in the maxilla.

On the other extreme, a large clinical study from 33 U.S. Department of Veterans Affairs (VA) hospitals by the Dental Implant Clinical Research Group (DICRG) states quality 1 bone had the highest surgical failure rate

WWW.HIGHDENT.IR

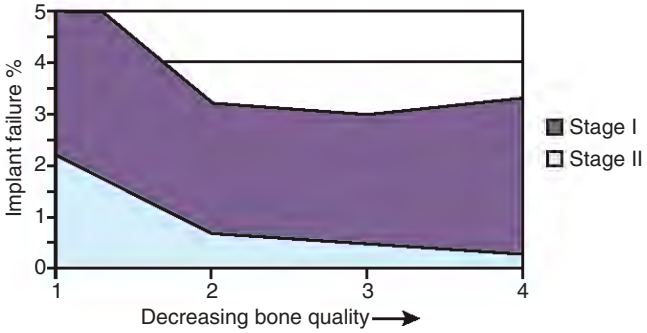


Figure 29-1 The Dental Implant Research Group represents 33 different hospitals that place dental implants. The highest surgical failure rate was quality 1 bone, followed by quality 4 bone. The fewest surgical failures were observed in quality 3 bone.

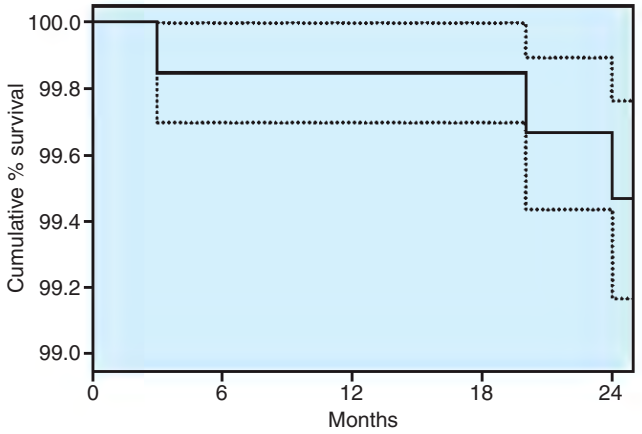


Figure 29-2 A multicenter study reported a surgical success of 99.6%, regardless of bone quality. *(Data from Misch CE, Hoar JB, Beck G et al: A bone quality based implant system: a preliminary report of stage I and stage II,* Implant Dent *7:35-42, 1998.)*

(4.3%), followed by quality 4 (3.9%), quality 2 (2.9%), and quality 3 with the fewest failures at 2.6% (Figure 29-1). The overall implant surgical failure was 3%; the maxilla had better success at stage II surgery (98.1%) than the mandible (96.4%).[18] It must be emphasized that these reports only present implant failures up to stage II uncovery. The DICRG also noted that the failure rate was twice as great for surgeons who had placed fewer than 50 implants, compared with more experienced surgeons. The literature contains many published reports that indicate an implant surgical failure range from 3.2% to 5% in the mandible and 1.9% to 20% in the maxilla, with most reports indicating the greatest failure rates in maxillary implants with soft bone. It is clear from these reports that a wide range of results may be achieved and therefore consideration should be given to methods that improve surgical survival.

Misch developed a different surgical protocol for different bone qualities in 1988. Following these specific methods, prospective and retrospective multicenter clinical studies in a wide range of office settings found surgical survival above 99%, regardless of the density type of bone, the arch (mandible versus maxilla), sex and age of the patient.[19-24] Therefore a different surgical protocol for different bone densities appears warranted.

The implant design, surgical protocol, healing, treatment plans, and progressive loading time spans are unique for each bone density type.[1,2,25] More recently the use of improved rotary instruments, implant designs, and surgical approaches for different bone qualities has been recognized as a valid recommendation. A multicenter prospective clinical study by Misch et al. of 364 consecutive implants in 104 consecutive patients found a surgical survival rate (up to abutment connection) at stage II of 100% for D1, 98.4% for D2, 99.8% for D3, and 100% for D4 implants[19] (Figure 29-2). Altering the surgical approach and implant design for each bone density can yield an overall implant surgical survival of 99.8% and a 2-year survival of 99.4%. In this chapter the surgical considerations and optimal healing time are discussed relative to each bone category, based on the literature, prospective clinical studies, and long-term experience.

BONE DENSITY CLASSIFICATIONS

Misch defined four bone density groups in all regions of the jaws that vary in both macroscopic cortical and trabecular bone types.[2] The regions of the jaws are divided into: (1) the anterior maxilla (second premolar to second premolar); (2) posterior maxilla (molar region); (3) anterior mandible (first premolar to first premolar); and (4) posterior mandible (second premolar and molars). The regions of the jaws often have similar bone densities.

In general, the anterior mandible is usually D2 bone; the posterior mandible, D3 bone; the anterior maxilla, D3 bone; and the posterior maxilla, often D4 bone. This generalization is used for the initial treatment plan. However, resorbed anterior mandibles may be D1 bone in approximately 25% of male patients and the posterior maxilla may have D3 bone after 6 months in the majority of sinus graft patients. The regional locations of the different densities of cortical bone are more consistent than the highly variable trabecular bone. Bone density may be most precisely determined before surgery by a computed tomography (CT) scan of the edentulous site (accompanied by Hounsfield values of the bone). Reformatted software allows "electronic surgery" of the CT images and relates the Hounsfield values at the implant-bone interface. It may also be grossly estimated by radiographic evaluation of tomograms. Conventional dental radiographs, such as periapical, panoramic, or lateral cephalometric images, are usually not diagnostic.

A common point at which to evaluate bone quality is during surgery. The presence and thickness of a crestal cortical plate and the density of trabecular bone are easily determined during implant osteotomy

WWW.HIGHDENT.IR

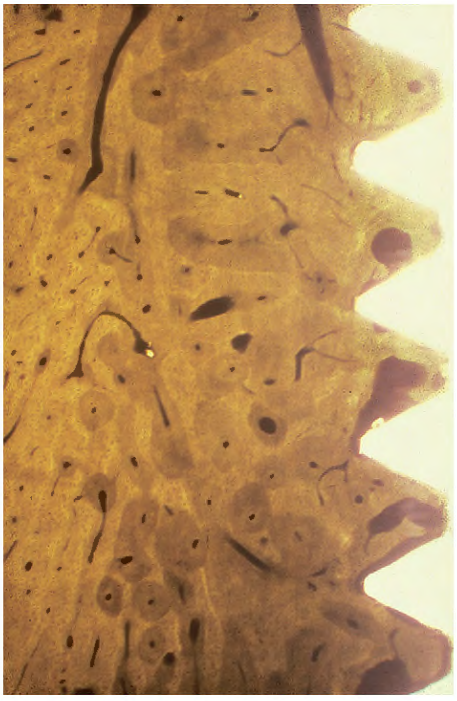


Figure 29-3 D1 bone has the highest bone-implant contact (BIC), which is usually greater than 80% after initial bone healing. Thus the strongest bone is also the bone with the greatest BIC. Both these conditions make D1 bone most suitable for occlusal loading.

preparation. The density of bone is determined by the initial bone drill, and evaluation continues until the final osteotomy preparation.

It should be emphasized that the bone density (D1 to D4) classification of Misch is slightly different from Lekholm and Zarb's bone quality types (Q1 to Q4). According to Misch et al., D3 bone has fine trabeculae and is 47% to 68% weaker than D2 trabeculae, and 20% stronger than D4 trabeculae, whereas Lekholm and Zarb stated that Q3 bone has favorable-strength trabeculae similar to Q2.[7,8] In other words, the actual strength of the trabecular bone is different for each bone density, regardless of the presence or absence of cortical bone adjacent to the implant. In addition, the Lekholm and Zarb bone quality only evaluated bone in the anterior maxilla and mandible. The Misch bone density scale also evaluated the posterior molar regions of the jaws. As a result, a primary difference between D3 and D4 bone is also the presence of cortical bone in D3, which increases its overall strength and modulus of elasticity.[6,7] The quality 4 bone of Lekholm and Zarb is similar to Misch's D3, whereas D4 bone is even weaker because little to no cortical bone is present to improve the strength or the elastic modulus of the fine trabecular bone.

Dense Cortical (D1) Bone

The very dense (oak- or maplelike) D1 bone is composed of almost all dense cortical bone. The maxilla almost never presents D1 bone. In Division A bone, approximately 4% of the anterior mandibles and 2% of the posterior mandibles have this dense bone category. In Division C–h bone of an anterior mandible, these numbers increase and may reach 25% in males, whereas weaker bone density D3 and D4 bone are less commonly encountered.

Advantages of D1 Bone

The homogeneous, dense D1 bone type presents several advantages for implant dentistry. Composed histologically of dense lamellar bone with complete haversian systems, it is highly mineralized and able to withstand great occlusal loads. The cortical lamellar bone may heal with little interim woven bone formation, ensuring excellent bone strength while healing next to the implant.[26,27] Four splinted, threaded, titanium plasma spray implants immediately loaded in the anterior mandible have yielded very predictable long-term success (above 94%), to support an overdenture.[28] Other screw-type implants inserted with a similar surgical and prosthetic approach also resulted in similar long-term survival in this bone region.[29]

D1 bone is more often found in anterior mandibles with moderate to severe resorption and greater crown/implant ratios. A threaded implant design provides greater surface area than a cylinder, especially in shorter lengths, and improves the dissipation of stresses in the crestal cortical region despite higher moments of force from the greater crown height, to sustain long-term functional stress.

The percentage of light microscopic contact of bone at the implant interface is greatest in D1 bone type and greater than 80% (Figure 29-3). In addition, this bone density exhibits greater strength than any other type.[6,7] The strongest bone also benefits from the greatest bone-implant contact (BIC). Less stresses are transmitted to the apical third of the implants than in other bone types. As a result, shorter implants can better withstand greater loads than in any other bone densities. In fact, the placement of longer implants may decrease surgical survival rates, as overheating during osteotomy preparation is a primary concern in this bone type. Greater heat is often generated at the apical portion of the osteotomy, especially when preparing dense cortical bone.[30]

Disadvantages of D1 Bone

Dense cortical bone also presents several disadvantages. The implant height is often limited to less than 12 mm in the atrophic mandible, and the crown height space is often greater than 15 mm. As a result, additional force-multiplying factors (such as cantilevers or lateral forces) are further magnified on the implant-prosthetic system. Stress-reducing factors may be incorporated in the prosthesis design to reduce these effects, not only on the bone, but also on the prosthetic components.

WWW.HIGHDENT.IR

Box 29-1 Implant Osteotomy Preparation in Dense Compact (D1) Bone

Overheating during Osteotomy

Abundant external or internal irrigation
Cooled saline irrigation
Intermittent pressure on drill
Pause every 3 to 5 seconds; keep irrigating
New drill designs, flutes, geometry
Incremental drill sequence (more drills; pass same drill more than once)

Blood Supply

Primarily from periosteum
Minimal reflection

Final Osteotomy Drill

Greater width
Greater height
Slower speed used

Bone Tap

Short of full osteotomy depth
Allows passive implant fit
Prevents internal implant-body/implant-bone interface microfracture
Removes drill remnants

Final Implant Placement at or above Bone Level

Unthread $^1/_2$ turn to relieve internal stresses

Slower Healing Rate

Lamellar bone
Fewer blood vessels
Five months to achieve mature interface

Stage II Uncovery

Three to 4 months
May often use immediate loading (when prosthesis is biomechanically stable)

D1 bone has fewer blood vessels than the other three types, and therefore it is more dependent on the periosteum for its nutrition. The cortical bone receives the outer one third of all its arterial and venous supply from the periosteum.[31,32] This bone density is almost all cortical, and the capacity of regeneration is impaired because of the poor blood circulation.[33] Therefore delicate and minimal periosteal reflection is indicated. When D1 density is present, the bone width is usually abundant. Fortunately, there are few occurrences when facial or lingual undercuts are observed with D1 bone densities, and flap reflection can be safely kept to a minimum. The precise closure of the periosteum and the overlaying tissue has been shown to help recover the blood supply and is encouraged.[34]

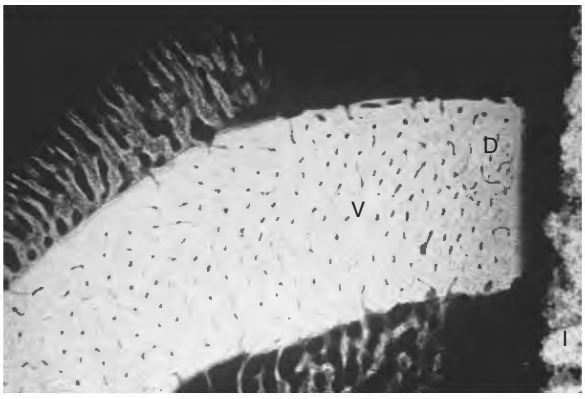


Figure 29-4 Devital zone *(D)* of bone next to the implant *(I)* is primarily created by heat generated during surgery, which radiates from the site, especially in cortical bone. Other contributing factors include lack of blood supply, pressure necrosis from implant placement, microfracture from bone tapping, and implant insertion. *V,* Vital bone.

Implant Osteotomy

The primary surgical problem of D1 bone is that the dense cortical bone is more difficult to prepare for endosteal implants than any other bone density (Box 25-1). The most common cause of implant failure in this bone quality is surgical trauma resulting from overheating the bone during the implant osteotomy procedures, because rotary drills progress with more difficulty. The zone of devitalized bone that forms around the implant is larger in this bone density and must be remodeled and replaced by vital bone for the interface to be load bearing (Figure 29-4). As a result, implant surgical failure may be greater in D1 bone than any other bone density.[2,18] Therefore the dental surgeon must strive to minimize the thermal trauma. The heat generated during an implant osteotomy is related to the presence and temperature of irrigation,[35-37] amount of bone being prepared,[38,39] drill sharpness and design,[40-43] time of preparation,[44] depth of the osteotomy,[37,30,41,45] pressure on the drill,[40] drill speed,[39,46,47] and variation in cortical thickness.[35,48]

Bone cell survival is very susceptible to heat. Eriksson has demonstrated that in rabbit, bone temperature as low as 3° C above normal (40° C) can cause bone cell necrosis.[49] Therefore a conscious effort is made to control temperature elevation every time a rotary instrument is placed in contact with bone. At least 50 mL/min of cooled irrigation, such as sterile physiologic saline, is used as a profuse irrigant and is a critical element to reduce heat.[35-37,50] Distilled water should not be used, as rapid cell death may occur in this medium.[51] Intravenous dextrose solution (D_5W) also may be used, with the clinical advantage of decreasing hand piece breakdown occurring from the effects of the salt in a saline solution, although the surgical gloves often feel sticky near the conclusion of the surgery. The irrigant also acts as a lubricant and removes bone particles from the implant osteotomy site. Without irrigation, drill temperatures

WWW.HIGHDENT.IR

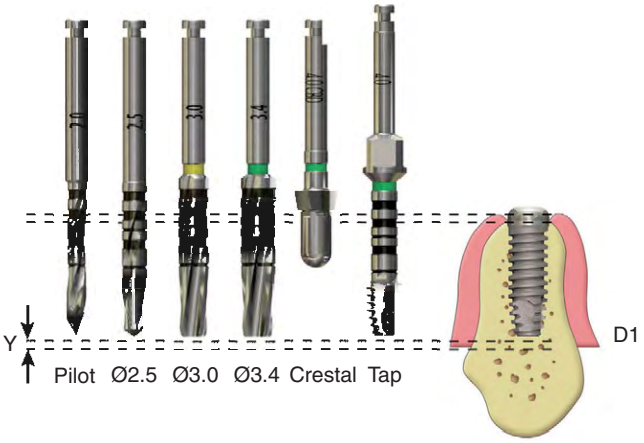


Figure 29-5 The number of steps in the preparation of the osteotomy is related to the bone density. D1 bone requires four drills, a crestal bone drill and a bone tap. D3 requires only three drills and no crestal drill or bone tap.

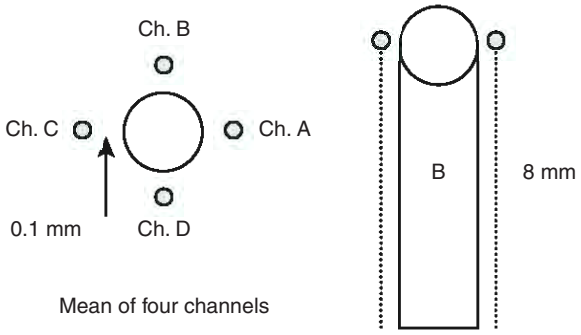


Figure 29-6 Thermocouples were positioned within 1 mm of the drill site and inserted into bone for a depth of 8 mm. The wires were connected to a computer to measure the temperature, time of preparation, and time the bone temperature was elevated.

above 100° C are reached within seconds during the osteotomy,[52] and consistent temperatures above 47° C are measured several millimeters away from the implant osteotomy. The temperatures of the irrigant can also affect the bone temperature.[36,39] Copious irrigation is suggested, especially in D1 bone.

The amount of heat produced in the bone is directly related to the amount of bone removed by each drill.[53] A 3-mm pilot drill generates greater heat than a 2-mm pilot drill.[39,54] As a result, most manufacturers suggest the first drill be 2 mm in diameter. In a similar fashion, the amount of heat generated by successive drills is also directly related to the increase in drill diameter.[52,43] A 3-mm drill after a 2-mm drill cuts 0.5 mm on each side of the drill. A 2.5-mm drill after a 2-mm drill only cuts 0.25 mm of bone on each side of the osteotomy. The smaller incremental drill size allows the surgeon to prepare the site faster, with less pressure and less heat.[42] In addition, when large increases in drill diameter are used to prepare bone, the surgeon may inadvertently change the angulation of the drill, because the larger drill is removing a greater bone volume and the tactile sense is decreased. As a result, an elliptical osteotomy

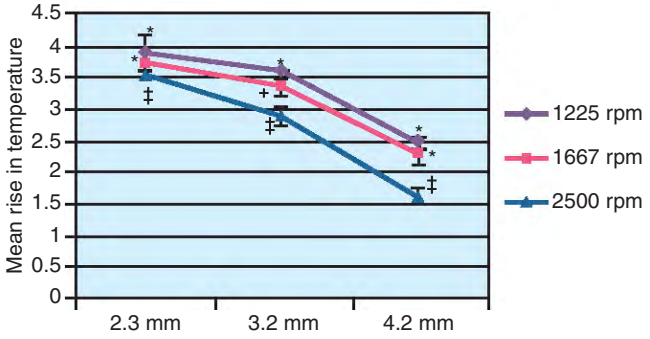


Figure 29-7 The internally cooled drill of the Paragon implant system recorded 41° C with the first drill at 1225 rpm. A rpm of 2500 reduced the bone temperature preparation for all drill diameters.

may be prepared that does not correspond accurately to the round implant diameter. The gradual increase in osteotomy size also reduces the drill shatter at the crestal opening, which can inadvertently chip away pieces of bone on the crest where complete bony contact is especially desired. The gradual increase in drill diameter also keeps each drill sharper for a longer period, which also reduces the heat generated (Figure 29-5).

In the past, the rotational speed of the drill has been suggested to be less than 2000 rpm, and several manufacturers have recommended speeds al low as 750 rpm. It has been an accepted theory that the higher the speed, the higher the bone temperature during preparation.[4] This is false; otherwise, slow-speed hand pieces would be used to prepare natural teeth. A high-speed hand piece (300,000 rpm) can remove bone over an impacted tooth or during an apicoectomy and still allow bone regeneration. Rafel prepared bone at 350,000 rpm in a human mandible and found a temperature of only 23.5° C at a distance of 3 mm from the drill periphery.[47] High-speed drills at 300,000 rpm have been used to prepare blade implant osteotomies, yet bone grew over the blade shoulder and was in direct contact with the implant.[55]

A study by Sharawy et al. compared four drill designs (two internal irrigated and two external irrigated) at speeds of 1225, 1667, and 2500 rpm.[39] Thermocouples connected to a computer to record temperature and time were placed within 1 mm of the osteotomy site in D2-type bone (Figure 29-6). All drill designs in the study recorded lower bone temperatures with the greatest rpm and, conversely, found the highest bone temperatures with the lowest rpm (Figures 29-7 and 29-8). As important, the slowest rpm resulted in bone temperatures at or above 40° C, which may be a threshold of bone-cell death. The highest rpm (2500) increased the bone temperature by 2° to 3.5° C, whereas the 1225 rpm recorded a bone temperature above 41° C. Therefore the higher speed of 2500 rpm may prepare bone at a lower temperature than 1500 rpm, especially when in dense bone.[39] The rotational speed of the drill is one of the more critical criteria to reduce bone temperatures.

WWW.HIGHDENT.IR

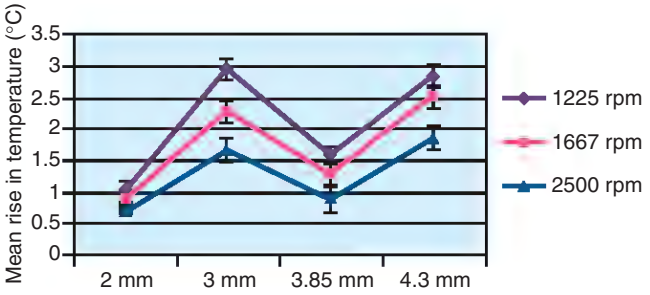


Figure 29-8 The externally cooled drills of the Brånemark implant system recorded reduced temperatures at 2500 rpm compared with slower speeds.

Eriksson reported bone cell death when a temperature of 40° C was applied for 7 minutes, or when a temperature of 47° C was applied for 1 minute.[49] In other words, time and temperature are interrelated critical factors in implant site preparation. As the temperature increases, the time the bone temperature is elevated must be reduced. In the study by Sharawy et al., the time the bone temperature remained elevated was recorded for each rpm evaluated.[39] When the drill prepared an 8 mm depth osteotomy, the temperature remained elevated for 45 to 58 seconds (Figure 29-9). The slower the rpm (1225), the longer the bone temperature remained above the baseline. Because two to three drills are used to prepare an implant site, at 1225 rpm the first drill may increase the temperature to 41° C, the second drill to 45° C, and the third drill to 49° C, when the time between each sequence is not extended more than 1 minute. Therefore, after each drill is used within an osteotomy, a 1-minute pause should be observed before the next drill. In the Sharawy et al. study, the first drill diameter recorded the longest preparation time and the highest temperature, and the longest recovery time.[39] Therefore to reduce the preparation time within the bone to a minimum in D1 bone, the surgeon should not apply constant pressure to the drill, but "bone dance" with intermittent pressure for 1 second in the D1 bone and 1 to 2 seconds out of the bone while the cooled irrigation is allowed to perfuse the site. The surgeon should also pause approximately every 5 to 10 seconds of bone preparation for 1 minute to allow the limited blood supply access to the surgical site, permit dissipation of heat, irrigate the area to reduce bone temperatures, and remove debris from the osteotomy site.

The pressure used against the bone should neither be so hard as to stall the drill, nor so light that the drill only creates heat and does not prepare the bone. Hobkirk and Rusiniak found that the average force placed on a hand piece during preparation of an osteotomy is 1.2 kg.[48] Matthews and Hirsch concluded that the force applied to the hand piece was more influential than the drill speed in temperature elevation.[40] When the pressure on the hand piece was increased appropriately, drill speeds from 345 to 2900 rpm did not affect the temperature. They found that increasing both speed and pressure allowed the drill to cut more efficiently and generated less heat. The effect of drill speed and pressure related to bone temperature was also reported by Brisman.[46] In cortical bone, speeds of 1800 rpm with a load of 1.2 kg produced the same heat as when speed increased to 2400 rpm with pressure of 2.4 kg. The greater speed and greater pressure was more efficient than low speeds. Increasing pressure alone increased heat; increasing speed alone also increased heat. Different amounts of pressure are therefore used in response to the density of the bone. Sufficient pressure should be used on the drill to proceed at least 2 mm every 5 seconds. If this is not achieved, new (sharper) or smaller-diameter drills are indicated for each site preparation. The pressure on the drills should not reduce the rpm, which makes the drill less efficient and increases heat. Hand pieces of sufficient torque should be used to prevent this complication.

Lavelle has demonstrated lower drilling temperatures in cortical bone with internally irrigated drills.[56]

Figure 29-9 The internally cooled drills of the Nobel Biocare, Steri-Os implant system demonstrate the temperature in the bone remains elevated for an extended period (up to 58 seconds) after site preparations in D2 bone. The lower the rpm, the longer the temperature remains elevated.

WWW.HIGHDENT.IR

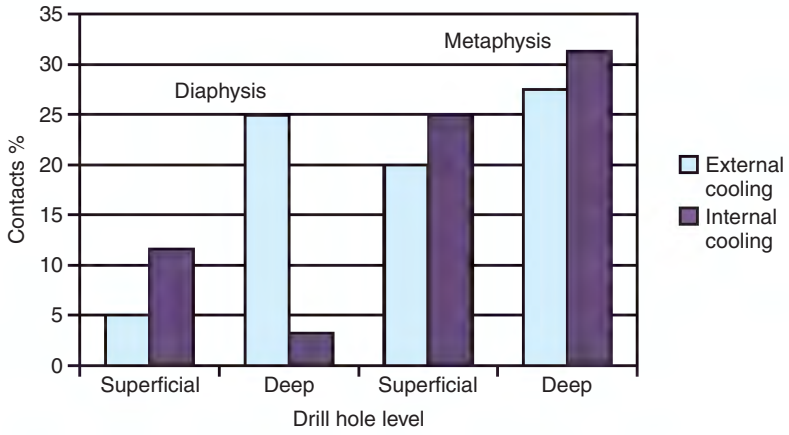


Figure 29-10 The externally irrigated drill had more bone-implant contact (BIC) after 4 weeks of healing in trabecular bone (metaphysics) and for shallow osteotomies in cortical bone (diaphysis). The internally irrigated drill had a higher BIC in deeper osteotomies of cortical bone.

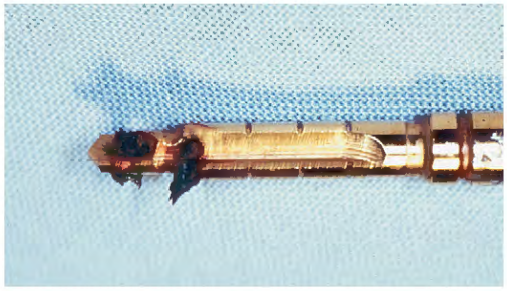


Figure 29-11 Organic debris from a previous surgery may be retained in the chamber of an internally irrigated drill.

Thus many manufacturers suggest that an internally irrigated drill be used during implant osteotomy preparation. However, the bone temperature in the study by Sharawy et al. compared two different internal irrigated drill systems (Zimmer and Nobel Biocare) with two different external irrigated systems (Nobel Biocare and BioHorizons).[39] The two external irrigated drill systems prepared the bone osteotomy at lower bone temperatures than the two internal drill systems. The difference in the bone temperatures between the four systems ranged from 1° to 3° C (depending upon the drill diameter). This study did not use the same drill design when comparing internal versus external irrigation. Therefore, the difference in bone temperature elevation may be related more to drill design than to the method of irrigation. Benington et al. tested internal and external irrigation systems on bovine bone. At a constant load of 1.7 kg, no difference was found between the two designs.[43]

Internal irrigation of drills may present several disadvantages. The portal of the internal-cooled drill that exits at the drill apex often becomes clogged once the drill is in the bone deeper than 5 mm.[57] As a result, no irrigant escapes the portal and the drilling must be repeatedly stopped to remove debris. The pressure required during drilling dense bone also may completely prevent saline from flowing out of the drill in deeper preparations, with bone debris obturating the portal of the irrigation chamber. In a histologic study by Watzek et al., the BIC percentages between internal and external irrigated drill systems in cortical and trabecular bone were compared 4 weeks after implantation. In cortical bone, the BIC was highest in internal irrigated drills when the osteotomy was deep. In trabecular bone or less deep cortical sites, the external by irrigated dills recorded greater BIC values[45] (Figure 29-10).

Complete cleaning of the internal chamber of the internal irrigated drill is virtually impossible, and the drill usually retains organic contaminants in the chamber from a previous surgery[57] (Figure 29-11). Although these organic remnants may be sterilized as the drills are autoclaved after each surgery, it seems unwise to allow organic debris to be transmitted from one patient to another. The external irrigated drill may be sterilized for a following surgery. In addition, the internally cooled drill fabrication in smaller diameters poses technical problems and may cause fracture during use in more dense bone. The externally irrigated drill is also less expensive to fabricate and can be made in smaller diameters.

The externally irrigated drill also presents disadvantages. The external irrigant may not be applied at the bone-cutting surface, but inadvertently on the shank of the drill or on the external aspects of the bone. Some external irrigated systems require a surgical assistant to spray the surgical site while the dental surgeon prepares the osteotomy. It is beneficial when the irrigation portal is attached to the head of the hand piece. The irrigation may not adequately flood the surgical site. As the depth of the osteotomy increases, the risk of the inadequate irrigation increases.[30,45] Either internal or external irrigating drilling techniques (or a combination of the two) may be used satisfactorily, with understanding

WWW.HIGHDENT.IR

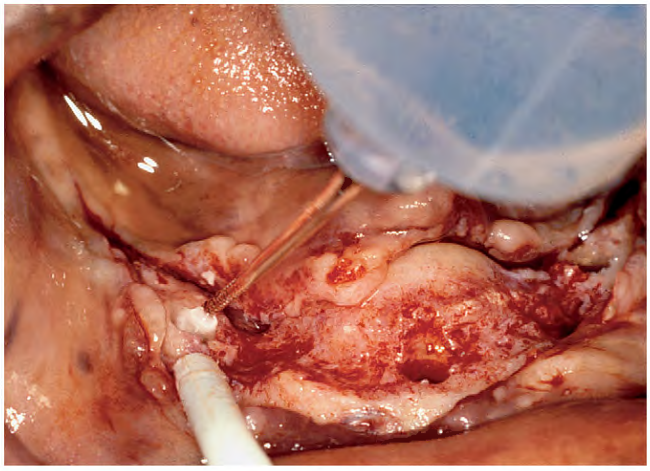


Figure 29-12 Bone chip debris should be frequently removed by irrigation in D1 bone to improve efficiency of the following drill and before implant insertion after bone tapping.

Figure 29-13 The bone debris in the drill should be evaluated. A brown or beige color indicates the temperature is too high and the bone is devital.

Figure 29-14 The bone debris in the drill should be reddish, which indicates vital bone and ideal preparation conditions.

of the limitations. Irrigation, drill design, rpm, and drill sizing are paramount to reduce heat, not whether internal or external irrigation is used.

Bone chips in the osteotomy should be frequently removed by irrigation in dense bone to maintain optimal cutting action (Figure 29-12). In addition, the bone debris should be frequently wiped off the cutting flutes of the drill with a surgical sponge. These bone shavings prevent coolant from reaching the bone and make the drill less efficient. The color of these bone shavings is important to evaluate. Any beige coloration to the bone debris indicates excessive heat is being generated and the bone debris is nonvital (Figure 29-13). A brownish color indicates the bone cell death extends several millimeters away from the implant osteotomy. The color of the bone debris should be reddish, which indicates vital bone (Figure 29-14). As a general rule, the first drill introduced into the implant osteotomy will have beige-colored bone debris. However, the last drill in the sequence should have reddish bone within the cutting flutes.

The use of new drills with a sharp cutting flute is most critical for D1 bone surgery. Bone drills become dull after repeated use. Chacon et al. evaluated three different drill systems after repeated drilling and sterilization. The bone temperature 0.5 mm from the osteotomy preparation increased every 25 uses of the system, even though light microscopic evaluation showed little wear.[42] Many manufacturers suggest the drills be reused, without attention to longevity of the cutting surface. The doctor may inadvertently use the same drills for hundreds of implant osteotomies. When the drills become dull, the doctor may not appreciate it in softer bone, but when D1 bone is prepared, the drill sharpness can become critical. Another method to improve the sharpness of the drill in D1 bone may be the use of coatings. However, not all coatings are of benefit. In a study by Ercoli et al., titanium nitride-coated drills (SteriOss and Paragon) showed greater wear and significantly lower bone removal rates than noncoated drills.[37] Diamond coatings, although expensive, may add longevity to the drill sharpness and are suggested for the initial drills used for the D1 bone osteotomy.

Reduced bone temperature is the primary method to decrease the incidence of surgical healing failure. A secondary cause of failure may be related to mechanical trauma of the bone. There are several methods to reduce mechanical trauma in D1 bone, and one of these is related to final drill size selection. In D1 bone, the final bone preparation may be sized slightly larger in both width and height, especially for a threaded implant, than the manufacturer-recommended surgical protocol. This reduces the risk of microfracture trauma between the implant threads during insertion, which may lead to fibrous tissue formation at the bone-implant interface. In addition, a final drill dimension only used in D1 bone remains sharper for this critical step. If a drill of slightly greater diameter is not available with the implant system, the implant surgeon can use the final drill size available and pass it within the osteotomy several times. This is an effective method to slightly oversize the site.

Most implant designs have a larger crest module compared with the body of the implant. This design feature ensures a bone "seal" around the top portion of the implant after it is threaded into position. The crest

WWW.HIGHDENT.IR

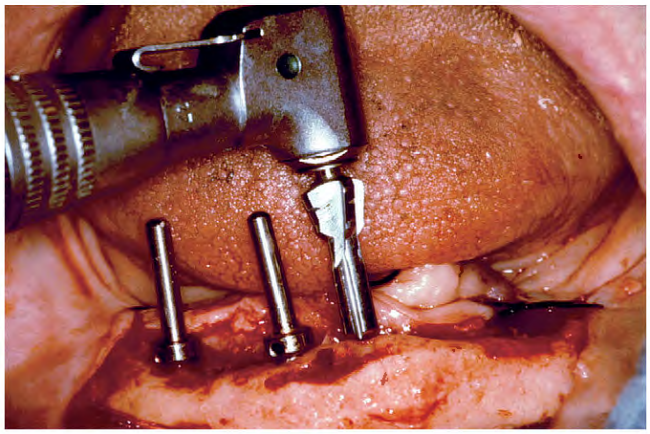
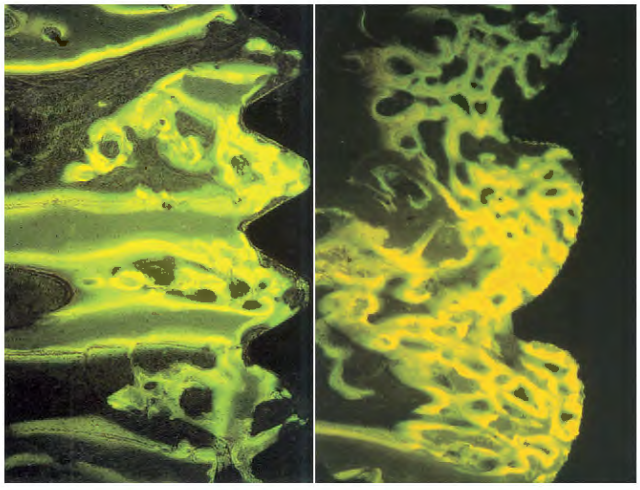


Figure 29-15 A crestal bone tap should be used in D1 bone to prepare marginal bone to receive the crest module of the implant body, which is larger in diameter than the implant body.



A **B**

Figure 29-16 **A,** A pretapped bone site has less woven (newly generated) bone and illustrated less trauma upon implant insertion. **B,** A self-tapping implant insertion causes greater bone trauma and exhibits massive woven regenerative bone formation as a consequence. *(From Watzek G, Danhel-Mayhauser M, Matejka M et al:* Experimental comparison of Brånemark and TPS dental implants in sheep *[abstract]. Presented at UCLA Symposium: Implants in the partially edentulous patient, Palm Springs, Calif, 1990.)*

module is usually 4.1 mm for a 3.75-mm-diameter implant. Because the final osteotomy drill of many systems is in the 3.2-mm-diameter range, the 0.9-mm difference is substantial, especially in crestal cortical bone. As a result, a crestal bone drill is used in D1 bone, which prepares the larger diameter at the top of the osteotomy (Figure 29-15). In a study by Novaes et al., the difference in crestal bone loss after an initial healing period of 3 months was 1.5 mm between using a crestal drill compared with no crestal drill.[58] The additional bone trauma from compressing a larger crest module into the osteotomy is significant and may increase surgical bone loss around the implant. Therefore a

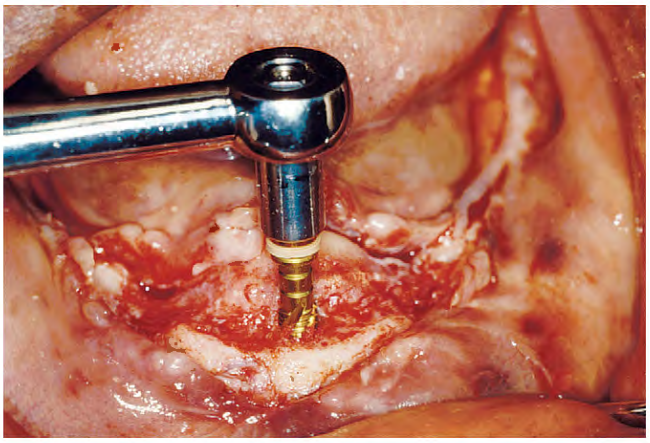


Figure 29-17 A hand wrench and bone tap should be used to prepare the threads for the implant when the bone is D1. A handpiece may stall or break down when attempting to tap the bone in this density.

crestal bone drill should be used in D1 bone as the last drilling step in the preparation of the osteotomy.

A bone tap should be used in D1 bone before insertion of a threaded implant. There are several reasons for the use of a bone tap. Because the final drill osteotomy is almost 1 mm smaller than the outer diameter of the implant, the bone tap creates the space for the thread of the implant. This device has open flutes, which permit the shaving of the bone to accumulate and be removed before placing the implant. A self-tapping implant insertion compresses the bone in the region of the threads. This is an advantage in softer bone types, but not in cortical bone. The tap reduces the mechanical trauma to the bone while the implant is inserted. The bone is also able to slightly recover from the trauma of the tap once it is removed and permits a more passive implant placement. Watzek et al. found a higher woven bone interface (a sign of bone trauma) when a self-threading implant design was used, compared with a pretapped implant site[59] (Figure 29-16, *A*, *B*). Satomi et al. found a higher BIC after initial healing with pretapped implant osteotomies compared with a self-tapping site, also indicative of less bone trauma.[60] The use of a self-tapping implant insertion technique in dense bone qualities has demonstrated a significantly higher degree of hard tissue trauma and is therefore not recommended in D1 bone.[59,60]

The bone tap should be used with a hand ratchet and irrigation (Figure 29-17). The slow-speed, high-torque hand piece is very efficient and has several advantages in D2 bone. However, D1 bone is so strong that the hand piece gears may strip, and the hand piece is more likely to require repeated repair.

The hand position of the surgeon is important in maintaining constant force and direction on the hand ratchet during the bone-tapping process. When using a ratchet, the horizontal rotation on the tap causes it to tip back and forth around the vertical axis. Therefore,

WWW.HIGHDENT.IR

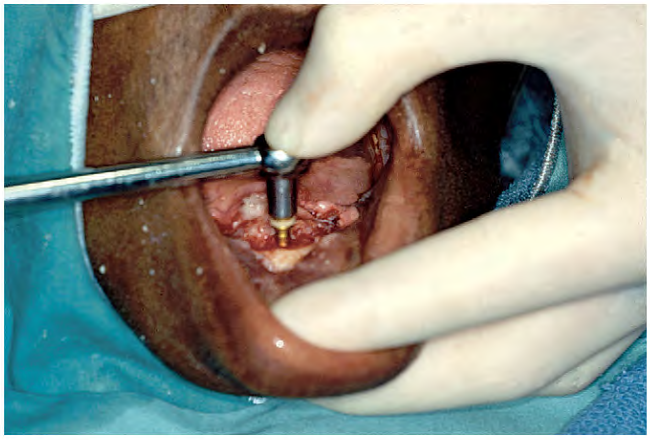


Figure 29-18 The hand position in D1 bone of an anterior mandible is demonstrated. One hand uses a thumb on the wrench with pressure in the long axis of the osteotomy; a forefinger retracts the lip; and the middle finger is placed under the chin and directs the tap in the apical direction.

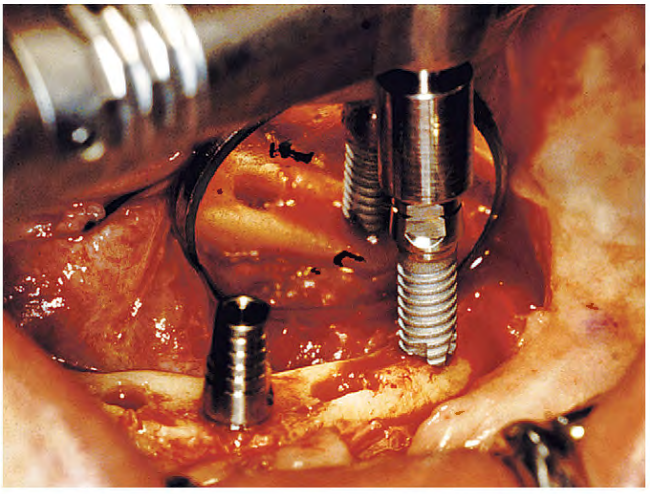


Figure 29-19 A high-torque, low-speed handpiece may thread the implant into the pretapped osteotomy at 30 rpm.

when using a ratchet, the ratchet is held while the thumb of the other hand is placed directly over the bone tap; the index finger of the same hand retracts the lip for improved access and vision; and the middle finger is placed under the mandible in the direct path of the osteotomy. The ratchet rotates the tap with one hand while the thumb and middle finger of the other hand apply constant pressure and direction to the tap, so it does not tip back and forth or strip the osteotomy site (which may happen if the tap does not continue to advance within the osteotomy each turn of the tap) (Figure 29-18).

A bone tap in D1 bone prevents the antirotational component of the implant body from being damaged during implant insertion in this dense bone type. A minor advantage to tapping may be the fact that drill remnants are more likely to be left in the implant osteotomy during preparation in dense bone or with a new drill and cutting edge.[56] The bone tap may remove these remnants and decrease the risk of long-term corrosion from dissimilar metals contacting within the bone, although no reports in the literature have indicated this to be a problem.

Once the tapping process is complete, the osteotomy is irrigated and suctioned. The implant may be inserted with a slow-speed, high-torque hand piece or a hand ratchet (Figure 29-19). The implant should not be tightened with a high torque pressure (>75 N-cm) to the full depth of the osteotomy; this causes it to "bottom out" and may set up microfractures along the implant interface (Figure 29-20). Instead, once the threaded implant is introduced into the osteotomy and in final position, it is often unthreaded one half turn to ensure that there is no residual pressure along the bone interface. This step is primarily used in D1 bone, because excessive initial strain may form at the interface of the cortical bone with even one extra rotation of the implant.[61] The

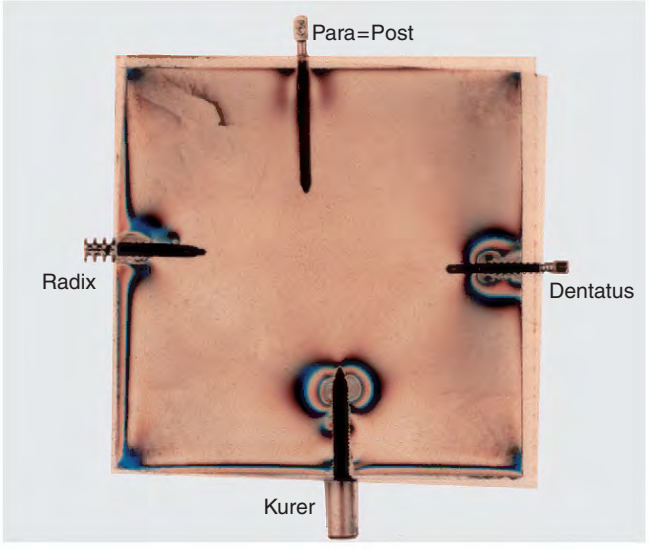


Figure 29-20 The implant should not be tightened into position, which may increase the strain to the surrounding bone. In this in vitro study, self-threading posts were placed in a dentine simulant (similar to cortical bone strength) in the 3, 6, and 9 o'clock positions. In the 12 o'clock position is a predrilled passive post insertion.

rotational stress is highest at the crestal region, which may even cause mechanical bone microfracture and marginal bone loss.[62,63]

The ideal implant length for D1 bone is 12 mm for a 4-mm-diameter implant. There is little, if any, benefit to increased implant length beyond 12 mm in D1 bone for a threaded implant body, as most all the stresses after healing are limited to the crestal half of the implant with occlusal loading. The longer implant makes bone preparation more difficult and generates more heat in this bone type.[37,30,45]

The final placement of the implant in relation to the crest of the ridge is related to its design and the bone density. A one-stage surgical approach is often used in D1 bone. The implant may be placed so that the external

WWW.HIGHDENT.IR

Figure 29-21 The implant in D1 bone may be placed with the external hex above the bone. A one-stage surgical approach is often used.

Figure 29-22 D2 bone has a dense to porous cortical crest, and inner trabecular bone is coarse. It is found most often in the mandible.

hex (or smooth crest module) is above the bone (Figure 29-21). A permucosal element (PME) may be added and permit the implant to heal above the soft tissue, thus eliminating a second-stage surgery. The crest module of the implant is often smooth for approximately 0.5 to 2 mm and provides minimum support to the implant system once it is loaded. The D1 dense compact bone is often of decreased height. Therefore the actual support system of the implant may be increased in division C–h limited-height bone type by not countersinking the smooth portion of the implant crest module below the crest of the ridge (see Figure 29-21). The smooth portion of the implant body may be placed above the ridge if no load is applied to the implant during initial healing, and the risk of micromovement during this period is minimal. A crestal drill is used corresponding to the final implant placement, often short of the final depth, to permit placement of the smooth crest module above the crestal bone.

It should be noted that this approach to increase implant body surface area within the bone is beneficial only when the implant body extends the entire height of available bone and only the smooth portion of the implant crestal module is above the bone. For example, for 11-mm bone height, a 13-mm threaded implant with a 2-mm smooth crest module may be used, leaving the 2-mm smooth crest module above the bone. There may be an increase of more than 35% surface area for load resistance in this example because this increases the implant design load to all 11 mm of bone, rather than using a 10-mm implant with 8 mm of thread. However, if a 10-mm implant were placed with the crest module above the bone, the same bone implant area would be obtained as the 10-mm implant level with the bone, once crestal bone height was lost from the biological width and loading.

Bone Healing

Many of the cutting cones that develop from monocytes in the circulating blood and are responsible for bone remodeling at the implant interface come from the blood vessels found in well-vascularized trabecular bone, which has a greater capacity for regeneration than compact bone. Therefore in some aspects cortical bone requires greater healing time compared with trabecular bone.[33]

On the other hand, because of the load-bearing capability of D1 bone and the excellent bone-implant contact, prosthetic loading of D1 bone may start before the completion of the initial healing phase. Conditions that contribute to a lack of movement during healing are primordial to achieve a direct bone-implant interface. D1 bone is strong and often able to resist micromovement regardless of whether an implant is loaded. As a result, immediate implant loading is often possible when multiple implants are splinted together, without compromise to the overall survival rate of the implant.[64,65] However, it is usually safer to wait for a longer healing period rather than immediate load, unless some other treatment plan criteria affects the decision. Most often, a blend of treatment conditions result in a minimum 3-month unloaded healing period in this bone type.

Once the bone-implant interface is established, it exhibits the strongest load-bearing properties of any bone type. As a result, progressive bone loading is not necessary to develop a stable condition. The restoring dentist may proceed as rapidly as desired to the final prosthesis.

Dense-to-Thick Porous Cortical and Coarse Trabecular Bone (D2)

The second density of bone found in the edentulous jaws (D2) is a combination of dense-to-porous cortical bone on the crest and coarse trabecular bone on the inside (Figure 29-22). The Hounsfield values on reformatted CT images are 750 to 1250 units for this bone quality. The tactile feeling when preparing this bone density is similar to preparations in spruce or

WWW.HIGHDENT.IR

white pine wood. The D2 bone trabeculae are 40% to 60% stronger than D3 trabeculae. This bone type occurs most frequently in the anterior mandible, followed by the posterior mandible. On occasion it is observed in the anterior maxilla, especially for a single missing tooth, although the dense-to-porous cortical bone is then found primarily on the lingual surface of the implant site.[66] The minimum ideal implant height for a V-thread implant design in D2 bone is usually 12 mm, for a 4-mm-diameter implant.

Advantages of D2 Bone

D2 bone provides excellent implant interface healing, and osteointegration is very predictable. Most implant systems refer to this density of bone for their usual surgical protocol (Figure 29-23). The dense-to-porous cortical bone on the crest or lateral portions of the implant site provide a secure initial rigid interface. Osteoplasty to gain additional width of bone before implant placement or countersinking below the crestal bone does not compromise support because the lateral cortices and coarse internal trabecular bone provide rigid fixation. The implant may even be placed slightly above the crest of the ridge with decreased compromise or risk of movement at the interface during healing, compared with softer bone types. The intrabony blood supply allows bleeding during the osteotomy, which helps control overheating during preparation and is most beneficial for bone-implant interface healing.[33]

Implant Osteotomy

The rotations of the drill during the implant site preparation should be approximately 2500 rpm. Sharawy et al. demonstrated that, regardless of the drill design or method of irrigation, 2500 rpm prepared bone at a lower temperature than slower speeds.[39] Copious cooled sterile saline irrigation is used during the bone preparation. The surgeon should allow the cutting surface of the drill to contact D2 bone fewer than 5 of every 10 seconds. Ideally, a pumping up-and-down motion is used to prepare the osteotomy and provide constant irrigation to the drill cutting surface. It also maintains a constant drill speed and reduces the friction time against the bone, all of which reduce heat. The surgical assistant counts for 5 seconds while the drill is progressing in the bone. When the surgeon stops or lifts the hand piece for more than a few seconds, the counting may begin again. If the assistant counts to 5 seconds and the drill is still in the bone, a gentle touch on the back of the surgeon's hand is a reminder to pause a few moments before proceeding. The irrigation should continue while the pause occurs. Not only can this method decrease heat, but constant irrigation flushes debris out of the osteotomy. The time in contact with bone and the amount of irrigation are two very important factors to reduce thermal damage to the bone.

The drill sequence for D2 bone is similar to D1 bone. A 2.0-mm twist drill is used to initiate the process, followed by a 2.5-mm twist drill. The final osteotomy diameter of the implant determines the final drill used. A 3.75-mm implant body most often uses a 3.0-mm drill and a bone tap or a 3.2-mm drill for a self-tapping implant insertion. A 4.0-mm implant body often uses a 3.4-mm drill for the final osteotomy preparation. The heat generated is less with a three-drill sequence (e.g., 2.0 mm, 2.4 mm, 3.2 mm) compared with a two-drill sequence (e.g., 2.0 mm, 3.2 mm) because less bone is removed with each drill in the prior scenario.

A crestal bone drill should be used for most implant designs in D2 bone (Figure 29-24). Because cortical bone is present on the crest of the ridge and the crest module of the implant enlarges to 4.1 mm, the crestal bone drill reduces the mechanical trauma to the bone upon implant insertion. Novaes et al. found a median average 1.5 mm less marginal bone loss after a 3-month healing period when a crestal bone drill was used, compared with implant insertion with no crestal drill

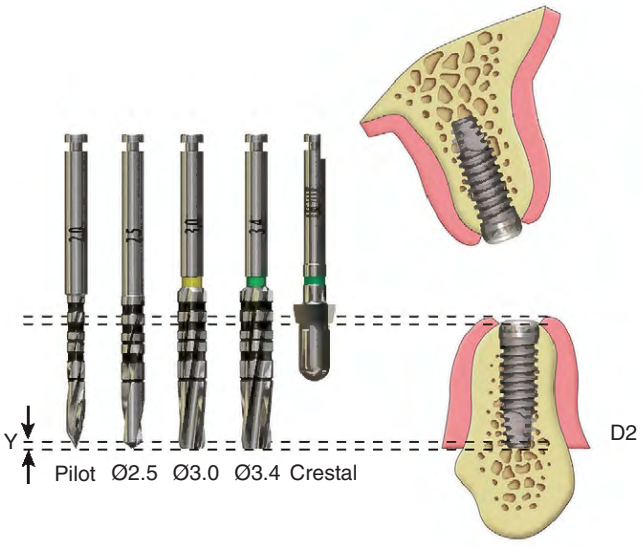


Figure 29-23 The standard drill protocol used by manufacturers is usually osteotomy preparation in D2 bone.

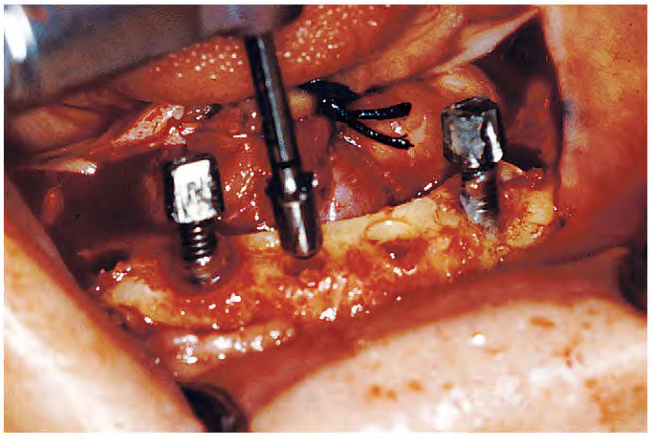


Figure 29-24 A crestal drill is used in D2 bone to prepare the cortical bone for the implant crest module, which is wider than the implant body.

WWW.HIGHDENT.IR

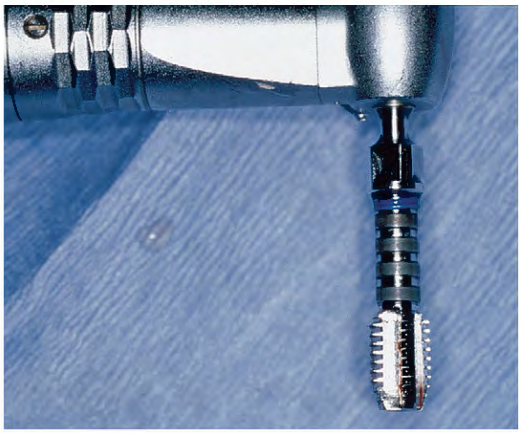


Figure 29-25 A bone tap is used with a high-torque, low-speed hand piece in D2 bone, when the thread depth is deep or the thread shape is square.

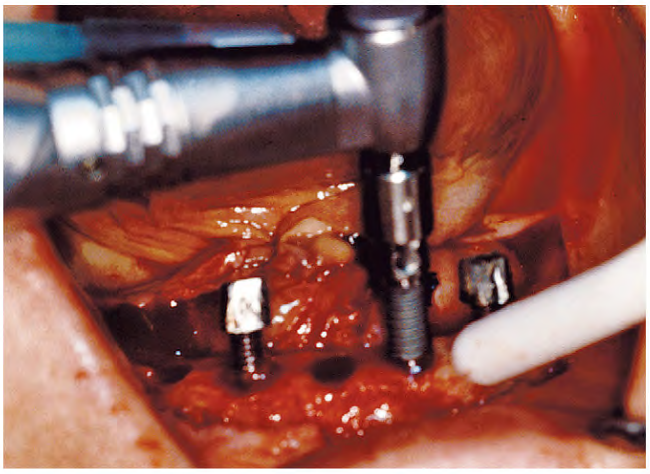


Figure 29-26 A high-torque, low-speed handpiece is used to thread the implant into the pretapped osteotomy in D2 bone.

after the final osteotomy preparation.[58] Sharawy et al. showed that D2 bone with an osteotomy depth of 8 mm could be prepared in 4 to 8 seconds, dependent upon drill design and rpm.[39] Therefore the osteotomy depth should not proceed slowly, creating additional heat. Enough pressure should be placed on the handpiece to proceed at least 5 mm every 5 seconds.

A bone tap may be used when the implant body engages the lateral or apical cortical bone. A hand piece at 30 rpm may be used to tap the D2 bone. This process is faster than using a hand ratchet as in D1 bone, and it is more precise because the direction and advancement of the tap is more precise with a hand piece. Irrigation is used during this process (Figure 29-25).

The use of a bone tap for D2 bone is dependent upon the final osteotomy size, the implant body size, the depth of the thread, and the shape of the thread. When the final osteotomy is 3.0 mm (e.g., Brånemark implant) and the implant body is 3.75 mm, a bone tap should be used in D2 bone. On the other hand, when the final osteotomy is 3.2 mm and a similar 3.75-mm implant body with apical flutes for bone debris (e.g., Zimmer) is used, the implant may be inserted without a pretapping process. A Steri-Oss implant may have a thread depth of 0.2 mm, compared with a Brånemark of 0.6 mm. A final osteotomy preparation of 3.2 mm for Steri-Oss, with a small thread depth (compared with a 3.0-mm preparation and 0.6-mm thread depth) means the Steri-Oss does not require a bone tap before the implant insertion. The square thread shape and the thread depth of BioHorizons dental implants combine to indicate a bone tap procedure before implant insertion in D2 bone. Before inserting the implants, the osteotomy is suctioned to remove bone debris, blood, and irrigation to minimize hydrostatic pressure during implant insertion.

The implant may be threaded into position with a low-speed (less than 30 rpm), high-torque (75 N-cm) hand piece, rather than using a hand ratchet. The hand piece allows a more precise implant rotation, and a constant pressure ensures the implant will progress into the site without risk of stripping the bone within the threads (Figure 29-26). During this process, the irrigation may be stopped so the patient does not attempt to close the mouth and swallow, which may contaminate the implant and cause it to be pushed off the axis of the implant osteotomy.

A threaded implant placed in the anterior mandible engages the cortical bone at the edentulous crest and often the lingual lateral side. In division C–h bone, the implant may also engage the apical cortical region. This provides immediate stability and proven long-term survival. The D2 density of bone in the posterior mandible does not provide apical cortical bone, as the inferior alveolar nerve limits implant height. The angulation or cortical bone above the submandibular fossa of the posterior mandible often permits the use of the mandibular lingual plates to this effect. Several implant designs have proven effective at 95% for as long as 10 years when placed into coarse trabecular alveolar bone with a cortical crest in the mandible. In posterior areas, larger-diameter threaded implant designs are suggested to increase the surface area and decrease stress, especially in the crestal regions.

When the anterior maxilla presents this bone density, it is treated similarly to the D2 mandible. A threaded implant should engage the palatal cortical plate rather than the labial cortical bone, which is thinner and porous. When this occurs, a bone tap is usually required to form the implant threads in cortical bone. Otherwise, the implant will be pushed more labial, even stripping the facial plate. The anterior maxilla usually has less available bone height than the anterior mandible. As a result, the apex of the implant may engage the thin cortical plate of the floor of the nose when a solid, traditional screw-type system is used. This is not indicated

WWW.HIGHDENT.IR

Figure 29-27 The bone-implant contact is approximately 70% in D2 bone after initial healing and is excellent for load-bearing capability.

if a hollow, cylinder implant is selected, as perforation of the bone and periosteum may compromise the ability to form bone in the internal basket. Hollow, basket-type implants are not recommended in areas where soft tissue may invade the basket.[67] As the greatest stresses after healing are primarily transmitted around the crest, the primary advantage of the apical end of the implant engaging cortical bone is initial stability during healing.

Healing

The excellent blood supply and rigid initial fixation of D2 bone permits adequate bone healing within 4 months. The lamellar bone–implant interface is more than 60% established at the 4-month healing interval. BIC is approximately 70% at this point in time, especially when cortical bone engages the lateral and lingual portions of the implant (Figure 29-27). Abutment placement and prosthodontic therapy may then commence. It should be noted that the time frame for initial bone healing is based on the density of the bone, not on the location in the jaws. Therefore a 4-month rigid healing phase is adequate for porous cortical and coarse trabecular (D2) bone, even when found in the maxilla. Progressive bone loading is usually not required for D2 bone, although an increase in BIC takes place during the initial loading period.

Thin Porous Cortical and Fine Trabecular Bone (D3)

The third density of bone (D3) is composed of thinner porous cortical bone on the crest and fine trabecular

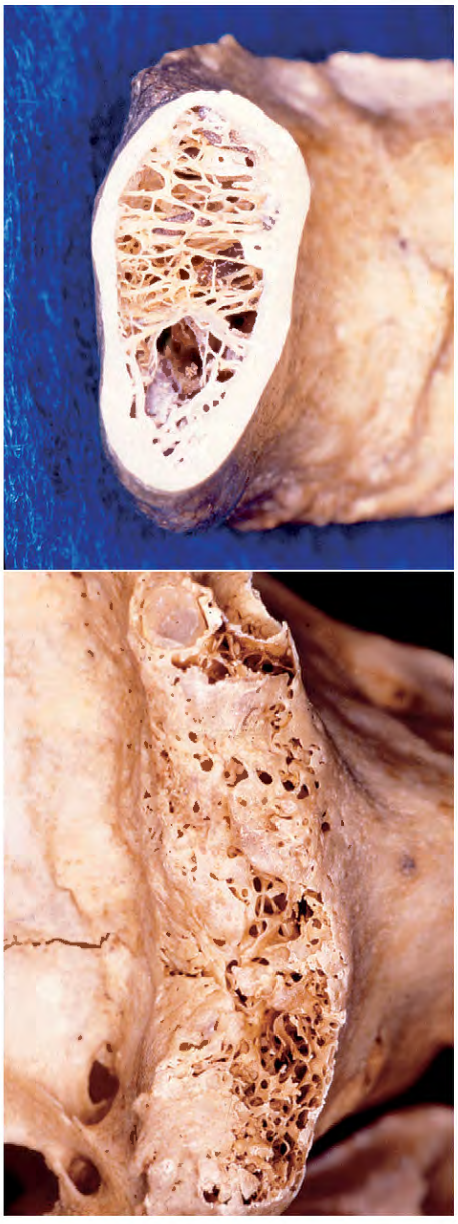


Figure 29-28 D3 bone has a thin, porous cortical crest and fine trabecular bone within the alveolus. It is frequently found in a posterior mandible.

bone within the ridge (Figure 29-28). The mandibular arch usually has more crestal cortical bone than the maxilla (Figure 29-29). The CT-reformatted images may have a range of 375 to 750 Hounsfield units. This bone quality provides the surgeon with a tactile sense similar to drilling in compressed balsa wood. The trabeculae are approximately 50% weaker than those in D2 bone. D3 bone is found most often in the anterior maxilla and posterior regions of the mouth in either arch. It may also be found in the Division B edentulous ridge modified by osteoplasty to provide adequate width for a root form implant placement. Sinus grafts are often D3 bone in the posterior maxilla after a healing period of 6 months or more.[68,69] D3 bone is least prevalent in Division C–h or D anterior mandibles. The ideal minimum

WWW.HIGHDENT.IR

Figure 29-29 Bone-implant contact is in the 50% range for D3 bone.

implant height in D3 bone for a V-shaped or square thread implant design is at least 12 mm when 4 mm in diameter. Larger-diameter implants (5 mm or 6 mm) are more essential in D3 bone in the molar regions than in the previous categories. A roughened implant body (such as acid etched or resorbable blast media) presents advantages in this bone density, regardless of design, to compensate for the limited initial bone contact and decreased bone strength inherent in the trabecular architecture.

The porous cortical layer is thinner on the crest and labial aspect of the maxilla, and the fine trabecular pattern is more discrete in wide edentulous sites. The D3 anterior maxilla is usually of less width than its mandibular D3 counterpart. The D3 bone is not only 50% weaker than D2 bone, the BIC is also less favorable in D3 bone. These additive factors can increase the risk of implant failure. Therefore, small-diameter implants are not suggested in most situations. Instead, bone spreading in this bone density is mechanically easier to perform and allows the placement of greater-diameter implants. The increased-diameter implants lead to improved prognosis, especially when lateral forces or greater force magnitudes are expected. In addition, bone spreading compacts the trabecular bone and increases its density after initial healing.

Advantages of D3 Bone

The main advantage of D3 porous compact and fine trabecular bone is that the implant osteotomy preparation time and difficulty is minimal for each drill size and is usually less than 10 seconds. The crest module drill and bone tap may be eliminated in the surgical protocol. Blood supply is excellent for initial healing, and intraosseous bleeding helps cool the osteotomy during preparation. As a result, this bone density may give the highest surgical survival (Table 29-1).

Table 29-1 Osteotomy Preparation

	OVER-HEATING	TIME	FINAL SIZE DRILLS, NUMBER, SPEED	CREST MODULE DRILL	TAP	INSERTION	HEALING	STAGE II UNCOVERY
D1	++++ Highest risk	1 sec in, 2 sec out "bone dance"	Increase number in sequence. Increase speed 2500 rpm	Yes	Yes (hand ratchet)	Above crest; hand piece unthread $^1/_2$ turn	Lamellar bone; decreased blood supply	3 to 4 months (immediate load)
D2	++ High risk	5 sec out of 10 sec in bone	Manufacturer's protocol; 1500-2500 rpm	Yes	Optional hand piece	At or above crest; hand piece	Ideal	4 months
D3	+		Decrease final size; decrease number; 2500 rpm	No	Optional* hand piece	At or below crest; handpiece	Excellent blood supply; risk of early movement (+)	5 months (rough surface implants)
D4			Minimum number; decrease final size; osteotome	No	No	Below crest; hand piece	Risk of micromove-ment (+++)	6 months (rough surface implants)

+, Degree of risk.
*Palatal plate in maxilla.

WWW.HIGHDENT.IR

Box 29-2 Disadvantages of Porous, Compact Fine Trabecular (D3) Bone

Bone Anatomy
Anterior maxilla often is narrow

Osteotomy
Lateral perforation
Oversize by mistake
Apical perforation

Bone-Implant Contact
Approximately 50%
Additional implant

Implant Placement
One time
Level with thin crestal cortical bone
Greater risk of load during healing
Use high-torque handpiece to insert self-tapping threaded implant

Implant Design
Titanium plasma spray (TPS) or hydroxyapatite (HA) coated
Increased cost
Threaded implant
Greater surface area, but more difficult to place
Press-fit
Easier to insert
May expand bone
If bullet-shaped, has less surface area

Healing Period
Six months to increase lamellar bone and mineralization
Progressive loading more important than for D1 or D2

Disadvantages of D3 Bone

D3 bone also presents several disadvantages (Box 29-2). It is more delicate to manage than the previous two bone density types because its preparation is so easy.

Bone preparation in D3 bone should be 2500 rpm and must be made with constant care of direction to avoid enlargement or elliptical preparation of the site. In the Sharawy et al. study, 2500 rpm prepared the bone at a lower temperature than slower rpm (1225 and 1667 rpm)[39] (Figures 29-30 and 29-31). The preparation time for D3 bone was less than 8 seconds for an 8-mm osteotomy depth, regardless of the rpm. However, the temperature remained elevated for 30 to 50 seconds (Figure 29-32).

A common mistake that causes an elliptical site to form is the use of a finger rest during the osteotomy. Because the drill is often longer than 20 mm, a finger rest results in an arched pathway of the drill into the bone. In dense bone, the side of the drill encroaches

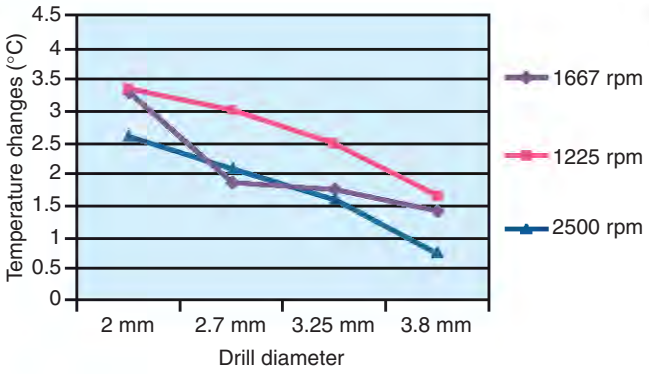


Figure 29-30 The internally irrigated drill of Steri-Oss prepared an 8-mm-deep osteotomy in D3 bone at a lower temperature when the rpm was 2500, compared with 1225 or 1667 rpm.

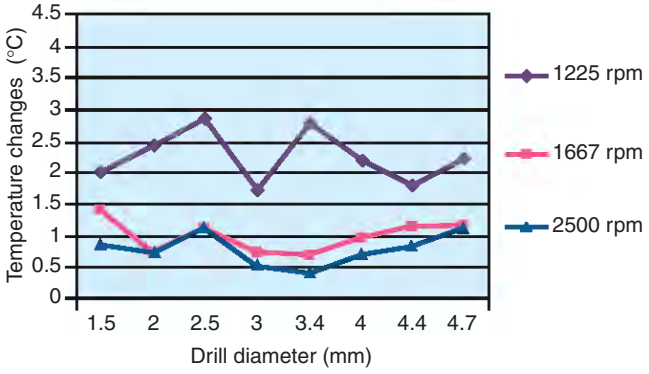


Figure 29-31 The externally irrigated drill of BioHorizons prepared the bone at a lower temperature than the internally irrigated drills; it was lowest at the 2500 rpm speed and highest at 1225 rpm.

upon the dense cortical crest, which opposes the movement and stops the rotation before the crestal osteotomy is enlarged. In D3 bone the arc pathway is not stopped and the osteotomy at the level of the crestal bone is of greater diameter than the drill. If the implant design does not increase at the crestal region, the surgical defect created around the top of the implant may heal with fibrous tissue rather than bone and cause an initial bony pocket.

To improve rigid fixation of traditional root form designs during healing, the opposing thin cortical bone of the nasal or antral floor is often engaged in the maxilla or the apicolingual plate in the mandible, when immediate loading is considered. If the original implant height determined before surgery does not engage the opposing cortical bone, the osteotomy is increased in depth until it is engaged and even perforated. Slightly longer implants are placed in this approach to further increase surface area of support. However, it should be remembered that this technique improves stability during healing but does not decrease the crestal loads to bone after healing. Instead, implant crest module design and the crestal one third of the implant body design are necessary to decrease stress when the implant prosthesis is loaded.

WWW.HIGHDENT.IR

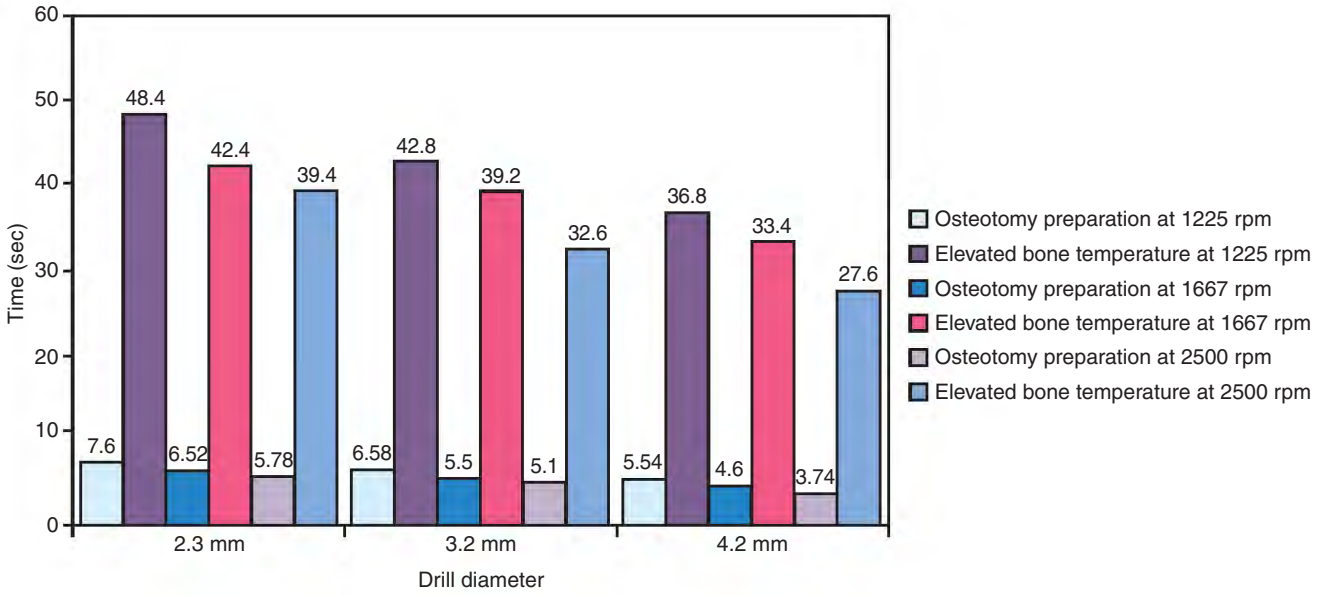


Figure 29-32 The duration during which the temperature was elevated was related to the rpm, with slower rpm being less efficient and associated with a longer elevated temperature.

Figure 29-33 The drill sequence for D3 bone is shortened when compared with standard D2 osteotomy preparation.

The surgeon must be careful to avoid undesired lateral perforations of the cortical bone during osteotomy procedures, especially on the thin, labial porous cortical plate of the maxilla. A common mistake is the stripping of the thin facial plates during the osteotomy. The initial and intermediate drills proceed through the fine trabecular bone without incident (Figure 29-33). However, the lingual aspect of the end-cutting drill hits the thick palatal cortical bone within the osteotomy, which resists preparation, and pushes the drill facially, which may strip the facial plate. A very firm hand, which prevents lateral displacement of the drill and hand piece during the implant osteotomy and does not permit the drill to move facially, is mandatory to prevent this unwanted complication.

A crestal bone drill should not be used in D3 bone. The thin, porous cortical bone on the crest provides improved initial stability of the implant when it is compressed against the crest module of the implant. Unlike D1 and D2 bone, the final drill diameter (3.0 to 3.4 mm for a standard-diameter implant) is of benefit for the 4.1-mm to 4.2-mm crest module dimension to compress the weaker bone. The compressed soft bone not only provides greater stability, it heals with a higher BIC, which is a benefit during the initial bone-loading process.

A bone tap is usually not indicated in D3 bone when Division A bone volume is present. D3 fine trabeculae are 50% weaker than D2 trabeculae, and when the implant is threaded into position, it compresses the bone. This provides improved initial stability and increases the BIC during initial healing. Bone compaction is a benefit when the bone density is poor.

Because crest module drills and a bone tap are usually not used in D3 bone, the number of steps and time of preparation are reduced. The final-size drill should not be passed more than once in the osteotomy to avoid oversizing the preparation. When the residual crest of bone is less than 6 mm wide, a round bone spreader may be tapped into the implant site after the initial 2-mm twist drill to obtain the final osteotomy dimension.[68] The less dense D3 bone easily expands and often permits larger-diameter implants to be inserted.

On the other hand, a complication often occurs when inserting the threaded implant into the prepared bone site of the anterior maxilla. The threaded implant does not completely thread into the more dense palatal plate of bone in this region, and the implant is pushed

WWW.HIGHDENT.IR

facially, often stripping the facial bone as the implant is threaded into position. Although a bone tap in Division A bone volume is usually not necessary in D3 bone, the practitioner may have to both prepare the palatal aspect of the osteotomy separately with a side-cutting drill (e.g., Lindemann drill) to widen this final dimension more palatal, and often should tap the osteotomy in the anterior maxilla with a bone tap to ensure a smooth, atraumatic insertion of the threaded implant in the proper position (Figure 29-34).

A hand ratchet should not be used to tap D3 bone. When a hand ratchet is used, the bone tap is pulled in the direction of the rotating arm of the ratchet. This causes the tap to distort and widen the top of the osteotomy and impair proper bone contact with the crest module of the implant. In addition, the hand ratchet and tap technique do not counter the effect of the more dense bone on the palatal aspect, which forces the tap more facial toward the softer bone. This causes the implant to be positioned more facial than originally prepared and may even strip the thin cortical plate on the facial aspect of the osteotomy.

In abundant bone volume, the implant may self-tap the soft, thin, trabecular bone to enhance initial stability. An implant with a wider crest module can compress the crestal bone when inserted without using a countersink drill. The implant should not be removed and reinserted, as initial rigid fixation may be compromised. If the only cortical bone is on the crest of the ridge, as in a posterior mandible, the implants are not countersunk below the crest in this density of bone. The thin, porous cortical plate provides greater initial stability than the fine trabecular bone underneath. This is especially important in the posterior mandible of a clenching parafunctional patient, as bone torsion occurs during heavy biting pressures.

For a threaded implant, a low-speed (30 rpm), high-torque hand piece should be used rather than a hand wrench for self-tapping implant insertion. This decreases the risk of oversizing the osteotomy with an elliptical implant insertion, which usually results from hand wrench placement in softer bone. A firm hand during hand piece insertion also can prevent this implant from being pushed facially, away from the thicker lingual cortical plate. Tightening a threaded implant to increase fixation once completely inserted is not recommended, because stripping of the threads and decreased fixation may occur.

A roughened surface condition or coating on a threaded implant body is advantageous in this soft bone condition, to enhance initial stability and the amount of initial trabecular bone at the bone-implant interface. The amount of bone initially at the bone-implant interface is reduced in comparison with bone types D1 and D2. If the lingual and apical cortical bone are not engaged at the time of implant placement, less than 50% of the implant surface may actually contact bone.

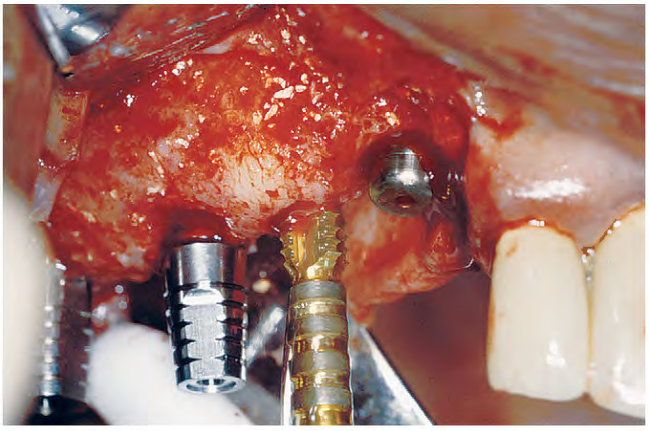


Figure 29-34 A bone tap may be indicated in specific D3 bone conditions, such as the anterior maxilla with dense palatal cortical bone. Otherwise, the threads of the implant do not engage the palatal plate and the implant is pushed into a more facial position.

An additional implant may be used to improve load distribution and prosthodontic support during the early loading period.

Healing

The time frame for atraumatic healing is usually 5 or more months. The actual implant interface develops more rapidly than D2 bone; however, the extended time permits the regional acceleratory phenomenon (RAP) from implant surgery to stimulate the formation of more trabecular bone patterns. In addition, the more advanced bone mineralization within the extra months also increases its strength before loading. An extended gradual loading period is also recommended to further improve this bone density during the initial bone loading.

Fine Trabecular Bone (D4)

Fine trabecular (D4) bone has very little density and little or no cortical crestal bone. It is the opposite spectrum of dense cortical (D1) bone. The most common locations for this type of bone are the posterior molar region of a maxilla in the long-term edentulous patient, or in an augmented ridge in height and width with particulate bone or substitutes, or in a sinus graft. It is rarely observed in the mandible but on occasion does exist. These edentulous ridges are often very wide but have reduced vertical height. This bone type is also present after osteoplasty in wide D3 ridges because the crestal cortical bone is removed during this procedure.

The tactile sense during osteotomy preparation of this bone is similar to stiff, dense Styrofoam or soft balsa wood. The bone trabeculae may be up to 10 times weaker than the cortical bone of D1. The BIC after initial loading is often less than 25% (Figure 29-35). A CT scan with reformatted images of D4 bone has a Hounsfield number of less than 375 units.

WWW.HIGHDENT.IR

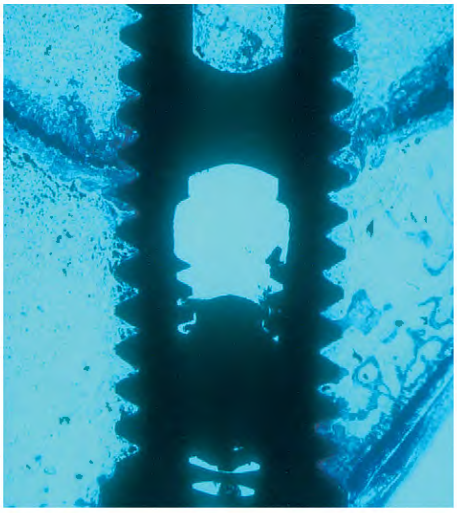


Figure 29-35 The posterior maxillary region may be D4 bone, with BIC after initial loading no greater than 25%.

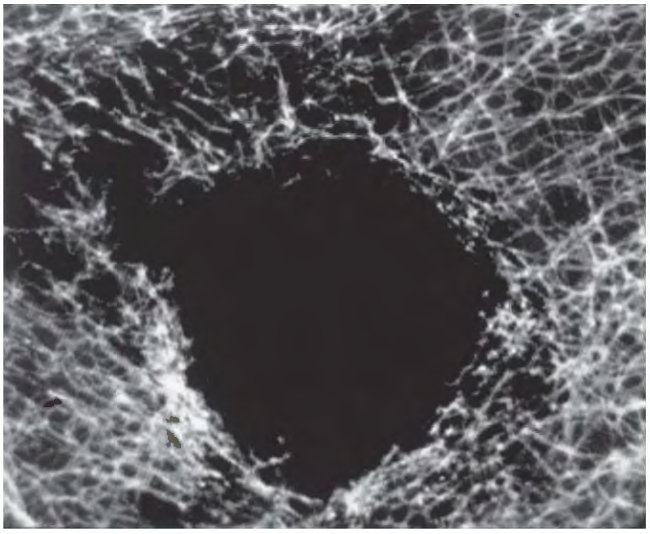


Figure 29-36 A conventional drilling procedure uses an extraction technique that removes bone from the site.

Box 29-3 Disadvantages of Fine Trabecular (D4) Bone

Bone Anatomy

Location
No cortical crest
Decreased height
Sinus graft

Osteotomy

Decreased final width and length
Perforation

Bone-Implant Contact

Approximately 25%
Additional implant indicated

Implant Placement

One time
Countersunk

Implant Design

Needs coating

The ideal implant height in D4 bone for a V-shaped thread implant design approaches 15 mm. Power thread implant designs may use implants as short as 12 mm. The requirement for minimum height of available bone is greater in D4 bone than in other types. Sinus grafts are often indicated to ensure adequate height for ideal implant surface area of support. In addition, sinus grafts permit the implant to engage the original sinus floor composed of a thin porous cortical bone, which helps further stabilize the implant.

Division B implants are not suggested in this bone type. Bone spreading is easiest in this bone density, and larger-diameter implants are suggested whenever possible. A roughened surface or hydroxyapatite (HA) coating is almost mandatory to improve the amount of bone-implant contact in this bone quality after initial healing.

Disadvantages of D4 Bone

Fine trabecular bone presents the most arduous endeavor to obtain rigid fixation. Bone trabeculae are sparse and, as a result, initial fixation of any implant design presents a surgical challenge (Box 29-3). The implant surgeon should not prepare D4 bone with rotating drills, which use an extraction technique to remove bone preparation of the osteotomy (Figure 29-36). The initial drill to determine site depth and angulation is the only one that should be used in this bone type, after which osteotomes may be used with a surgical mallet or hand piece to compress the bone site, rather than remove bone, as the osteotomy increases in size (Figures 29-37 to 29-39). The compaction technique of the site is prepared with great care. The bone site is easily distorted, resulting in reduced initial stability of the implant. The final osteotomy diameter is similar to the D3 preparation. The residual ridge is easily expanded in this bone type. The osteotomy may both compress the bone trabeculae and expand the osteotomy site.

The implant should self-tap the bone or shape the implant receptor site while being seated with a slow-speed, high-torque hand piece (Figure 29-40). A hand wrench is contraindicated. The pressure on the implant during insertion corresponds to the speed of rotation, and the implant proceeds to self-tap the soft bone. It is difficult to thread an implant in soft bone in difficult

WWW.HIGHDENT.IR

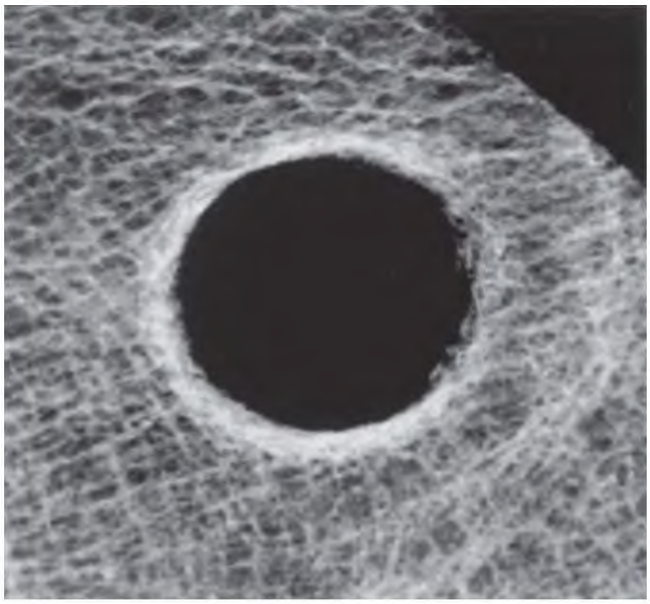


Figure 29-37 A bone compaction technique to prepare the implant site, compress the bone, and make it more dense.

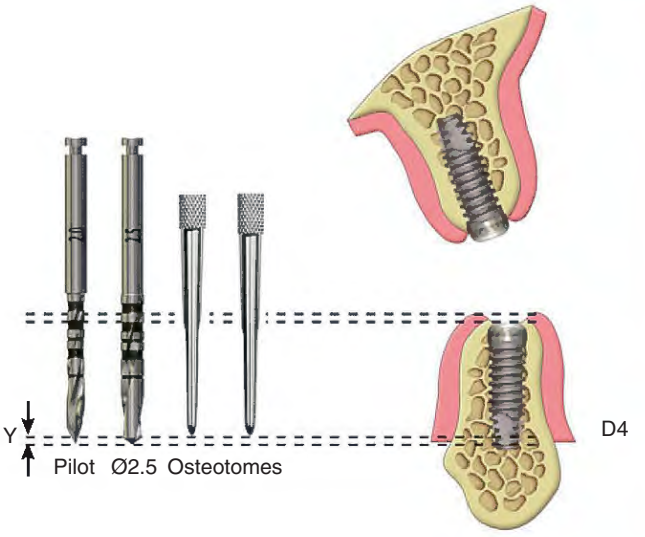


Figure 29-38 Osteotomes of increasing size may be used in D4 bone to compress the residual bone, making it more dense to provide enhanced initial support the implant.

access regions. If there is any cortical bone in the opposing landmark, it is engaged to enhance stability and simultaneously ensure the maximum length of implant. An implant with a greater crestal diameter presents the added benefit to further compress the crestal bone for stability.

Once inserted, the implant should not be removed and reinserted; instead, one-time placement is mandatory. The implant is countersunk in this bone if any risk of loading is expected during healing (e.g., under a soft tissue–borne denture). Countersinking the implant

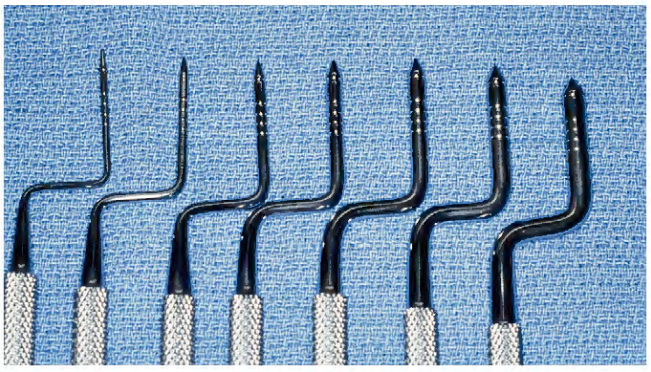


Figure 29-39 Bone compaction instruments are used after the initial pilot drill to prepare the osteotomy.

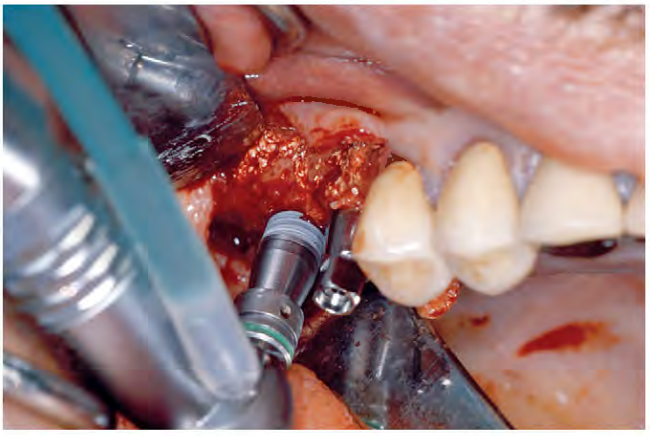


Figure 29-40 A slow-speed, high-torque hand piece is used to thread the implant into position for D4 bone. No crestal bone drill or tapping procedure is indicated in this soft bone. This ensures the implant osteotomy is not widened by the hand movement and ratchet.

below the crest reduces the risk of micromovement during healing in this very soft bone (Figure 29-41). No countersink drill is used before countersinking.

Additional implants are placed to improve implant-bone loading distribution and prosthodontic rehabilitation, especially during the first critical year of function. For fixed restorations, one implant is used for each buccal tooth root replaced, and no cantilever on the prosthesis is used with this bone density. An additional implant may be placed at the time of surgery in the second molar region to further improve support during the first critical year of loading. The implant of choice in the wide posterior maxilla with D4 bone is a greater diameter, roughened surface or HA-coated threaded implant. When properly inserted, the HA-threaded implant can be more stable and provides greater surface area. The larger-diameter implant offers greater surface area for support, further compresses the fine trabecular bone for greater initial rigidity, has a greater chance to engage the lateral regions of cortical bone for support, and improves stress transfer during loading.

WWW.HIGHDENT.IR

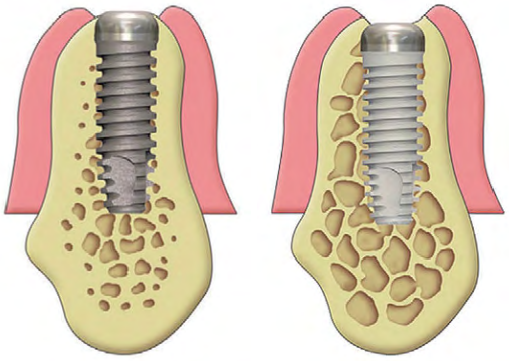


Figure 29-41 In D4 bone, the implant is often countersunk below the crest of the ridge, wherever a soft tissue-borne restoration is worn during the initial healing phase. In D2 bone, the implant is usually placed at the crest of the bone.

Healing

The healing and progressive bone loading sequence for D4 bone require more time than the other three types of bone. Time is needed to allow bone to remodel at the surface and to intensify its trabecular pattern. The additional time also allows a more advanced bone mineralization and increased strength. Six or more months of undisturbed healing is suggested. The compression technique for surgery, the extended healing time, and progressive bone loading protocol allow the remodeling of this bone into a more organized and load-bearing quality similar to D3 bone before the final prosthetic loading of the implants.

SUMMARY

Bone remodels in relation to the forces exerted upon it. Depending on the location of the edentulous ridge and the amount of time the area has been edentulous, the density of bone is variable. Clinically, the surgeon can correlate the hardness of the trabecular bone and the presence of a cortical plate with four different densities of bone. The typical locations of these different densities, the alteration in surgical technique with each type, and the advantages and disadvantages of each have been related to each density classification. The dense cortical bone of D1 is the strongest bone, approximately 10 times greater than D4 bone, and is the most difficult to prepare. The thick, porous cortical and coarse trabecular D2 bone is twice as strong as D3 bone and is ideal for implant support. The thin, porous cortical and fine trabecular D3 bone is similar to preparations in compressed balsa wood. The fine trabecular bone of D4 is similar to osteotomies in dense Styrofoam. The initial drills may be used to distinguish among the four bone density types.

Type D1 bone heals with a lamellar bone interface and has the greatest percentage of bone at the implant body contact regions. Type D2 bone heals with woven and lamellar bone, is adequately mineralized at 4 months, and often has approximately 70% bone in initial contact after healing with the implant body. An ideal length, V-shaped, threaded-screw implant should have 12-mm minimum height. Type D3 bone has about 50% bone at the initial implant interface after healing and benefits from a roughened surface on the screw-shaped implant body to increase initial fixation and bone contact. A minimum of 12-mm implant height is typically required for initial fixation and loading. An additional 1 month (total of 5 months) is used for initial bone healing, compared with D2 bone, to permit a greater percentage of bone trabeculae to mineralize and form around the implant. D4 bone density has the least amount of trabeculae at implant placement. The minimum implant height of a V-shaped thread design is approximately 15 mm, and HA coatings or a roughened surface should be used on the implant body. Compressive thread designs may gain stability and optimum stress distribution with implants 12 mm long and should be rough or HA coated. Additional time for bone healing and incremental bone loading will improve the density and result in implant survival similar to that of other bone densities.

References

1. Misch CE: Bone character: second vital implant criterion, *Dent Today* 7:39-40, 1988.
2. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing, and progressive bone loading, *Int J Oral Implantol* 6:23-31, 1990.
3. Brånemark PI, Albrektsson T: Microcirculation and healing of artificial implants in bone. In *Proceedings of the 2nd World Congress for Microcirculation* 2:59-60, 1979.
4. Adell R, Lekholm U, Rockler B: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 10:387-416, 1981.
5. Brunski JB, Moccia AF Jr, Pollack SR et al: The influence of functional use of endosseous implants on the tissue-implant interface: histological aspects, *J Dent Res* 58:1953, 1979.
6. Qu Z: *Mechanical properties of trabecular bone in human mandible* [doctoral thesis], Birmingham, Ala, 1994, University of Alabama at Birmingham.
7. Misch CE, Qu Z, Bidez MW, Mechanical properties of trabecular bone in the human mandible: implications for dental implant treatment planning and surgical placement, *J Oral Maxillofac Surg* 57:700-706, 1999.
8. Lekholm U, Zarb GA: Patient selection and preparation. In Brånemark P-I, Zarb GA, Albrektsson T, editors: *Tissue-integrated prostheses,* Chicago, 1985, Quintessence.
9. Engquist B, Bergendal T, Kallus T et al: A retrospective multicenter evaluation of osseointegrated implants supporting overdentures, *Int J Oral Maxillofac Implants* 3:129-134, 1988.
10. Jaffin RA, Berman CL: The excessive loss of Brånemark fixtures in type IV bone: a 5-year analysis, *J Periodontol* 62:2-4, 1991.
11. Friberg B, Jemt T, Lekholm U: Early failures in 4,641 consecutively placed Brånemark dental implants: a study from stage I surgery to the connection of completed prostheses, *Int J Oral Maxillofac Implants* 6:142-146, 1991.

WWW.HIGHDENT.IR

12. Quirynen M, Naert I, van Steenberghe D et al: A study of 589 consecutive implants supporting complete fixed prostheses: dental and periodontal aspects, *J Prosthet Dent* 68:655-663, 1992.
13. Fugazzotto PA, Wheeler SL, Lindsay JA: Success and failure rates of cylinder implants in type IV bone, *J Periodontol* 64:1085-1087, 1993.
14. Hutton JE, Heath MR, Chai JY et al: Factors related to success and failure rates at 3 year follow up in a multicenter study of overdentures supported by Branemark implants, *Int J Oral Maxillofac Implants* 10:33-42, 1995.
15. Sullivan DY, Sherwood RL, Collins TA et al: The reverse-torque test: a clinical report, *Int J Oral Maxillofac Implants* 11:179-185, 1996.
16. Snauwaert K, Duyck D, van Steenberghe D et al: Time dependent failure rate and marginal bone loss of implant supported prostheses: a 15-year follow-up. *Study Clin Oral Invest* 4:13-20, 2000.
17. Herrmann I, Lekholm U, Holm S et al: Evaluation of patient and implant characteristics as potential prognostic factors for oral implant failures, *Int J Oral Maxillofac Implants* 20:220-230, 2005.
18. Truhlar RS, Morris HF, Ochi S et al: Second stage failures related to bone quality in patients receiving endosseous dental implants: DICRG Interim Report No. 7. Dental Implant Clinical Research Group, *Implant Dent* 3:252-255, 1994.
19. Misch CE, Hoar JB, Beck G et al: A bone quality based implant system: a preliminary report of stage I and stage II, *Implant Dent* 7:35-42, 1998.
20. Misch CE, Poitras Y, Dietsh-Misch F: Endosteal implants in the edentulous posterior maxilla—rationale and clinical results, *Oral Health* 7-16, August 2000.
21. Kline R, Hoar JE, Beck GH et al: A prospective multicenter clinical investigation of a bone quality based dental implant system. *Implant Dent* 11:224-234, 2002.
22. Weng D, Tarnow D, Hurzeler M et al: A prospective multicenter clinical trial of 3i machined surface implants: results after 6 year follow up, *Int J Oral Maxillofac Implants* 18:417-423, 2003.
23. Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study. *J Periodontol* 77: 1340-1347, 2006.
24. Misch CE, Misch-Dietsh F, Steigenga et al: Posterior implant single tooth replacement and status of adjacent teeth: a 10-year multicenter retrospective study (submitted for publication).
25. Misch CE: *Bone density and root form implants,* Dearborn, Mich, 1984, Quest Implants.
26. Roberts EW, Turley PK, Brezniak N et al: Bone physiology and metabolism, *J Calif Dent Assoc* 15:54-61, 1987.
27. Roberts WE: Fundamental principles of bone physiology, metabolism and loading. In Naert I, van Steenberghe D, Worthington P, editors: *Osseointegration in oral rehabilitation*, Carol Stream, Ill, 1993, Quintessence.
28. Babbush C: Titanium plasma spray screw implant system for reconstruction of the edentulous mandible, *Dent Clin North Am* 30:117-131, 1986.
29. Chiapasco M, Gatti C: Implant-retained mandibular overdentures with immediate loadings: a 3-to 8-year prospective study on 328 implants, *Clin Implant Dent Relat Res* 5:29-38, 2003.
30. Haider R, Watzek G, Plenk H Jr: Influences of drill cooling and bone structure on primary implant fixation, *Int J Oral Maxillofac Implants* 8:83-91, 1993.
31. Vaughan JM: *The physiology of bone,* Oxford, 1970, Clarendon Press.
32. Chanavaz M: Anatomy and histophysiology of the periosteum: classification of the periosteal blood supply to the adjacent bone with 855r and gamma spectrometry, *J Oral Implantol* 21:214-219, 1995.
33. Rhinelander FW: The normal circulation of bone and its response to surgical intervention, *J Biomed Mater Res* 8:87-90, 1974.
34. Crock JG, Morrisson WA: A vascularized periosteal flap: anatomical study, *Br J Plast Surg* 45:474-478, 1992.
35. Eriksson AR, Albrektsson T, Albrektsson B: Heat caused by drilling in cortical bone. Temperature measured in vivo in patients and animals, *Acta Orthop Scand* 55:629-631, 1984.
36. Schroeder A: Preparation of the implant bed. In Schroeder A, Sutter F, editors: *Oral Implantology,* New York, 1996, Thieme.
37. Ercoli C, Funkenbusch PD, Lee HJ et al: The influence of drill wear on cutting efficiency and heat production during osteotomy preparation for dental implants: a study of drill durability, *Int J Oral Maxillofac Implants* 19:335-349, 2004.
38. Eriksson R, Adell R: Temperatures during drilling for the placement of implants using the osseointegration technique, *J Oral Maxillofac Surg* 44:4-7, 1986.
39. Sharawy M, Misch CE, Weller N et al: Heat generation during implant drilling: the significance of motor speed, *Oral Maxillofac Surg* 60:1160-1169, 2002.
40. Matthews J, Hirsch C: Temperatures measured in human cortical bone when drilling, *J Bone Joint Surg* 45A: 297-308, 1972.
41. Wiggins KL, Malkin S: Drilling of bone, *J Biomech* 9:553-559, 1976.
42. Chacon GE, Bower DL, Larsen PE et al: Heat production by 3 implant drill systems after repeated drilling and sterilization, *J Oral Maxillofac Surg* 64:265-269, 2006.
43. Benington IC, Biagioni PA, Briggs J et al: Thermal changes observed at implant sites during internal and external irrigation, *Clin Oral Implants Res* 13:293-297, 2002.
44. Adell R, Lekholm U, Brånemark PI: Surgical procedures. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue-integrated prostheses osseointegration in clinical dentistry,* Chicago, 1985, Quintessence.
45. Watzek G, Haider R, Gitsch M et al: *Influence of drill cooling and bone structure on primary implant fixation* [abstract], Boston, 1991, American Academy of Osseo-integration.
46. Brisman DL: The effect of speed, pressure, and time on bone temperature during the drilling of implant sites, *Int J Oral Maxillofac Implants* 11:35-37, 1996.
47. Rafel SS: Temperature changes during high-speed drilling on bone, *J Oral Surg Anesth Hosp Dent Serv* 20:475-477, 1962.
48. Hobkirk J, Rusiniak K: Investigation of variable factors in drilling bone, *J Oral Surg* 35:968-973, 1977.
49. Eriksson RA, Albrektsson T: Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit, *J Prosthet Dent* 50:101-107, 1983.
50. Hiener P: *Bohr maschine fur die implantologie. Eine experimentelle untersuchung,* [dissertation], Aachen, Germany, 1992, Aachen University.
51. Marx RE: *Principles of hard and soft tissue reconstruction of the jaws* [abstract ML 315], New Orleans, 1990, American Association of Oral and Maxillofacial Surgeons.

WWW.HIGHDENT.IR

52. Yacker M, Klein M: The effect of irrigation on osteotomy depth and bur diameter, *Int J Oral Maxillofac Implants* 11:635-638, 1996.
53. Davidson SR, James DF: Drilling in bone: modeling heat generation and temperature distribution, *J Biomech Eng* 125:305-314, 2003.
54. Cordioli G, Majzoub Z: Heat generation during implant site preparation: an in vitro study, *Int J Oral Maxillofac Implants* 12:186-193, 1997.
55. Babbush CA: The endosteal blade vent implant—the histology of animal studies and scanning electron microscope observations. In Babbush CA, editor: *Surgical atlas of dental implant techniques,* Philadelphia, 1980, WB Saunders.
56. Lavelle CH, Wedgewood D: Effect of internal irrigations on frictional heat generated from bone drilling, *J Oral Surg* 38:499, 1980.
57. Misch CE, Sharawy M: Contaminants in the internal chamber of internally cooled bone drills—a pilot study (to be published).
58. Novaes AB Jr, de Oliveira RR, Taba M Jr et al: Crestal bone loss minimized when following the crestal preparation protocol: a histomorphometric study in dogs, *J Oral Implantol* 31:276-282, 2005.
59. Watzek G, Danhel-Mayhauser M, Matejka M et al: *Experimental comparison of Brånemark and TPS dental implants in sheep* [abstract]. Presented at UCLA Symposium: Implants in the partially edentulous patient, Palm Springs, Calif, 1990.
60. Satomi K, Akagawa Y, Nikai H et al: Bone implant interface structures after nontapping and tapping insertion of screw type titanium alloy endosseous implants, *J Prosthet Dent* 59:339-342, 1988.
61. Beer A, Gahleitner A, Holm A et al: Adapted preparation technique for screw-type implants: explorative in vitro pilot study in a porcine bone model, *Clin Oral Implants Res* 18:103-107, 2007.
62. Haraldson T: A photoelastic study of some biomechanical factors affecting the anchorage of osseointegrated implants in the jaw, *Scand J Plast Reconstr Surg* 14:209-214, 1980.
63. Adell R, Lekholm U, Rockler B et al: Marginal tissue reactions at osseointegrated titanium fixtures (1): a 3 year longitudinal prospective study, *Int J Oral Maxillofac Surg* 15:39, 1986.
64. Misch CE, Wang H-L, Misch CM et al: Rationale for the application of immediate load in implant dentistry: part I, *Implant Dent* 13:207-215, 2004.
65. Misch CE, Wang H-L, Misch CM et al: Rationale for the application of immediate load in implant dentistry: part II, *Implant Dent* 13:310-321, 2004.
66. Orenstein IH, Synan WJ, Truhlar RS et al: Bone quality in patients receiving endosseous dental implants: DICRG Interim Report No. 1, *Implant Dent* 3:90-94, 1994.
67. Takeshita F, Suetsugu T, Higuchi Y et al: A histologic study of failed hollow implants, *Int J Oral Maxillofac Implants* 11:245-250, 1996.
68. Tatum OH: Maxillary and sinus implant reconstructions, *Dent Clin North Am* 30:207-229, 1986.
69. Misch CE: Maxillary sinus augmentation for edentulous arches for implant dentistry: organized alternative treatment plans, *Int J Oral Implantol* 4:7-12, 1987.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 30

Posterior Single-Tooth Replacement: Surgical Guidelines

Carl E. Misch

Seventy percent of the dentate U.S. population is missing at least one tooth. Compared with past generations, single-tooth replacement will most likely comprise a larger percentage of prosthetic dentistry in the future. In 1960 the average American older than age 55 had just seven original teeth. Today the average 65 year old has 18 teeth, whereas the baby boomers can expect to have at least 24 teeth left when they reach age 65. Currently the first teeth lost are usually between the ages of 35 and 54 years. Almost 30% of the 50 to 59 year olds examined in a U.S. national survey exhibited either single or multiple posterior edentulous spaces bordered by natural teeth.[1] Treatment to replace single teeth in the posterior regions represents nearly 7% of the annual dental care reimbursement from insurance companies, and total estimated treatment costs for all patients missing a posterior tooth approximate $10 billion U.S. each year.[2,3]

POSTERIOR MISSING TOOTH

The first molars are the first permanent teeth to erupt in the mouth and are often the first to decay. The adult patient typically has had one or more crowns fabricated to restore the integrity of the tooth and replace previous large restorations. Longevity reports of crowns have yielded very disparate results, with the mean life span at failure reported to be 10.3 years. The primary cause of failure of the crown is endodontic therapy, porcelain or tooth fracture, or uncemented restoration, which leads to decay. The posterior tooth is at risk for extraction as a result of these complications.[1-9] Replacement of the posterior tooth is frequently necessary, and at least one of the adjacent natural teeth are virgin or minimally restored more than 80% of the time. Therefore, a traditional three-unit fixed partial denture to replace a single tooth would remove the enamel on a completely healthy tooth and begin a cascade of potential complications, which often leads to loss of the tooth.

SINGLE-TOOTH IMPLANTS

From 1993 to the present, single-tooth implants have become the most predictable method of tooth replacement and therefore the treatment of choice. There are more refereed reports in the literature than for any other tooth replacement method. Becker et al. reported a more than 90% success rate in a study over 4 years of 282 molar implants.[10,11] Simon et al. followed 70 molar implants over a period ranging from 6 months to 10 years with a 97.1% success rate.[12] Levin et al. reported in 2006 on single-molar replacement with implants from 6 to 125 months (>10 years) with a 93.6% success rate.[13]

A 10-year report by Priest indicated the posterior single tooth was more than 97% successful.[3] More important, there were no adjacent teeth lost from endodontic failure or causes, and only one tooth required endodontic therapy after implant insertion. This report clearly identifies the adjacent teeth to be least at risk when the missing tooth is replaced with an implant. In a 2007 study, Misch et al. reported a 99% survival rate (11 initial healing failures and three late failures) for 1377 posterior single-tooth implants for as long as 10 years.[14] A review of the literature by Goodacre et al. from 1981 to 2003 found single-tooth replacement with an implant had the highest implant prosthesis survival rate and averaged 97% survival.[15] The most common complication reported was abutment screw loosening, which did not cause the prosthesis or implant to fail. In a consensus statement, Salinas et al. reported pooled success for single-tooth restorations exceeded the success rate of traditional fixed partial dentures.[16]

IMPLANT BODY SELECTION

The implant body for a posterior single-tooth implant should include specific features to reduce complications. The most common problem associated with a single

WWW.HIGHDENT.IR

tooth is abutment screw loosening.[10,11,15,16] The higher the torque used to tighten the screw, the less the risk of loosening. Crest module and abutment connection designs, which decrease forces to the abutment screw, are also indicated. The implant must have an antirotational feature (e.g., an external or internal hex). The greater its height or depth, the less force transmitted to the abutment screw. However, the diameter of the implant is more significant to reduce the stress. Accuracy of component fit, the abutment screw design, and number of threads on the fixation screw are other critical features.

The implant body should be made of titanium alloy to reduce the risk of long-term fracture, as it is four times more resistant to fracture than grade 1 titanium and twice as strong as grade 3 titanium. Sullivan reported a 14% implant fracture rate for single molars fabricated on standard-diameter Nobel Biocare implants (grade 1 titanium) and concluded that this is not a viable treatment.[17] A threaded implant provides greater functional surface area than a cylinder, and a tapered implant provides less surface area than a parallel-walled implant body. When implant bodies are internal hex designs, the dimension of the implant in the posterior regions should be at least 4 mm or more in diameter to increase the outer body wall thickness and reduce the risk of long-term body fracture.

The ideal diameter of a single-tooth implant is dependent upon the mesiodistal dimension of the missing tooth and the buccolingual dimension of the implant site. When the facial bone thickness is inferior to 1.4 mm, bone loss may result and implant failures may occur with greater frequency.[18] Horizontal bone loss around the implant causes increased probing depths or increased risk of soft tissue shrinkage. These may affect the bacterial flora or cervical esthetics of the soft tissue drape. This may be why gingival recession around wide-diameter implants has been noted to be greater than with a standard diameter.[19,20] The ideal implant size is 1.5 to 2 mm from an adjacent tooth and has at least 0.5 mm of bone on the lateral aspects of the implant body. The ideal implant diameter in the intratooth posterior region should be at least 3 mm less than the mesiodistal dimension of the missing tooth (from cement-enamel junction [CEJ] to CEJ) and 3 mm narrower than the buccolingual dimension of bone. As a general rule, the molar implant should be larger in diameter than a premolar implant.

PREMOLAR IMPLANT REPLACEMENT

The most ideal posterior tooth to replace with an implant is the first premolar, in either arch. The vertical available bone usually is greater than any other posterior tooth positions. In the maxilla, it is almost always anterior or below the maxillary sinus, and the mandibular first premolar is almost always anterior to the mental foramen and mandibular neurovascular complex. The bone trajectory for implant insertion is more favorable in the mandibular first premolar than for any other tooth in the arch.

The maxillary premolars are often in the esthetic zone of patients with a high smile line. The need for bone grafting before maxillary first premolar implant placement is very common, as the thin buccal facial plate is often lost during the extraction process. Implant placement without bone grafting may result in a recessed emergence profile that, in the past, was corrected with a facial ridge lap crown, which does not allow proper hygiene or probing and should be avoided. To ensure proper esthetics and avoid the need for a ridge lap, the implant body often is positioned under the buccal cusp to improve the cervical emergence profile of the maxillary premolar crown.

The natural premolar tooth root is 4.2 mm in diameter on average at a distance of 2 mm below the CEJ. As a consequence, the most common implant diameter is about 4 mm at the crest module. This also provides approximately 1.5 mm of bone on the proximal surfaces adjacent to the natural teeth when the mesiodistal space is 7 mm or greater. However, when the mesiodistal dimension is only 6.5 mm, a 3.5-mm implant is suggested.

The maxillary canine root is often angled 11 degrees distally and presents a distal curve 32% of the time, which may extend over the shorter root of the maxillary first premolar. The mandibular canine also may have a distal inclination. The implant body is often longer than the natural tooth root of a premolar tooth. The surgeon may inadvertently place the implant parallel to the second premolar and, consequently, into the natural canine root. This may not only result in endodontic therapy of the canine, but also may cause root fracture and loss of the tooth (Figure 30-1, *A*). Therefore in the first premolar region, care is taken to evaluate the canine angulation. The first premolar implant may need to be placed parallel to the canine root and may need a shorter-than-ideal implant. A tapered implant body at the apical one third also may be of benefit, to further decrease the risk of inadvertently engaging the canine root (Figure 30-1, *B*).

The second premolar apex may be located over the mandibular neurovascular canal or maxillary sinus. This results in a reduced bone height compared with the anterior region of the jaws and a shorter implant.

MOLAR IMPLANT REPLACEMENT

The molar mesiodistal dimension usually ranges from 8 to 12 mm, depending on the original tooth size and the amount of mesial drift of the second molar before implant placement. When one 4-mm-diameter implant is placed to support a crown with a mesiodistal dimension of 12 mm, this may create a 4- to 5-mm cantilever on the

WWW.HIGHDENT.IR

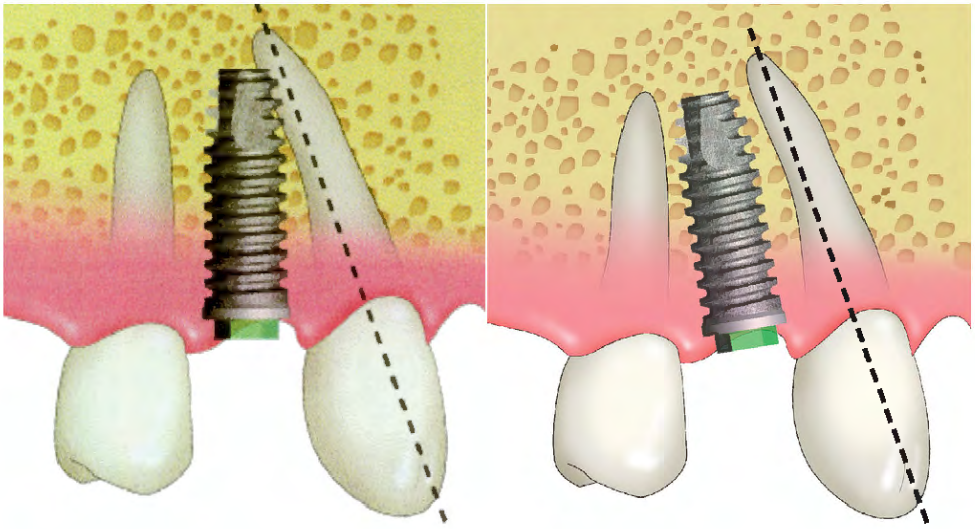


Figure 30-1 **A,** A maxillary canine root is often slanted 11 degrees distally and has a distal curvature 32% of the time. When the implant is placed parallel to the first premolar, it may inadvertently encroach upon the canine root. **B,** A maxillary first premolar implant may need to be parallel to the canine.

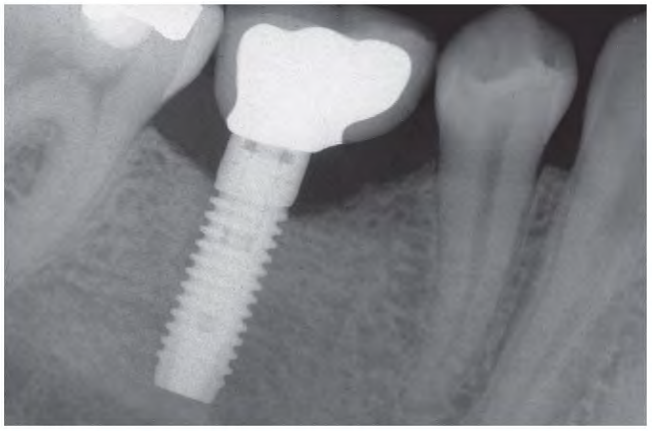


Figure 30-2 When a mesiodistal space is 14 mm, standard 4-mm-diameter implants will have a 5-mm cantilever, which increases the force to the cement, abutment screw, marginal bone, and implant body. Therefore an increased risk for biomechanical complications occurs.

marginal ridges. The magnified occlusal forces (especially important in parafunction) may cause abutment screw loosening, an exaggerated emergence profile on the crown, bone loss, and implant body fracture (Figure 30-2).

The most common complication of one regular 4-mm-diameter implant to replace a molar is abutment screw loosening. When the mesiodistal dimension permits, two 3.5- to 4-mm-diameter implants should be considered to restore the region to improve stress reduction and, in turn, reduce the incidence of abutment screw loosening. In a 3-year report of posterior first-molar replacements, Balshi et al.[21,22] found implant screw loosening was a common complication when one implant replaced the tooth (48%) and was reduced to 8% when two splinted implants replaced the first molar. Other studies found the one-wide diameter implant had greater screw loosening than two splinted implants.[23,24] A 50% decrease in mesiodistal and buccolingual stress was found between a 5-mm and two splinted, standard-diameter implants.[24] Therefore, whenever possible, two splinted implants should be used to replace a larger single-molar space.

As mentioned earlier, Sullivan reported a 14% implant fracture rate for single molars on a standard, 4.1-mm implant.[17] Rangert et al. reported overload-induced bone resorption appeared to precede implant fracture in a significant number of single-molar implants, which were 4 mm in diameter.[25]

When the mesiodistal dimension of the missing tooth is 8 to 12 mm with a buccolingual width greater than 7 mm, a 5- to 6-mm-diameter implant body is suggested to reduce the stress to the implant system. Langer et al.[26] also recommended the use of wide-diameter implants in bone of poor quality or for the immediate replacement of failed implants. The larger-diameter implant does not require as long an implant, which also is a benefit because of the reduced posterior vertical bone height due to anatomical limitations. When possible, a larger diameter implant (or two splinted, traditional-size implants) should be inserted to enhance the mechanical properties of the implant system through increased surface area, stronger resistance to component fracture, increased abutment stability, and enhanced emergence profile for the crown.

When the posterior space is 14 mm or greater, the largest implant diameter for two adjacent implants may be calculated by subtracting 6 mm (1.5 mm from each tooth and 3 mm between the implants) from the intratooth distance and dividing by 2 (Figure 30-3).

$$\frac{16\,\text{mm} - 6\,\text{mm}}{2} = 5\,\text{mm for each implant.}$$

WWW.HIGHDENT.IR

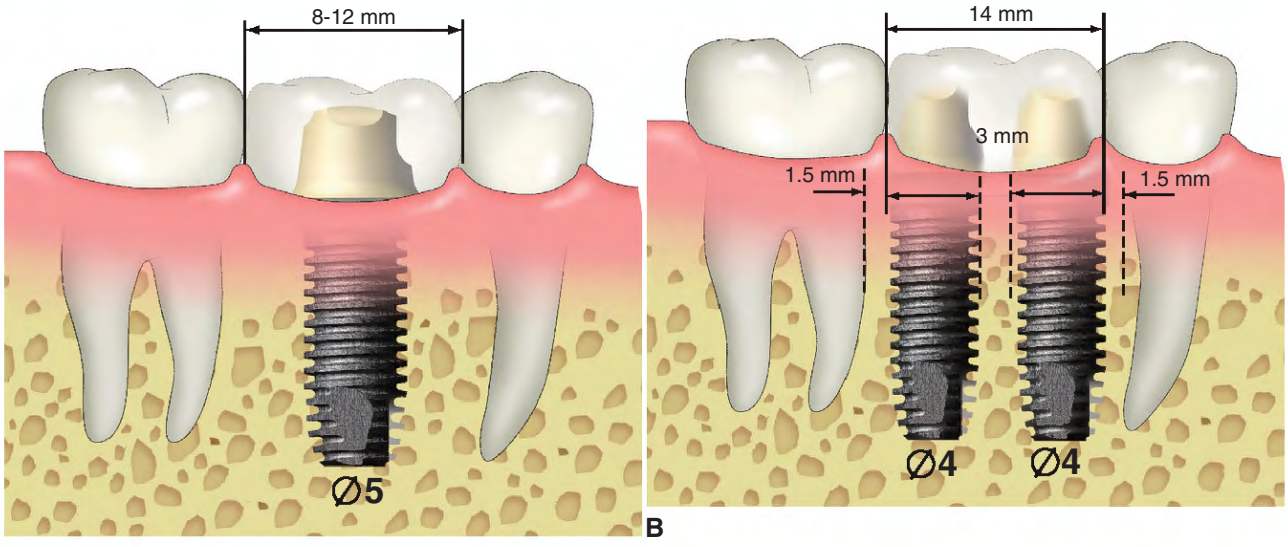


Figure 30-3 When the intratooth space is 14 mm, the ideal implant size is 14 mm – 6 mm (1.3 mm from each tooth and 3 mm between the implants) ÷ 2 = 4 mm for each implant.

It should be noted the diameter used is the crest module dimension, which is often 0.2 to 0.35 mm greater than the implant body dimension (i.e., 4.1/3.75 mm for Nobel Biocare SteriOss, 3-I, LifeCore). The two implants should be 3 mm apart, because the width of the crestal defect around an implant is usually less than 1.5 mm. The two adjacent implants 3 mm or more apart will not convert the angular defect next to each implant into a horizontal defect, which may increase sulcus depths or cause a loss of papilla height.[27] When possible, two regular-size or one regular and one large-diameter implant are suggested when replacing molars. The space is usually restored with two premolar-size crowns, rather than a large molar with a furcation (Figure 30-4).

When the posterior mesiodistal space between teeth is 12 to 14 mm, the treatment plan of choice is less obvious. A 5-mm-diameter implant may result in cantilevers up to 5 mm on each marginal ridge of the crown. However, two implants present a greater surgical, prosthetic, and hygiene risk. The first option is to slightly reduce the standard implant diameter to 3.5 mm, rather than 3.75 or 4.0 mm. The primary space requirement is reduced to 13 mm of space, instead of 14 mm. Additional space also may be gained in several ways:

1. Enamoplasty of the adjacent teeth proximal contours to increase the mesiodistal dimension of the missing tooth. It is not unusual that the distal natural tooth has tipped toward the edentulous space. An enamoplasty may be even more effective in these cases to increase space (Figure 30-5).
2. Orthodontics to upright a tilted second molar or increase the intratooth space. One anterior implant

Figure 30-4 **A,** When the intratooth space is 14 to 20 mm, two implants are used to restore the space. The implants are positioned 1.5 to 2.0 mm from each tooth. **B,** The two implant crowns to restore a space 14 to 20 mm are splinted together and usually made the size of premolars.

WWW.HIGHDENT.IR

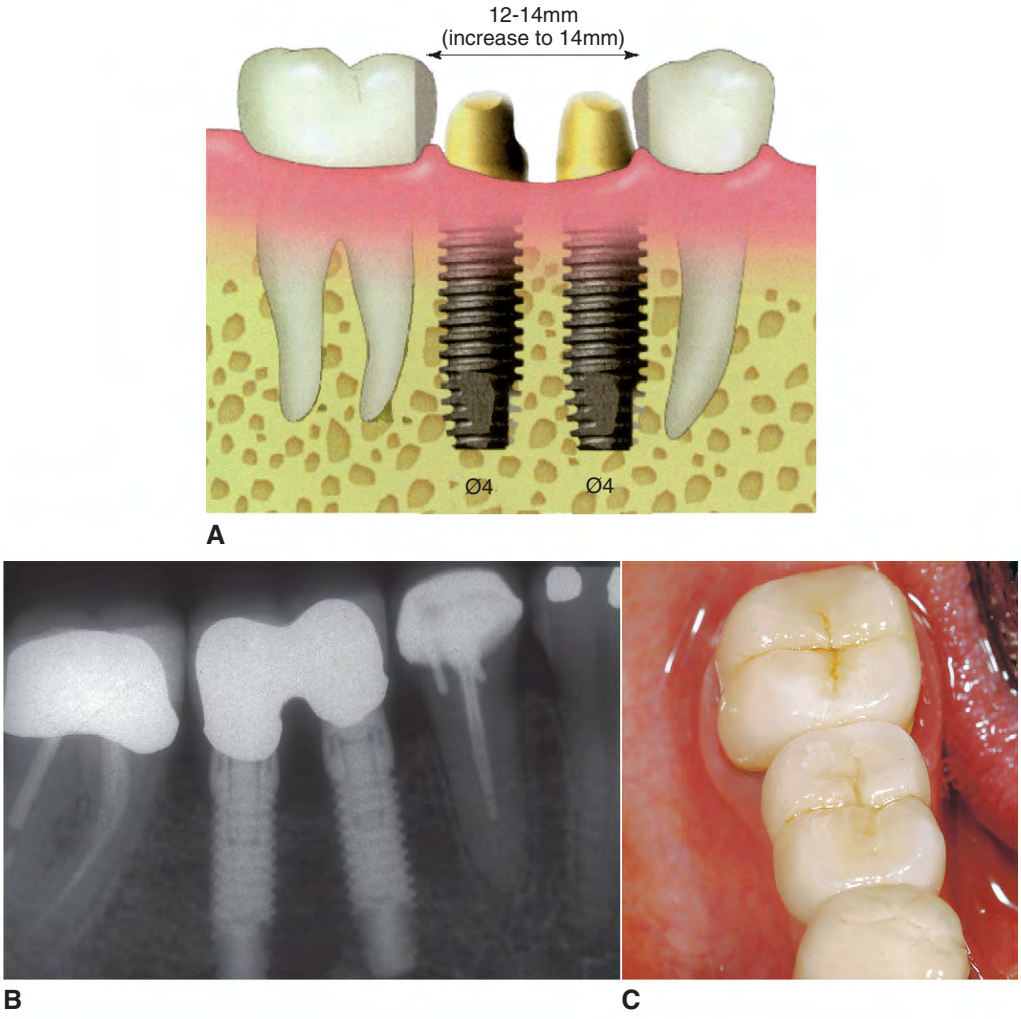


Figure 30-5 **A,** When 12 to 14 mm of space is available, the mesial and distal contour of the adjacent teeth is modified to gain addition space. **B,** A periapical radiograph of two 3.7-mm implants to replace one molar, in which the proximal contours of adjacent teeth were reduced. **C,** An intraoral view of the mandibular first molar, replaced by two 3.7-mm implants (BioHorizons Maestro Dental Implants).

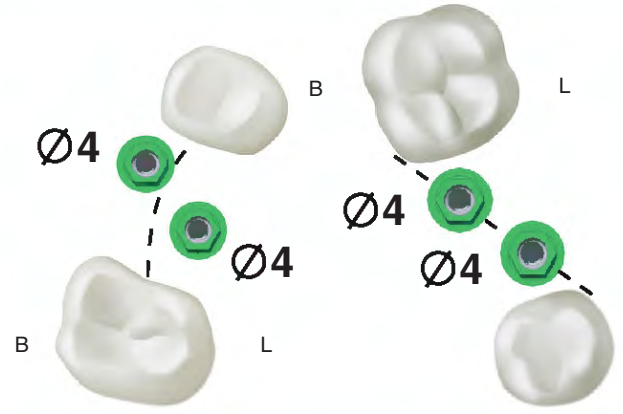
may be placed and an orthodontic spring incorporated in the transitional crown. The spring pushes the distal tooth more distal and, after orthodontic movement, the second implant may be inserted with less risk and improved hygiene between each implant. Another option is to reduce the space orthodontically and place only one implant and crown.

3. The implants may be offset with one implant placed buccal and the other implant on a diagonal toward the lingual[28] (Figure 30-6, *A*). The diagonal dimension increases the mesiodistal space by 0.5 to 1 mm. In the mandible, the most anterior implant is placed to the lingual aspect of the midcrest and the more distal implant is placed to the facial aspect to facilitate access of a floss threader from the vestibule into the intraimplant space. The occlusal contacts also are slightly modified on the buccal aspect of the mesial implant to occlude over the central fossa (Figure 30-6, *B*). In the maxilla, the anterior implant is placed facially and the distal implant palatally to improve esthetics. The distal occlusal contact is placed over the lingual cusp and the mesial occlusal contact is located in the central fossa position. The cervical esthetics of the maxillary molar are compromised on the distal half of the tooth to the benefit of greater intratooth distance and easier access for home care. This maxillary implant placement requires the intraimplant furcation to be approached from the palate, rather than the buccal approach, as the mandible.

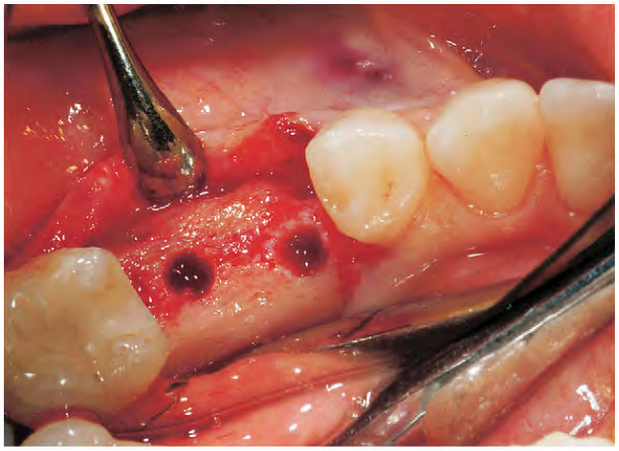
POSTERIOR SINGLE-TOOTH IMPLANT SURGERY

The ideal location to begin a surgical experience in implant dentistry is a posterior single tooth, out of the esthetic zone, and in abundant bone volume. Surgical success rates for single-tooth implants are often more than 98%. University reports show predoctoral students

WWW.HIGHDENT.IR



A



B

Figure 30-6 **A,** When the mesiodistal space is 12 to 14 mm, the implants may be offset to increase space between the implants. In the mandible *(left)* the distal implant is positioned more buccal and the mesial implant more lingual. In the maxilla, the mesial implant is more buccal and the distal implant is more lingual. **B,** An intraoral view of a mandibular first molar with offset implants. The distal implant is more buccal, which facilitates floss threader insertion for oral hygiene.

have achieved excellent results in these conditions.[28-31] For example, Bell et al. reported no failure in 120 implants during a 4-year period with selected predoctoral dental students.[28] Cummings et al. found senior predoctoral dental students had no implant failures of 71 implants during 5 years.[29]

Posterior single-tooth implants may be technically easier and have less risk than performing an endodontic treatment on a tooth with a single canal. Rather than working on a diseased pulp with different local conditions, an implant surgery is in healthy bone and an ideal clinical situation. In endodontics, the files and obturation should be within 0.5 mm of a tooth apex. Implants may be prepared and inserted 0.5 to 10 mm from an opposing landmark. Endodontics requires the removal of necrotic debris from an oblong canal, with lateral canals and root curvatures or dilacerations. An implant osteotomy is round in diameter and made with round-diameter drills and always a straight channel. Endodontics requires obturation with a plastic material, able to fill irregular-diameter and shaped canals. Underfills may result in failure of the procedure. Implant round-diameter osteotomies are obturated with a round-diameter implant precisely fabricated for the size of the osteotomy. Underfills of the osteotomy will fill in with bone next to the implant, with no clinical consequence. An implant surgery may be completely aborted at almost any time, and several months later the bone conditions are healed and similar to the original bone site conditions.

Table 30-1 Comparison Between Two-Stage, One-Stage, and Direct One-Stage Surgeries

TOPIC	TWO-STAGE	ONE-STAGE	DIRECT ONE-STAGE
Bone anatomy visual control	Yes	Yes	No
Osteotomy visual control	Yes	Yes	No
Cover screw/ PME	Below tissue	Above tissue	Above tissue
Bone graft	Yes	Maybe	No
Exposure to intraoral bacteria	No	Yes	Yes
Surgical appointments	2	1	1
Prosthetic connection/ implant platform level	At or below tissue More difficult	At or above tissue Easier	Above tissue Easier
Reposition keratinized facially	Yes	Yes	No
Transitional appliance	Yes	Yes-No	Yes-No
Stage II needed	Yes	No	No

PME, Permucosal healing extension.

SURGICAL PROCEDURE

There are three approaches for posterior single-tooth implant surgery: (1) two-stage surgery, (2) one-stage surgery, and (3) direct, one-stage surgery (Table 30-1).

Two-Stage Surgery

In an early learning curve, the two-stage surgical approach offers several advantages. This method provides direct vision of the crestal bone width and length. The direct observation of crestal bone ensures adequate width of bone is available for implant placement. The width of bone is often reduced after initial socket healing. If

WWW.HIGHDENT.IR

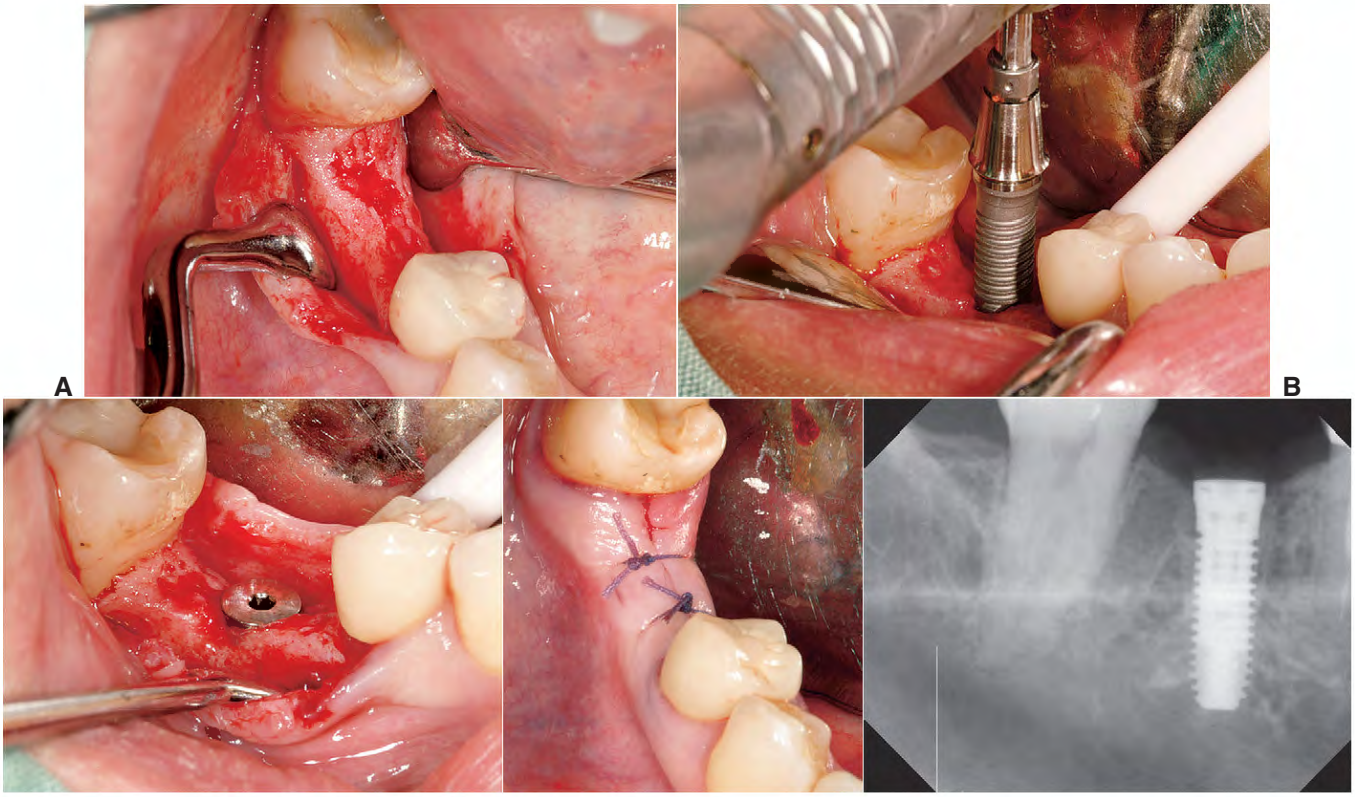


Figure 30-7 **A,** A two-stage surgery reflects the soft tissue and directly observes the underlying bone. **B,** A single-tooth implant is threaded into the osteotomy (BioHorizons Internal Dental Implant) for a posterior single-tooth replacement. **C,** A low-profile cover screw is inserted into the implant body. **D,** The first-stage surgery uses primary closure over the implant site during initial bone healing. **E,** A periapical radiograph of the implant in position to replace a single molar.

inadequate, a bone graft should be performed before implant insertion. In more experienced situations, the implant may be inserted at the same appointment as the bone graft. The implant is inserted at or slightly below the crest of bone in this technique. A low-profile cover screw is then inserted into the implant body. The tissues are then approximated over the implant for primary closure. A removable transitional prosthesis may be delivered post surgery when in the esthetic zone and in the absence of a bone graft (Figure 30-7).

The advantages of this approach include: (1) direct observation of crestal bone volume before osteotomy; (2) direct observation during osteotomy preparation; (3) ability to bone graft the site at the time of implant placement; (4) implant body healing at or below the crest of bone, which reduces risk of early loading during initial bone healing; (5) local hygiene issues or anaerobic bacterial infiltration are not critical factors during initial healing; and (6) the ability to deliver a soft tissue–borne transitional appliance in the esthetic zone.

The technique requires a second-stage surgery to uncover the implant body. The advantages of this procedure are the crestal and facial region may be directly evaluated and minor defects corrected before beginning the prosthetic reconstruction. On occasion, soft tissue has invaded the crestal zone of the implant, and a one-stage approach does not evaluate the region before restoration. The facial plate of bone may also resorb during initial healing and may be grafted at the reentry surgery.

One-Stage Surgery

A one-stage surgery uses a similar incision and reflection technique to observe directly the crestal bone volume. However, at the conclusion of the implant surgery, a permucosal healing element (PME) is placed into the implant body. The implant body also is usually placed slightly above the crest of the bone. The soft tissue is then placed around the PME (Figure 30-8). The advantages of the one-stage surgery are:

1. The soft tissue matures while the bone interface is healing. This permits the restoration to be fabricated with complete assessment of the soft tissue profile. In the two-step procedure, the soft tissue is less mature when the prosthesis is fabricated, as a stage II surgery is required to uncover the implant and place a PME or abutment.
2. A second surgical procedure and suture removal appointment are not necessary. This saves the patient

Text continued on p.677.

WWW.HIGHDENT.IR

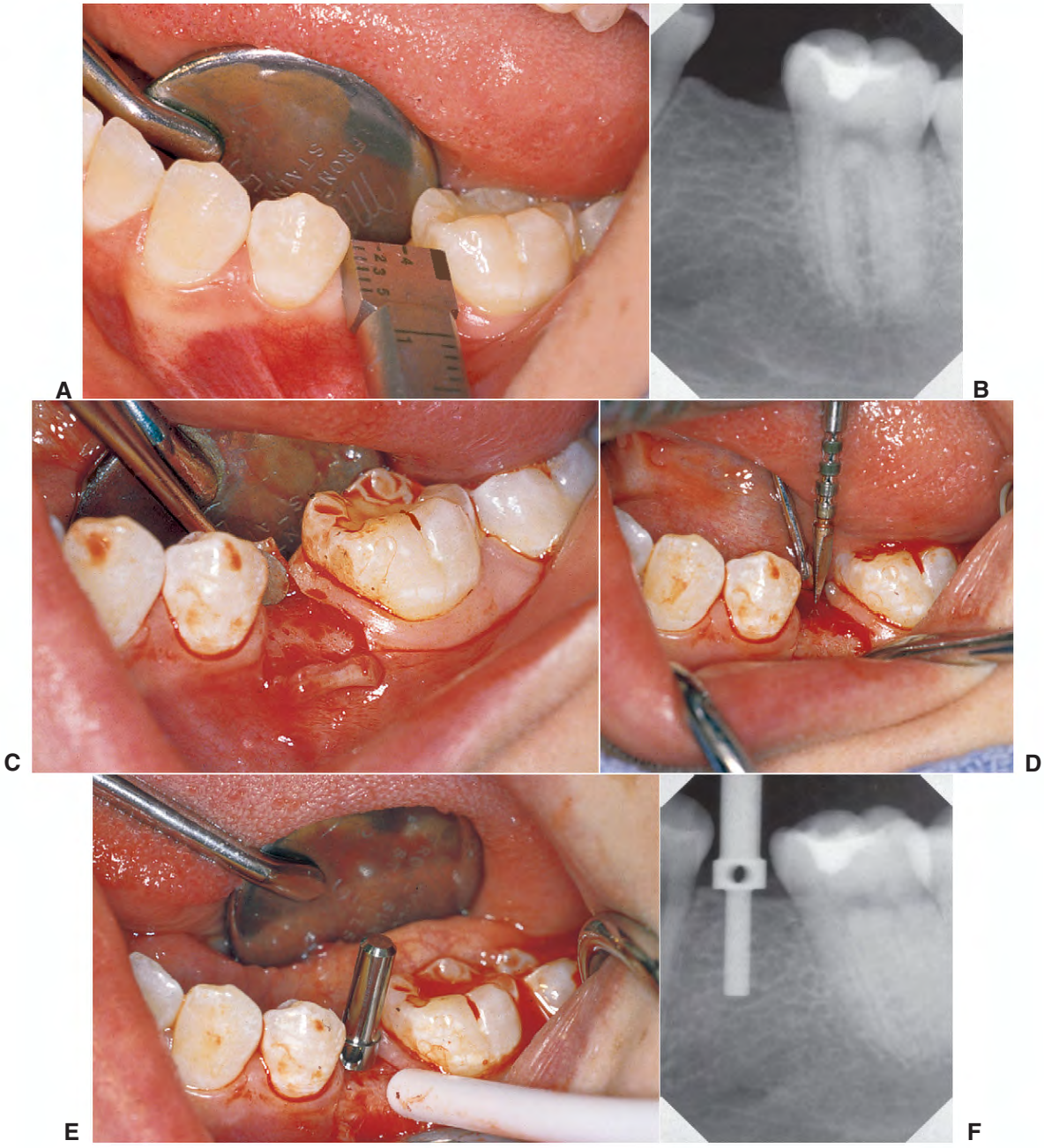


Figure 30-8 **A,** The mesiodistal dimension of a missing tooth for a single-tooth implant should be at least 6.5 mm in the posterior region of the mouth (measured between the CEJs). This permits an implant at least 3.5 mm in diameter. **B,** A periapical radiograph is used to evaluate available bone height. It should be at least 11 mm from the crest to the opposing landmark (i.e., the mandibular foramen and mandibular canal). **C,** In the present case, a one-stage surgical approach may be selected instead of a direct one-stage approach to reposition the keratinized tissue facially. The soft tissues are reflected for direct observation of the bone volume and dimension during the preparation of the implant osteotomy. **D,** A 2-mm-diameter, end-cutting starter drill is used initially under copious cooled sterile saline. **E,** A 2-mm-diameter, 9-mm-long force direction indicator with a 4-mm-diameter "stop" is placed in the osteotomy. **F,** A periapical radiograph is made of the force direction indicator (depth gauge). The adjacent roots are evaluated in relation to the device, and the distance from the opposing landmark is evaluated.

Continued

WWW.HIGHDENT.IR

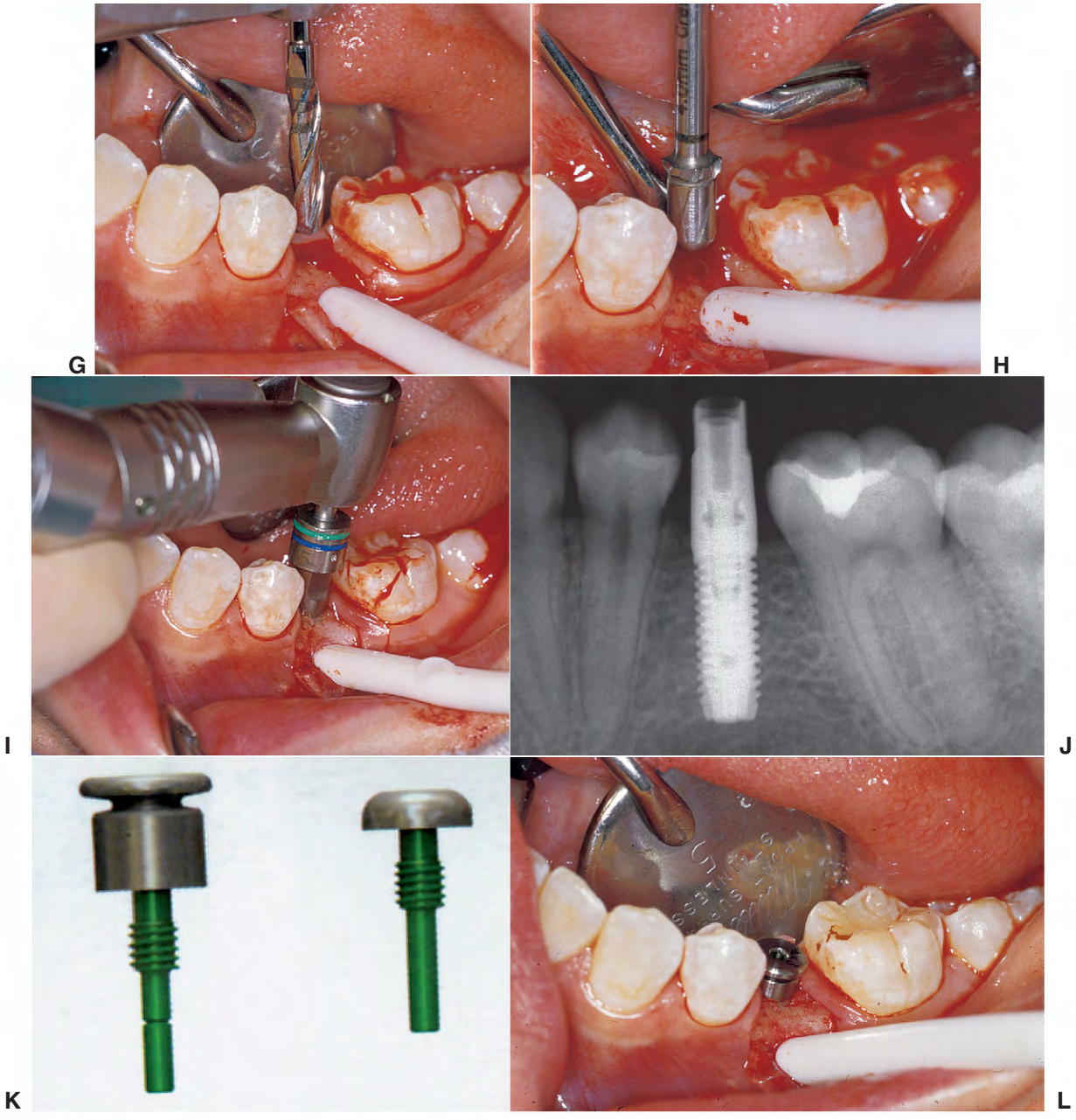


Figure 30-8, cont'd **G,** The osteotomy is prepared according to the drill sequence indicated for the bone density specific to the site. **H,** A crestal side-cutting drill is used to prepare the crest module dimension of the implant. **I,** The implant is inserted into the osteotomy with a hand piece at reduced rpm. **J,** A postoperative radiograph is made when the implant is in its final position. It is evaluated relative to depth and the opposing landmark, the adjacent tooth roots, and for crestal position. **K,** In a two-stage surgical approach, a low-profile healing cap is threaded into the implant body *(left).* After hard and soft tissue healing, a higher-profile permucosal extension (PME) is placed with a second surgery *(right).* A one-stage surgery places the PME (right side) at the initial surgery. The suture groove region decreases the risk of soft tissue growing over the PME during healing (BioHorizons Dental Implants). **L,** In a one-stage surgical approach, a PME is placed after the insertion of the implant.

Continued

WWW.HIGHDENT.IR

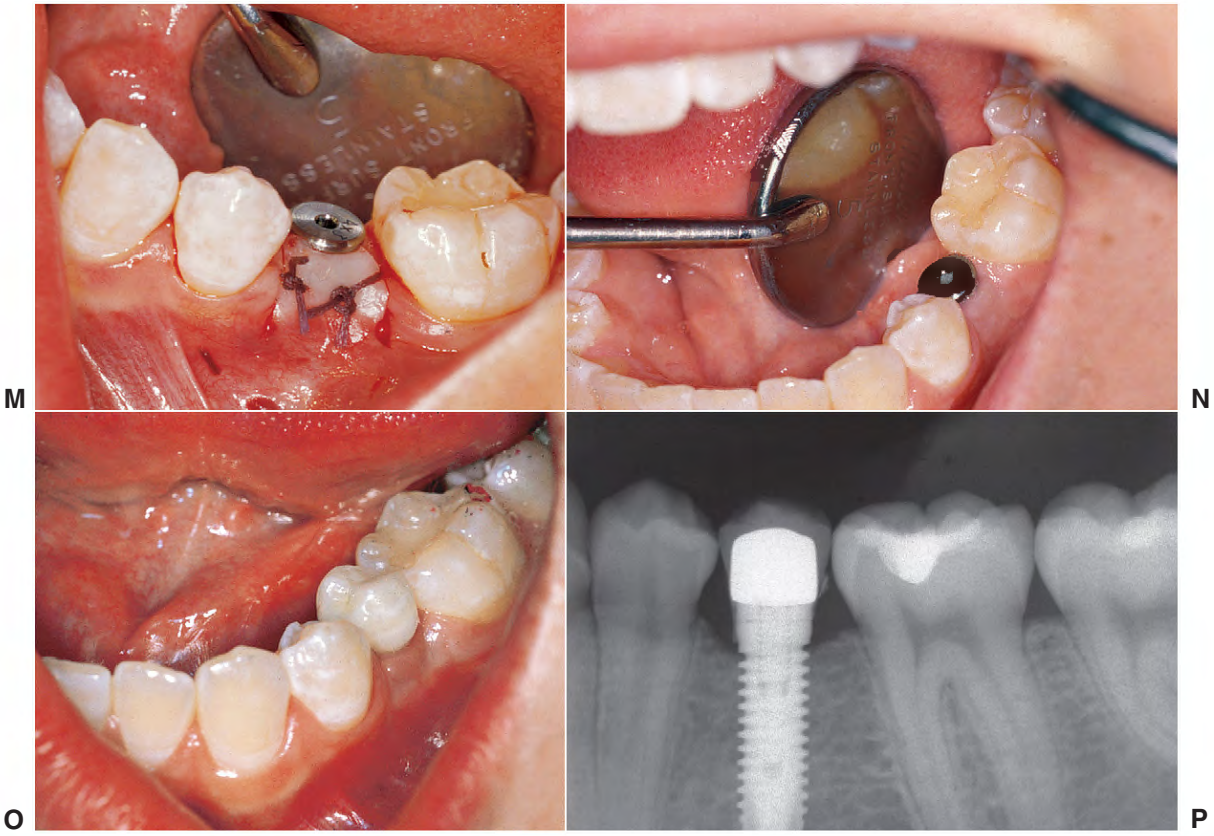


Figure 30-8, cont'd **M,** The tissue is sutured around the PME during the initial implant surgery for a one-stage surgical approach. **N,** After healing, the prosthesis fabrication may be started. No second-stage incision or tissue reflection is needed in this case. **O,** Final prosthesis delivery. **P,** Periapical radiograph of final restoration.

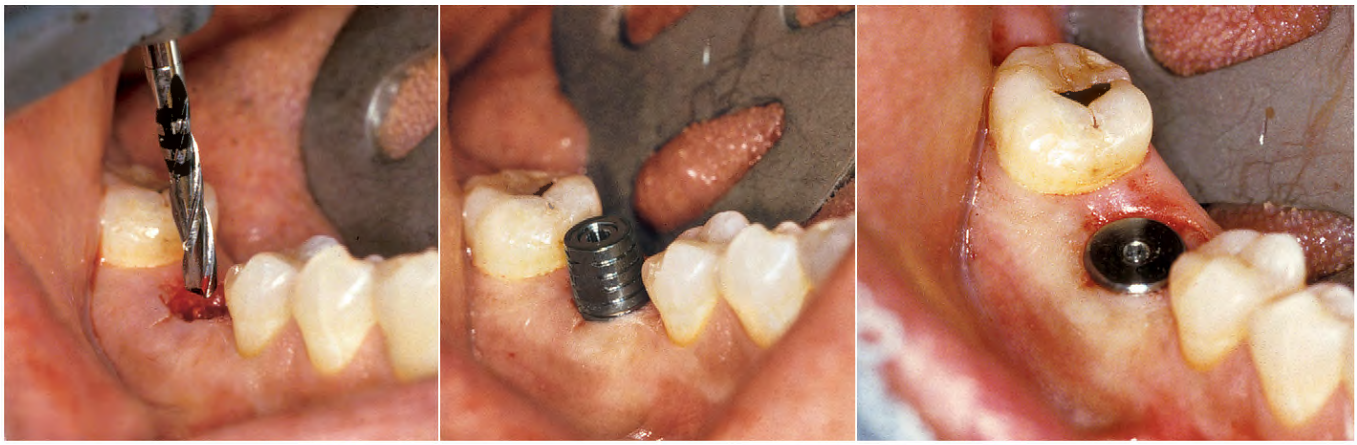
discomfort and the surgeon two appointments (stage II uncovery and suture removal).

3. The abutment to implant connection may be placed above the crest of bone in the one-stage surgery. This higher location of the implant-abutment connection may reduce some of the early crestal bone loss in a developing implant interface. In addition, Weber observed an improved hemidesmosome soft tissue–implant connection when the components above the bone were not removed and reinserted, as when the healing gap connection is below the bone.[32] Depending upon crest module design, the one-stage surgical approach may have less early crestal bone loss.
4. The higher-profile implant body also allows the restoring dentist to attach the prosthetic abutment with greater ease and tactile ability. Impression caps also may be inserted when the abutment connection is above the bone, which further simplifies the restorative procedure (Table 30-1).

An advantage of the one-stage surgery, compared with a direct or flapless one-stage approach, is the attached keratinized tissue may be repositioned to ensure an adequate zone on the facial aspect of the PME and implant abutment. In addition, direct observation of the bony crest permits bone grafting and a precise implant crest module position relative to the crest because it can be directly observed. In theory:

1. The higher-profile PME is more at risk of loading during healing, especially when an overlying soft tissue–borne transitional restoration is worn. Therefore a disadvantage may be a higher healing failure rate. However, clinical studies of one-stage surgery indicate similar implant survival rates in good bone volumes and quality.
2. The amount of keratinized tissue should be adequate to reposition at least 1.5 mm to the facial aspect of the PME healing cap.
3. When a bone graft is considered at the time of implant insertion, primary closure of the soft tissues improves the environment to grow bone. Therefore the one-stage approach is less indicated under these conditions.
4. The implant PME may be unesthetic when the maxillary esthetic zone of the mouth is in a premolar site.

WWW.HIGHDENT.IR



A B C

Figure 30-9 **A,** A direct one-stage surgery uses a flapless approach. A core of soft tissue is removed over the implant site. The osteotomy is prepared in the center of the core. **B,** The implant is threaded into position. It is often difficult to determine the position of the implant in relation to the crest of the bone, as it is not directly visible below the soft tissue. **C,** A permucosal healing element (PME) is threaded into position and seals the soft tissue opening.

Direct (Flapless) One-Stage Surgery

The one-stage surgical approach may be modified with a direct flapless surgical approach. This technique does not reflect the crestal soft tissue. Instead, a core of keratinized tissue (the size of the implant crest modale diameter) is removed over the crestal bone. The implant osteotomy is then performed in the center of the core of the exposed bone. This technique requires no sutures around the PME. The advantages of this technique are similar to a one-stage surgical approach. In addition, there is less soft tissue trauma because the tissues are not reflected. As a result, discomfort, tenderness, and swelling are usually minimal (Figure 30-9).

The primary disadvantage of the direct approach is the inability to assess the bone volume before or during the implant osteotomy or insertion. Therefore this technique should only be used when the bone width is abundant (>8 mm). Bone grafting needs and procedures cannot be precisely evaluated. The soft tissue around the implant site should be ideal in amount of attached keratinized mucosa, as the soft tissue pouch is over the bone site, not in a region related to the soft tissue. Often, the keratinized tissue is reduced on the buccal half of the ridge and the tissue punch may inadvertently remove all the keratinized tissue on the facial aspect of the implant. As the crest of the ridge is below the soft tissue, it is difficult to see lines on the drill to access the depth of drilling. Therefore stops on the drill are particularly beneficial. The surgeon also has trouble assessing the location of the implant crest module in relation to the crest of bone because it is also below the soft tissue. In addition, the interdental papillae may not be elevated with this technique. Therefore the soft tissue drape should be ideal in volume of keratinized tissue, both faciolingually and mesiodistally.

STEP-BY-STEP SURGERY

Implant Site Evaluation

The missing tooth space is first evaluated from a mesiodistal dimension, at the level of the CEJ of each adjacent tooth. This dimension should be at least 6.5 mm in width. The selected implant diameter is 3 mm narrower than this dimension (i.e., at least 3.5 mm). The buccolingual crestal dimension is then evaluated with the soft tissue intact. The edentulous site should be at least 9 mm wide with the tissue intact to consider a direct (flapless) one-stage surgery (assuming the tissue thickness in each site is 1 mm). When a smaller dimension is present a one- or two-stage procedure is indicated and the ridge most often will require a bone graft, either before or in conjunction with implant insertion. If the facial aspect of the implant body is exposed at insertion, a two-stage approach is indicated, along with a membrane bone graft.

A periapical radiograph is then used to evaluate the missing tooth site. The minimum radiographic bone height should be 11 mm from the crest of the ridge to the opposing landmark (i.e., mental foramen and mandibular canal in the mandible and maxillary sinus in the maxilla). This dimension permits a 9-mm-long implant to be inserted at least 2 mm above the opposing landmark in the mandible. The roots of the adjacent teeth should not invade the vertical column of bone

WWW.HIGHDENT.IR

in the implant site. The 6.5-mm minimum mesiodistal dimension should exist for at least 11 mm between the adjacent roots. The minimum implant length selected is usually 9 mm, and the longest length is at least 2 mm less than the available bone height dimension in the mandible, closer in the maxilla, and may even proceed to the sinus floor.

Soft Tissue Reflection

Once the bone volume is assessed and the implant surgical option is identified (i.e., two-stage, one stage, direct), the implant procedure may commence. In the one- or two-stage approach, the soft tissue reflection is similar. A scalpel (#12 and/or #15) is used to incise the mid-crestal tissues. At least 1.5 mm of keratinized tissue should be left on the buccal to this incision line.

The incision line most often is extended through the interproximal and sulcular tissues of the adjacent teeth. This permits complete assessment of the crestal bone volume when the tissues are reflected. A Buser or Molt elevator is used to make a full-thickness soft tissue reflection and expose the crestal bone and a few millimeters of the lateral aspects of the implant site. The buccolingual dimension of bone is measured and confirmed to be greater than 7 mm in width. If the site is less than 7 mm wide, a division B membrane and layered-approach bone graft are used to augment the site, either in conjunction with implant insertion or as a separate surgical procedure before placing the implant.

Implant Osteotomy

A 2-mm-diameter, end-cutting starter drill is used in the mesiodistal and buccolingual centers of the crestal bone for implants in the mandible or out of the esthetic zone in the maxilla. The osteotomy is made with an electric motor at a preferred speed of 2500 rpm under copious amounts of chilled saline irrigant. The osteotomy is made 7 to 9 mm deep in the bone (Figure 30-10, *A*).

Once the initial osteotomy is prepared, it is assessed for proper position. If incorrect, the hole may be "stretched" to the proper location by a side-cutting Linderman bur (Figure 30-10, *B*). This bur makes the hole oblong toward the corrected center position. The following end-cutting drills may then be positioned into the correct position and create a round osteotomy of proper size before implant placement.

A 2-mm-diameter direction indicator (depth gauge) is then inserted into the osteotomy and the angulation and position assessed (Figure 30-10, *C*). When the "stop" on the direction indicator is the same size as the implant (i.e., 4-mm diameter), the final implant position buccolingual and mesiodistal also may be assessed, making sure it is at least 1.5 mm from each tooth and 1.5 mm from the outer crestal cortical plates. If it is too far in any direction, a side-cutting Linderman drill may stretch the osteotomy toward a more proper position. A periapical radiograph is made with this measuring tool. The radiograph ensures the proper angulation and position of the implant relative to the adjacent tooth roots. The final diameter of the implant may now be selected using the guidelines presented earlier. The opposing landmark is also evaluated. The implant is most often placed 2 mm or more from the mandibular neurovascular canal or 0.5 mm from the floor of the maxillary sinus and at least 9 mm in length. Although implants may be inserted closer than 2 mm of these landmarks without compromise, the 2-mm surgical rule is a common safe position in the mandible. When the opposing landmark is 14 to 17 mm farther away from the depth gauge and the osteotomy may be safely made deeper than 9 mm, the pilot 2-mm diameter drill may be reinserted into the osteotomy and prepared to a 12- or 15-mm length. Rarely is more than a 15-mm-long implant required.

When the opposing landmark is safely beyond the end of the osteotomy depth, the drills often proceed end-cutting 1 to 2 mm beyond the eventual depth of the implant. The slight recess area in the apical end of the osteotomy serves several purposes:

1. Bone debris may fill the region during a self-tapping implant body placement, because the threads scrape the bone as the implant is inserted. As a result, when the implant comes to its final position, the bone debris does not impede its progress.
2. The implant may be rotated in final position to align the flat section of the antirotation crest module, which helps identify the position location of prosthetic components.
3. The lingual border of the osteotomy is often higher than the facial bone level. A stop used on a drill for the implant length prevents the drill from proceeding to the facial crest, and the crest module height will be determined by the higher lingual plate of bone, which positions the facial aspect of the implant platform above the bone.

A 2.5-mm-diameter, end-cutting twist drill is then used in the initial osteotomy to the depth established. The drill also rotates at 2500 rpm with copious amounts of chilled sterile saline. The osteotomy location and angulation are reassessed at this point. A slight correction of position or angulation with a Linderman drill is possible with little consequence or risk. A 3-mm-diameter twist drill is then used (depending upon implant design and final diameter) to the desired length, followed by a 3.4-mm twist drill. Once again, the proper location and angulation of the osteotomy is verified (Figure 30-10, *D*, *E*, *F*, *G*). This is the final diameter for a 4-mm-diameter implant (BioHorizons Dental Implants). The final osteotomy diameter for a 4-mm-diameter implant is related to the implant diameter and manufacturer. For example, a Zimmer implant uses a 3.2-mm diameter for a 4-mm-diameter implant. Most implant drill kits clearly

WWW.HIGHDENT.IR

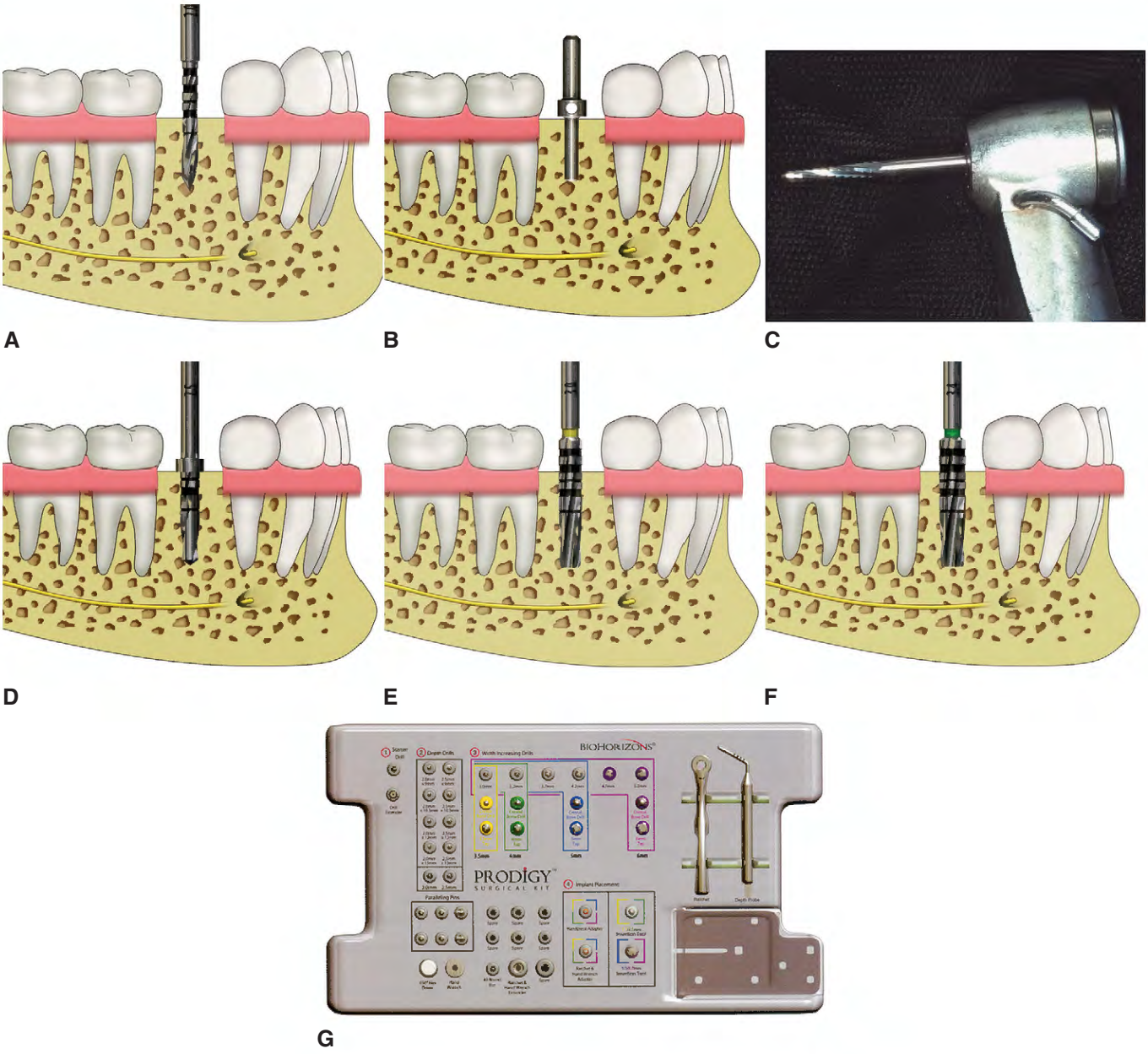


Figure 30-10 **A,** The soft tissue is reflected from the crest of the ridge and the width of bone is directly assessed. A 2-mm-diameter, end-cutting starter drill is used under copious sterile saline. **B,** A 2-mm-diameter, 9-mm-long force direction indicator is positioned into the initial osteotomy. **C,** A side-cutting Linderman drill is used to modify the initial osteotomy started with the pilot drill. **D,** A 2-mm-diameter pilot drill is inserted into the initial osteotomy and prepares the bone from 12 to 15 mm when the opposing landmark permits. A 2.5-mm, then a 3-mm drill sequence prepares the osteotomy. **E,** A 3-mm side-cutting twist drill widens the osteotomy. **F,** A 3.4-mm-diameter, end-cutting twist drill is used to prepare the final diameter for a 4-mm implant body. **G,** An implant drill kit with color coding most often identifies the drill sequence and the final diameter related to each implant size (BioHorizons Maestro Dental Implants).

Continued

identify the drill sequence and final osteotomy diameter related to each diameter implant (Figure 30-10, *H*).

Most implant crest modules are larger in diameter than the implant body. The larger diameter often requires a side-cutting crest module drill in D1 or D2 crestal bone situations to prepare the crestal aspect of the implant osteotomy. This drill is not used when the bone density is poor (D3, D4) (Figure 30-10, *I*).

In dense bone types, some implant thread designs require a bone tap or thread former to prepare the threads in the bone before implant insertion (Figure 30-10, *J*). When the bone is soft (D3 or D4) or implant thread design is shallow (e.g., 0.2 mm versus 0.5 mm), a thread former is not required. Most often for single-tooth implants, the thread formers or taps should use a high-torque, slow-speed handpiece and be rotated at less than 30 rpm into the bone. Irrigation is also of benefit to help lubricate and clean the bone tap and osteotomy site of debris during this process.

The implant site may then be prepared for implant insertion. The osteotomy is lavaged and aspirated to remove bone debris and stagnant blood. This reduces

WWW.HIGHDENT.IR

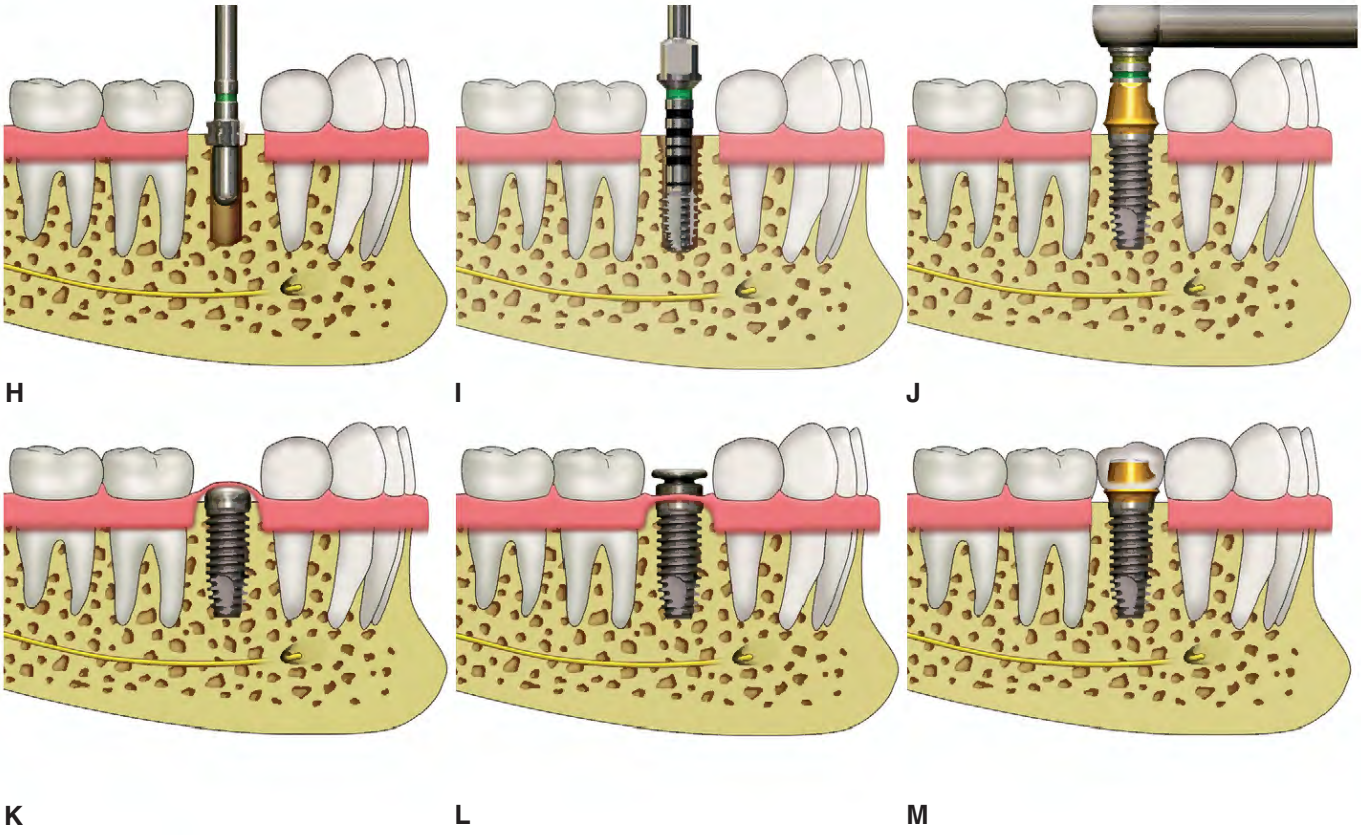


Figure 30-10, cont'd **H,** A side-cutting crestal bone drill prepares the top of the implant osteotomy in D1 and D2 bone, as the crest module of the implant is larger than the implant body. **I,** A bone tap is used in denser bone to prepare thread forms before implant insertion. **J,** A slow-speed, high-torque hand piece is usually used to thread the implant into the bone at 30 rpm or less. **K,** The abutment/mount of the implant body is removed once the implant is in final position. A low-profile cover screw is inserted for a two-stage surgical approach. **L,** A permucosal extension (PME) is inserted into the implant body for a one-stage surgical approach, which is used most often in the posterior region of the mouth. **M,** The most common, undisturbed healing time for a single-tooth implant is 4 months. At this point, both the hard and soft tissues are adequately healed and the final restoration may commence.

the risk of these materials being forced into the bone marrow spaces or neurovascular channels during implant insertion, thus causing hydrostatic pressure. This pressure may increase the devital zone of bone around the implant or even cause short-term anesthesia when the implant site is in the vicinity of the mandibular canal.

The implant is rotated less than 30 rpm into position by a low-speed, high-torque hand piece or a hand ratchet (Figure 30-10, *K*). The implant should not require a torque greater than 35 N-cm while threading into position. If this is necessary, the implant should be unthreaded and a bone tap used before insertion. The implant is usually inserted slightly above (one stage) or below (two stage) the crest of the bone, depending upon a two-stage or one-stage surgical approach, as previously described. The implant should be rigid upon placement, with no observable mobility under slight compressive forces. The implant should not be tightened into the osteotomy, such as a nut onto a bolt. A torque value up to 35 N-cm is considered safe with most threaded implant designs. However, excessive pressure to the bone may cause pressure necrosis and increase the devital zone of bone around the implant during healing. When in doubt, the implant may be unthreaded one half turn with a hand ratchet, to ensure it is not too tight within the osteotomy.

A postinsertion periapical radiograph is made once the implant is in final position. The radiograph is evaluated as to implant depth and the opposing landmark, the adjacent roots and the position of the crest module and the crest of the bone. Any correction is most easily made at this time, rather than attempting to modify the position at a later date.

It should be noted that the implant surgery may be aborted at any stage of the procedure. Unlike a crown preparation or endodontic therapy, an aborted procedure will allow the bone to reform in the osteotomy and allow the procedure to be performed with a similar condition as the initial surgery at a later date. Therefore if the implant position is not within the range of ideal, it may be removed, and a new implant reinserted several months later in a more ideal position.

The abutment mount of the implant body is then removed. When a two-stage implant insertion is used, a low-profile cover screw is inserted into the implant body (Figure 30-10, *L, M*). When the cover screw is in final position, it may be slightly tightened, loosened, and

WWW.HIGHDENT.IR

tightened again. The implant should not rotate during this procedure. A similar procedure is performed with a PME, when a one-stage or direct surgical approach is used. In the posterior of the mouth, a one-stage surgical approach is used most often.

In a one- or two-stage surgery, two interrupted sutures approximate the soft tissue for primary closure. Most often a 4-0 polyglycolic acid (PGA) material is used. Slight pressure to the approximated tissues for 4 to 6 minutes decreases bleeding and improves the adaptation of the tissue to the bone and PME. When not in the esthetic zone, no transitional device is worn. Postoperative instructions include the use of periodic ice to the region for 3 days, and postoperative medication is similar to a routine extraction. The sutures are removed after 10 to 14 days, although 7 days are usually sufficient. The most common undisturbed healing time for a single-tooth implant before restoration is 4 months. At this point, the hard and soft tissues are adequately healed and the final restoration may commence (Figure 30-10, *N*).

In the past, the most common choice to replace a missing posterior tooth was a three-unit fixed partial denture. The choice of not replacing the tooth is a more frequent option for mandibular second molars, but this option also may be selected when the intratooth space is small and the occlusion prevents migration. Today, with the improvements in implant materials, design, surgical approach, and prosthetic guidelines, and with reported success rates greater than 97%, the use of implants to replace a single tooth is often the treatment of choice. Improved hygiene, less risk of decay of the adjacent teeth, less endodontic risk, less risk of adjacent tooth loss, maintenance of bone, and prosthesis longevity all favor an implant restoration compared with the three-unit fixed partial denture prosthesis.

Posterior single-tooth implant surgery in abundant bone is one of the easier processes in implant surgery. It is often easier than performing endodontics for a single rooted tooth. Early learning curves are short, and very high surgical success rates are rapidly obtained. The guidelines within the chapter for implant body size (width and length) have proven that predictable long-term surgical success for single-tooth implants is more than 99%.

References

1. Palmquist S, Swartz B: Artificial crowns and fixed partial dentures 18 to 23 years after placement, *Int J Prosthodont* 6:279-205, 1993.
2. Holm C, Tidebag P, Tillberg A et al: Longevity and quality of FPDs: a retrospective study of restorations 30, 20 and 10 years after insertion, *Int J Prosthodont* 6:283-289, 2003.
3. Priest GF: Single tooth implants and their role in preserving remaining teeth: a 10-year survival study, *Int J Oral Maxillofac Implants*14:181-188, 1999.
4. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
5. Shillingburg HT, Hobo S, Whitsett LD et al: *Fundamentals of fixed prosthodontics*, ed 3, Chicago, 1997, Quintessence.
6. Cheung GSP, Dimmer A, Mellor R et al: A clinical evaluation of conventional bridgework, *J Oral Rehabil* 17:131-136, 1990.
7. Meskin LH, Brown LS: Prevalence and patterns of tooth loss in US employed adult senior populations, *J Dent Educ* 52:686-691, 1988.
8. Hayden JW: Dental health services research utilizing comprehensive clinical data bases and information technology, *J Dent Educ* 61:47-55, 1997.
9. Shugars DA, Bader JD, White BA et al: Survival rates of teeth adjacent to treated and untreated posterior bounded edentulous spaces, *J Am Dent Assoc* 129:1085-1095, 1998.
10. Becker W, Becker BE: Replacement of maxillary and mandibular molars with single endosseous implant restorations: a retrospective study, *J Prosthet Dent* 74:51-55, 1995.
11. Becker W, Becker BE, Alsuwyed A et al: Long-term evaluation of 282 implants in maxillary and mandibular molar positions: a prospective study, *J Periodontol* 70:896-901, 1999.
12. Simon RL: Single implant-supported molar and premolar crowns: a ten-year retrospective clinical report, *J Prosthet Dent* 90:517-521, 2003.
13. Levin L, Laviv A, Schwartz-Arad D: Long-term success of implants replacing a single molar. *J Periodontol* 77: 1528-1532, 2006.
14. Misch CE, Misch-Dietsh F, Steigenga J et al: Posterior implant single tooth replacement and status of abutment teeth: multi-center 10 year retrospective report (submitted for publication).
15. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses. *J Prosthet Dent* 90:121-132, 2003.
16. Salinas TJ, Eckert SE: In patients requiring single-tooth replacement, what are the outcomes of implant, as compared to tooth-supported restorations? *Int J Oral Maxillofac Implants* 22 (suppl):71-95, 2007.
17. Sullivan DY: Wide implants for wide teeth, *Dent Econ* 84:82-83, 1994.
18. Spray JR, Black CG, Morris HF et al: The influence of bone thickness on facial marginal bone response: stage 1 placement through stage 2 uncovering, *Ann Periodontol* 5:119-128, 2000.
19. Small PN, Tarnow DP, Cho SC: Gingival recession around wide-diameter versus standard-diameter implants: a 3- to 5-year longitudinal prospective study, *Pract Proceed Aesthet Dent* 13:143-146, 2001.
20. Malevez C, Hermans M, Daelemans P: Marginal bone levels at Brånemark system implants used for single tooth restoration. The influence of implant design and anatomical region, *Clin Oral Implants Res* 7:62-69, 1996.
21. Balshi TJ, Hernandez RE, Pryzlak MC et al: A comparative study of one implant versus two replacing single molar, *Int J Oral Maxillofac Implants* 11:372-378, 1996.
22. Balshi TJ, Wolfinger GJ: Two-implant-supported single molar replacement: interdental space requirements and comparison to alternative options, *Int J Periodontics Restorative Dent* 17:426-435, 1997.
23. Bakaeen LG, Winkler S, Neff PA: The effect of implant diameter, restoration design, and occlusal table variations on screw loosening of posterior single-tooth implant restorations, *J Oral Implantol* 27:63-72, 2001.
24. Geramy A, Morgano SM: Finite element analysis of three designs of an implant-supported molar crown, *J Periodontal Dent 92*:434-440, 2004.

WWW.HIGHDENT.IR

25. Rangert B, Krogh PH, Langer B et al: Bending overload and implant fracture: a retrospective clinical analysis, *Int J Oral Maxillofac Implants* 10:326-334, 1995.
26. Langer B, Langer L, Herrman I et al: The wide fixture: a solution of special bone situations and a rescue for the compromised implant, *Int J Oral Maxillofac Implants* 8:400-408, 1993.
27. Tarnow DP, Cho SC, Wallace SS: The effect of inter-implant distance on the height of inter-implant bone crest, *J Periodontol* 71:546-549, 2000.
28. Bell FA 3rd, Jones AA, Steward KL: A clinical implant program in the predoctoral curriculum, *J Dent Educ* 55: 169-171, 1991.
29. Cummings J, Arbree NS: Prosthodontic treatment of patients receiving implants by predoctoral students: five-year follow-up with IMZ system, *J Prosthet Dent* 74:56-59, 1995.
30. Minsk L, Polson AM, Weisgold A et al: Outcome failures of endosseous implants from a clinical training center, *Compend Contin Educ Dent* 17:848-850, 852-854, 856, 1996.
31. Kohavi D, Azran G, Shapira L et al: Retrospective clinical review of dental implants placed in a university training program, *J Oral Implantol* 30:23-29, 2004.
32. Weber HP, Fiorellini JP: The biology and morphology of the implant-tissue interface, *Alpha Omegan* 85:61-64, 1992.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 31

Root Form Surgery in the Edentulous Anterior and Posterior Mandible: Implant Insertion

Carl E. Misch

The primary goal of root form surgery is to insert an endosteal implant into the available bone of an edentulous site in the proper location and angulation so that it may be used as a prosthetic abutment. An endosteal implant, made of biocompatible material, will predictably obtain rigid fixation when specific surgical principles are followed. The soft tissue should cover or approximate the implant for primary healing. The bone should receive as little trauma as possible. The smaller the devital zone, which forms around an implant subsequent to surgical trauma, the more likely rigid fixation will occur. The bone should initially be within 30 to 40 µm of the implant body,[1] which should be rigidly fixated and not move during healing. Movement of 100 µm or more to the bone-implant interface has been shown to result in intervening fibrous tissue.[2] The surgical site should remain free of infection, which may result in a bacterial smear layer on the implant body and impair the development of a close bone-implant contact during healing. The health, volume, and density of the recipient bone are critical to the preparation, fixation, and healing of an endosteal implant.

Figure 31-1 The most common position for root form implants in an edentulous mandible is between the mental foramina.

There are more than 100 types of root form implant designs readily available on the commercial market. A consistent philosophy is the desire for clinical rigid fixation corresponding microscopically to a direct bone-implant interface, without intervening fibrous tissue on a major portion of the implant body.[3] The surgical procedures for endosteal root form placement are described in this book by region, bone volume, and bone density. This chapter addresses a generic surgical approach to the anterior and posterior mandible in adequate volume (Division A or B) and quality of bone (D2) for the placement of a screw-type root form implant.

SURGICAL APPROACH TO THE ANTERIOR MANDIBLE

The most common location for root form implants in the completely edentulous mandible is between the mental foramina, in the region of the mandibular symphysis (Figure 31-1). The prosthesis type and amount of support for the patient force factors and bone density are first determined. If a fixed or RP-4 prosthesis is planned, most often five root form implants are usually indicated (see Chapter 14) in the A, B, C, D, and E positions (starting from the right of the patient to the left).[4] Posterior implants may also be indicated, depending on the existing force factors and other parameters.

Panographic radiographs or computed tomography (CT) scan imaging is evaluated for potential pathology, bone volume, and anatomical landmarks such as mental foramina and crest of ridge. A lateral radiographic view may be taken with a transitional prosthesis in place, which permits the evaluation of bone volume in height, the angulation of the bone in the midline, and the crown/remaining bone height ratio. A surgical template is fabricated before surgery to indicate the incisal edge position of the final restoration and help determine

WWW.HIGHDENT.IR

Box 31-1 Anesthesia Block: Bilateral Akinosi

Lidocaine 2%, 1:100,000 epinephrine
Marcaine 0.5%, 1:200,000 epinephrine
- Higher "hit rate"
- Fewer reinjections at end of surgery
- Less postoperative pain
- Marcaine possibly at end of surgery

proper implant location and angulation for the specific prosthesis (see Chapter 13). A standard surgical setup is prepared (see Figure 31-56).

Patient Preparation

The patient is prepared for surgery following the pharmacologic protocol typical for implant surgery (see Chapter 21). Because two carpules have a higher "hit" rate for block anesthesia, compared with one carpule, bilateral mandibular block anesthesia is administered with both lidocaine 2% 1:100,000 epinephrine and a long-acting anesthetic, such as bupivacaine (Marcaine) 0.5% with 1:200,000 epinephrine (Box 31-1). The use of a long-acting anesthetic is beneficial in three ways: (1) the added volume of anesthetic increases the success of the mandibular block; (2) a full arch mandibular implant insertion surgery may last 2 hours; therefore repeated injections at the end of the surgery are less indicated; and (3) in addition, long-acting anesthetics may reduce postoperative pain. In fact, it is advantageous to readminister the long-lasting block anesthetics at the conclusion of the surgery when its duration is longer than 1 hour.[5]

Akinosi Technique

The Akinosi technique (closed mouth) for mandibular anesthesia is preferred in the completely edentulous patient, because the occlusal plane and opposing premolar position used as references for the traditional Halsted dental injections are unavailable or inaccurate (Figure 31-2).[6] The Akinosi block procedure is administered with the mouth in an almost closed position or, because the patients often wear a denture, in an approximate occlusal vertical dimension. In addition, it is a benefit when the patient positions the jaw toward the side of the injection (i.e., slide the jaw to the right for a right mandibular block). A long, 27-gauge needle is used in the syringe. The needle is bent 30 degrees near the base, so the needle direction on a horizontal plane will be away from the midline. This is advantageous because the ramus flares lateral as it proceeds distal. The syringe and needle are placed parallel to the occlusal plane, at the height of the maxillary mucogingival junction. The needle penetrates approximately half its length (25 to 30 mm) before aspiration and injection of anesthetic. There is no bony landmark.

The Akinosi block is usually less painful for the patient, because the anesthetic fluid is injected into the top of the pterygoid triangular space, which has more room for the solution. In addition, the top of this triangular space has fewer muscle fibers in the pathway of the injection compared with the penetration site of the Halsted block and therefore is associated with less discomfort (Box 31-2).

Additional injections of anesthesia with the administration of local anesthetic (such as lidocaine 2% with epinephrine 1:100,000) by infiltration in the labial, lingual vestibules, and the inferior border of the

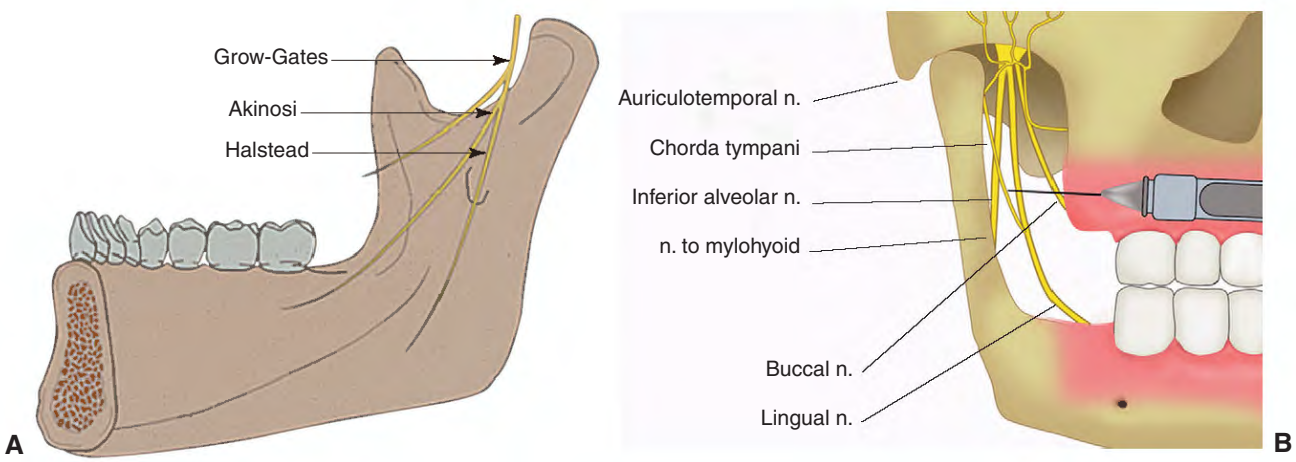


Figure 31-2 **A,** The Akinosi block procedure for local anesthesia is more effective than the traditional dental injection in the edentulous patient. **B,** Akinosi technique: The patient's mouth is almost completely closed or at the approximate occlusal vertical dimension in a denture wearer. The cheek is retracted with the free hand to expose the posterior teeth. The syringe is aligned parallel to the occlusal plane of the maxillary molars, and the needle is positioned level with the mucogingival junction of the maxillary second and third molars. The needle is inserted into the buccal mucosa as close as possible to the medial surface of the mandibular ramus to a depth of 25 to 30 mm without contacting bone. After careful aspiration, the anesthetic solution is deposited, approximately halfway between the mandibular foramen and the neck of the condyle into the middle of the pterygomandibular space.

WWW.HIGHDENT.IR

Box 31-2 Akinosi Nerve Block Advantages

- Anesthesia
  - Inferior alveolar
  - Lingual
  - Buccal
  - Mylohyoid
- High success rate
- Low rate of positive aspiration
- Reduced incidence of trismus, paresthesia
- Reduced pain

anterior mandible are also required, because the inferior third of the mandible is innervated by cervical nerves (C2-C3). Typically, the injections are in the facial sites in front of the mental foramina and the midline, whereas the lingual injections are lingual to each canine region.

An extraoral and intraoral scrubbing and draping of the patient are then performed. The benefit of aseptic technique is well documented in general.[7] However, a controversy exists regarding "sterile" versus clean surgical technique for endosteal implant placement.[8,9] Both the clean and sterile techniques use instruments, implant components, and hand pieces that are sterile. An actual sterile environment cannot be achieved within the oral cavity[10,11]; therefore the term *aseptic* is often used.

A primary issue is the need to use sterilized gloves, gowns, patient drapes and covers for the light, hand piece cord, and evacuation tips. In addition, can the surgeon and assistant touch nonsterile items in the operating room? Scharf and Tarnow compared sterile versus clean conditions with 386 implants in 92 patients.[12] Success rates were within 0.5% for the mandible and 1.6% for the maxilla. Kraut placed 2620 implants with a clean technique with a 1.26% surgical failure at implant uncovery and 0.5% early implant failure after function.[9] Therefore these reports suggest the clean technique is adequate for implant surgery.

Brånemark Surgical Approach

The original Brånemark surgical approach was used in a hospital environment, with an aseptic protocol similar to orthopedic implants. There are several advantages to this approach. Normal skin flora, including *Staphylococcus epidermidis* and *Staphylococcus aureus,* are able to adhere to implanted biomaterials.[13-15] A bacterial smear layer may develop, which will be difficult for the body to remove by phagocytosis or bone cell activity. Antimicrobial mouth rinses (e.g., chlorhexidine) significantly reduce bacterial count in the saliva for more than 4 hours.[16] Chlorhexidine has been shown to reduce microbial complications when used perioperatively.[17,18] Povidone-iodine alcohol solutions have antibacterial properties used in preoperative site disinfection.[19] Although a sterile technique is not possible within the mouth, scrubbing around and within the mouth with these agents reduces the risk of a bacterial smear layer by inadvertent contamination by direct contact with the implant or indirectly via a glove or instrument.

The clinical decision for an aseptic versus clean environment rests with the individual clinician, because documentation exists for either approach. However, it is prudent to reduce the risk of infection preoperatively and postoperatively. Although rare, infection may occur as a postoperative complication. Intraoral surgery is assimilated to a class II or clean contaminated surgery. Incidence of infection for class II surgery is 10% to 15%, but can be reduced to about 1% if appropriate technique and prophylactic antibiotics are prescribed.[20] The risk of infection with endosteal implants when preoperative and postoperative antibiotics are used appears to be even less.[9,21,22]

What Is Clean?

The question remains: What is "clean"? In one office, "clean" may result in the doctor looking for instruments in a drawer, adjusting a chair, touching the mask, moving the patient's hair out of the operative site, and so on. Therefore the issue appears to be not whether sterile gloves and gowns are used, but what protocols are in place to reduce the risk of contamination reaching the implant. It is prudent to train the staff and doctors in aseptic technique to reduce these risks. Training for an aseptic protocol relative to intraoral and extraoral scrubs and touching only sterile or scrubbed regions within the operatory are prudent to decrease risk.

More invasive procedures such as sinus bone grafts exhibit a range of postoperative infection from 3% to 20%.[23,24] Based on this, the author suggests that an aseptic surgical procedure be used for most sinus surgeries. For full-arch implants or bone grafts, larger regions of tissue are exposed with increased risks to patients if infection occurs, so aseptic technique should be considered. In many dental offices the staff needs constant retraining for procedures that are not often performed. The routine aseptic surgical approach ensures the quality and consistency of the asepsis when it is most indicated.

Intraoral and extraoral scrubbing with iodine, povidone-iodine (Betadine), or 0.12% chlorhexidine gluconate may be performed by a scrubbed assistant. A surgical scrub and gowning is performed by the doctor during the patient preparation. On return to the operating room, the patient's final drapes are positioned. The surgical gloves are rinsed and wiped with sterile saline to remove any powder or contaminant that may be inadvertently transmitted on the implant surface during the subsequent surgery. Infiltration with 2% lidocaine with 1:100,000 epinephrine into the labial and lingual mucoperiosteum evaluates the depth of anesthesia and provides some initial hemostasis. In

WWW.HIGHDENT.IR

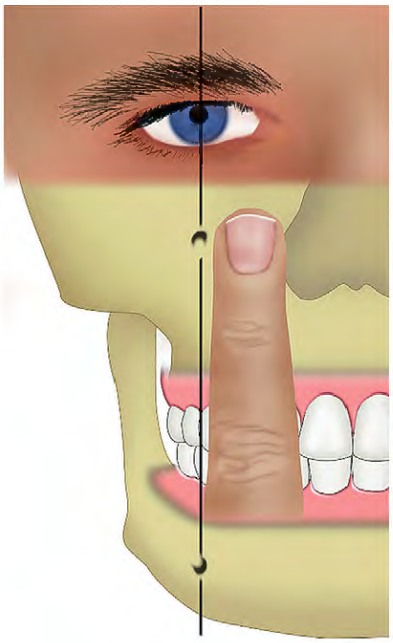


Figure 31-3 The position of the mental foramen may be approximated by drawing an imaginary vertical line from the pupil or using one finger width lateral to the nose through the infraorbital and mental foramina.

addition, when the local anesthetic is placed under the periosteum along the crest of the ridge, a hydroelevation of the soft tissue may help initial reflection of the soft tissue.

Incision

After anesthesia is achieved, the soft tissues and ridge height are evaluated. The lingual aspect of the mandible is palpated for undercuts and compared with the radiographic diagnostic images. Identification of the approximate position of the mental foramen facilitates the incision line design in moderately to severely atrophied mandibles and allows a more rapid reflection in regions around this landmark. The mandibular foramen region may be located by several methods. Palpation for the depressed foramen has been advocated, but is the most variable.[7] The overlying tissues usually prevent adequate feeling to identify the foramen with certainty.

Before the patient is surgically draped for the procedure, an imaginary line may be drawn between the pupils of the eyes. A perpendicular line may then be drawn through each pupil (Figure 31-3). This vertical line passes through the infraorbital foramen and mental foramen. A surgical pen is used to mark the gingival tissue over the mental foramen region. If the patient is draped and the eyes cannot be used as a landmark, the foramen region corresponds to a vertical line drawn one fingerwidth distal to the ala of the nose and slightly distal to the corner of the mouth.

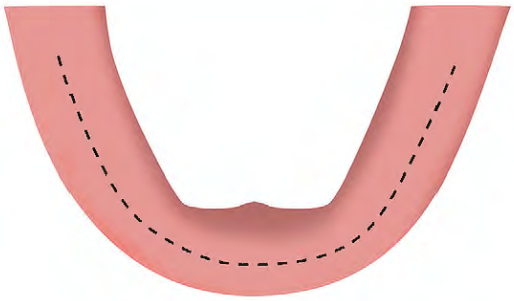


Figure 31-4 The initial incision bisects the keratinized gingiva on the crest of the ridge and extends from the first molar region to the contralateral first molar region. An accessory incision may be added off the midline to facilitate reflection and mental foramen identification.

Because suture line opening is the most common immediate postoperative complication, the surgical incision is designed to minimize this problem. If the crest of the ridge is above the floor of the mouth and there is greater than 3 mm of attached, keratinized gingiva on the crest of the ridge, a full-thickness incision is made, bisecting the attached tissue and scoring the bone on the crest of the ridge from the first molar region on one side to the anterior region and to the contralateral first molar region (Figure 31-4). If less than 3 mm of attached gingiva exists on the ridge, the full-thickness incision is placed so that at least 1.5 mm of the attached tissue is to the facial aspect of the incision line. This zone of attached tissue on the facial flap provides greater resistance for the sutures against tension of the mentalis muscle in the anterior region and buccinator muscle in the molar-premolar regions, which often cause the incision line opening. In addition, the blood vessels from the lingual artery may not cross over the crest of the ridge, but instead stop within the attached tissue.[25] As a result, an incision made facial to the attached tissue may cause partial ischemia to some of the crestal tissue. In addition, the incision in unkeratinized facial tissue also severs larger blood vessels, which increases bleeding and decreases vision during surgery. The incision should be done in one pass through the mucosa and periosteum and precisely score the periosteum. Additional scoring of the crestal bone after the initial incision design is usually beneficial. Areas of soft tissue invagination in prior extraction sockets are excised so as to create a clean margin for the flap, devoid of epithelium.

SOFT TISSUE REFLECTION—LANDMARK IDENTIFICATION

A periosteal elevator (e.g., Molt 2-4, Molt A-1, Molt 7 or 9) is used to reflect a full-thickness flap. The mucoperiosteal flap is first reflected on the lingual aspect, which is typically less tenaciously attached to the underlying bone. The lingual reflection begins in the

WWW.HIGHDENT.IR

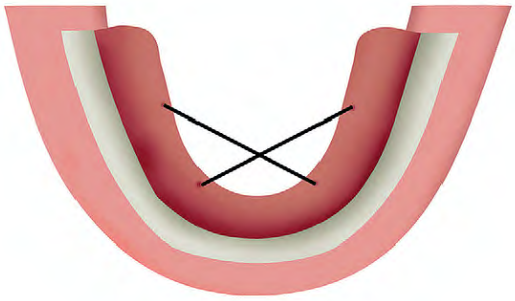


Figure 31-5 A periosteal elevator is used to first reflect the lingual full-thickness soft tissue flap. Two 2-0 sutures may then be used as retraction sutures, placed from each canine position to the contralateral molar site.

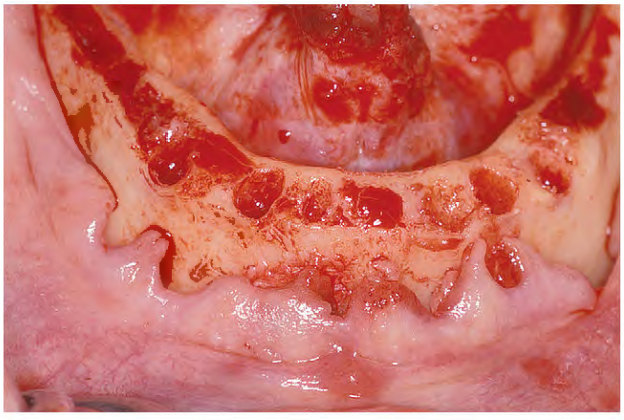


Figure 31-6 After the lingual flap is tied back with retraction sutures, the facial full-thickness tissue flap is reflected from the bilateral first molar region.

regions of the canine and proceeds to the molar region on the same side. The contralateral canine to molar region is then reflected. This phase of the procedure most often is performed within a few seconds. The lingual flap between the canines is then reflected. This region is more difficult to reflect, because some of the tissues are embedded into the residual alveolar crest. Retraction (tie-back) sutures with 2-0 silk are then used to keep the lingual flap out of the surgical site. The right lingual canine region is tied to the left molar area, and vice versa. The sutures engage at least 5 mm of tissue, with care taken to avoid the submandibular duct. The lingual flap retraction improves visibility, negates repeated surgical assistant retraction efforts during the procedure, and reduces trauma to the thin lingual periosteal tissues (Figure 31-5).

After a full-thickness periosteal flap is reflected, the periosteum must regenerate from the unreflected portion before this structure can participate in crestal bone healing.[26] Although adequate reflection to identify all bony landmarks is necessary, overzealous reflection increases soft tissue trauma and may delay bone callus formation around the implant, which may contribute to crestal bone healing.

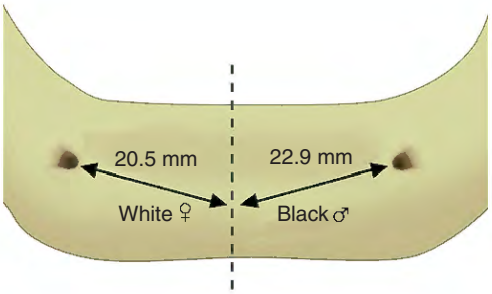


Figure 31-7 The mental foramen is approximately 22 mm from the midline; however, there is a slight variation, depending on race and sex.

The facial flap is then reflected. The exact location of the mental foramina and associated nerves, arteries, and veins are identified (Figure 31-6). These are essential surgical landmarks. An anthropometric analysis of the position of the mental foramina was performed by Cutright et al.[27] The average distance from the midline was 2.2 cm, which means the usual distance between the foramina is 44 mm. Because at least 7 mm of mesiodistal bone are usually required for each 4-mm-diameter implant, the average anterior mandible has room for six implants. However, the mental foramina position is variable, depending on race, sex, and skull size. The average interforamen distance for black males was 45.8 mm; white males, 45 mm; black females, 43.8 mm; and white females, 41 mm. Therefore white females often do not have room for six implants between the foramina (Figure 31-7).

When the mental foramen position is related to a tooth position, it may reside as far anterior as the canine to first premolar position, or as far distal as the second premolar to first molar root position.[28-31] The farther distal the foramen (and neurovascular canal), the farther distal the most distal implant (A and E sites) and the better the biomechanics for the prosthesis. When the foramen resides in its most distal position, six implants may be positioned between the foramina. When the foramen resides in its most anterior position, only four implants may be inserted to leave 3 mm of space between each implant. The most common mental foramina sites are between the first and second premolar roots, but they may be present below either the first or second premolar root. Under this condition, five implants may be positioned between the foramina.

Because the primary incision was extended to the first molar region, the approach to the foramina can proceed from both anterior and posterior, which facilitates their identification. After the periosteum is reflected off the residual crest, a moist surgical sponge can be used to wipe the periosteum off the dense labial cortical plate around each foramen. This permits rapid and safe exposure of the nerve complex without trauma from instruments. Soft tissue reflection is limited in the mandibular symphysis region to avoid ptosis of

WWW.HIGHDENT.IR

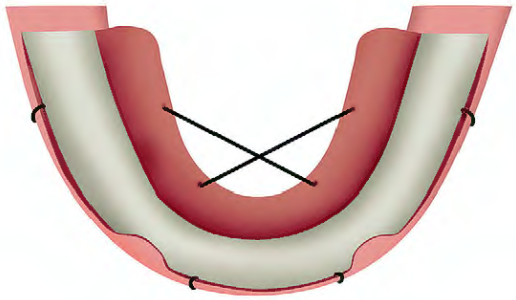


Figure 31-8 The facial full-thickness soft tissue flap is then reflected. The mental foramen contents are identified, along with the crest of the anterior ridge, both anterior and posterior to the position of the foramen. The tie-back sutures may be positioned both behind and in the front of the foramen on each side.

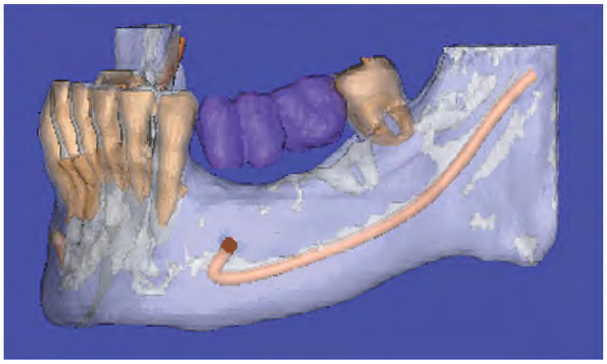


Figure 31-9 An anterior loop to the mandibular canal occurs when the canal proceeds inferior to the foramen, passes under the foramen for 1 to 3 mm, and then curves superior and distal to exit the foramen.

the chin, which may occur after overzealous reflection of the mentalis muscles' lower attachments in moderately to severely atrophied mandibles. Tie-back sutures may be positioned to retract the facial flap during surgery. Most often these are placed in front of the mental foramen (Figure 31-8). All fibrous tissue is removed from the crest of the edentulous ridge. A bone rongeur, followed by a moist surgical sponge rubbed across the crestal surface, eliminates most fibrous adhesions.

If the mandibular canal approaches the foramen at the same height, the nerve always enters the distal portion of the foramen. When this is clearly identified on the panoramic radiograph, the foramen is used as the distal landmark. The panoramic radiographs or CT images are observed to visualize a possible anterior loop of the mandibular neurovascular canal. A mandibular loop occurs when the mandibular neurovascular canal approaches the foramen from below, proceeds anterior to the foramen about 1 to 3 mm, and curves upward and distal to enter the medial aspect of the mental foramen (Figure 31-9). This has been observed almost 12% of the time for a distance of 1 to 3 mm on panoramic radiographs.[32] On the other hand, Rosenquist dissected 58 inferior alveolar neurovascular bundles and identified a 1-mm anterior loop

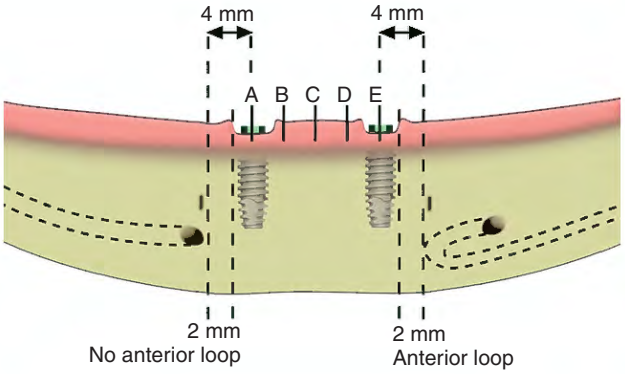


Figure 31-10 The position of the distal implant between the foramen is affected by the presence of an anterior loop. The goal is to have the implant 2 mm in front of the mandibular nerve. When an anterior loop is present, the distal implant is placed farther mesial.

in 43 cases.[33] A study on dry skulls noted anterior loops of 2 mm or more on 92% to 96% of the specimens.[34] Surgical dissection provided by Solar et al.[35] showed an anterior loop in 60% of mandibles ranging from 0.5 to 5 mm. Neiva et al. identified loops in 88% of cases with a range of 1 to 11 mm.[36] Dissection studies with comparison to radiographs were performed by Bavitz et al.,[37] who reported 54% of loops in periapical radiographs, but only 11% where verified by dissection. Other studies concurred with the conclusion that a high percentage of false-positive or false-negative findings may occur.[38,39] For such a topography, the most distal anterior implants are placed 2 mm anterior to the anterior loop, which becomes the landmark rather than the mental foramen itself (Figure 31-10).

It should be recognized that the inferior alveolar nerve complex is in the facial aspect of the bone as it exits the foramen. Therefore an implant placed in this region is most often lingual to the nerve, because the crest of the ridge is more medial than the foramen. As a result, even when a panoramic image may indicate that the implant is in the position of an anterior loop of the mandibular nerve, it most likely resides lingual to the nerve, with no paresthesia evident. Only CT scan imaging properly indicates the spatial relationship between these elements.

On occasion, the nerve complex around the foramen is not clearly identified on traditional films. An anterior loop may or may not be present. A CT scan of the region may provide the information needed. Another option is to determine whether there is an anterior loop during surgery. A curved Naber's 2N probe may be gently inserted inside and along the bone on the distal half of the foramen to determine whether the nerve proceeds from the posterior region directly into the foramen. If no nerve entry is felt along the distal half of the foramen, the nerve must loop anteriorly and enter from the anterior aspect (Figure 31-11). Probing the anterior half of the foramen is not diagnostic, because the mental

WWW.HIGHDENT.IR

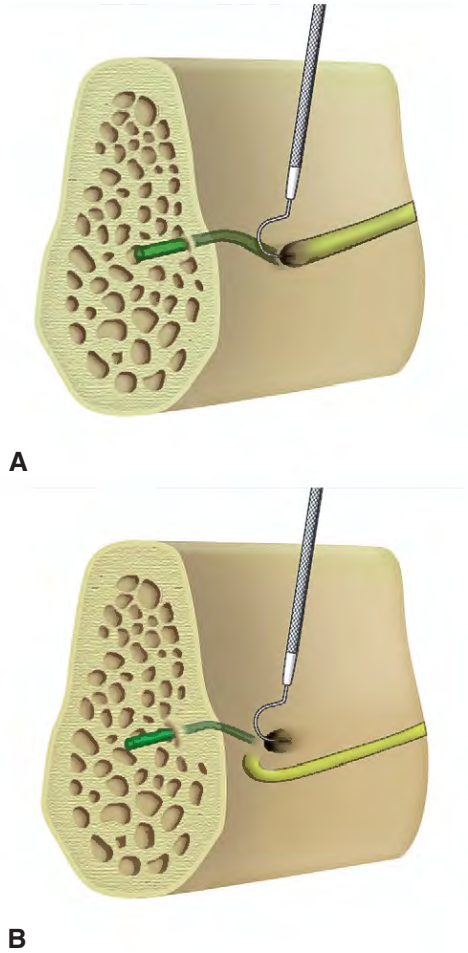


Figure 31-11 **A,** Naber's 2N probe may be used to determine the presence of an anterior loop to the mandibular canal. The probe is gently inserted along bone on the distal one-half of the foramen. **B,** If no nerve canal entry is palpated, the nerve must enter the foramen from the anterior aspect and an anterior loop is present.

nerve complex continues anteriorly from the foramen to innervate anterior teeth and bone, regardless of whether there is an anterior loop. Therefore there is always a canal entering the anterior portion of the foramen.

The incisive branch of the inferior alveolar nerve continues within the bone from the position of the foramen to the midline, where it may have some crossover from the contralateral side. This nerve complex innervates the mandibular anterior teeth, but does not provide feeling to the chin or lower lip. Therefore it does not cause paresthesia, even when violated with a drill or implant.

A score mark is made with a rotary instrument facial to the crest of the ridge to delineate the most anterior position of the mandibular nerve over each mandibular foramen. This score mark may correspond to the foramen or the anterior loop of the canal, whichever is more anterior. These marks position the boundaries of the posterior surgical implant placement. The most distal portion of the implant is ideally placed 2 mm anterior to this border to allow for surgical error.

The bone width at the crest is then evaluated. If it is greater than 6 mm, the crown height of the prosthesis is less than the available bone height (crown/implant ratio <1), and the direction of load to the implant body is within 30 degrees, a Division A bone volume is present. The prosthesis type is considered. An RP-4 or RP-5 prosthesis requires a minimum of 12 mm of crown height space from the occlusal plane to the crest of bone to fabricate the prosthesis and superstructure bar. If the crown height space is insufficient, an osteoplasty is indicated. An FP-1 restoration requires between 9 and 12 mm crown height space from the occlusal plane to the bony crest. If the crown-height space is greater, and the patient demands an FP-1 prosthesis, an augmentation procedure may be required before surgical placement of implants.

The available bone in the anterior mandible is often Division B, with similar crown-to-bone height relationships as Division A, but the residual crest bone is 2.5 to 6 mm in width, with the ridge usually more lingual. The surgical options are osteoplasty, augmentation, or small-diameter implants, depending on the prosthesis type and the force factors of the patient.

The first premolar region, usually just anterior to the mental foramen (A and E positions), is often narrower than the other anatomical sites in the mandible. As such, special attention relative to width is noted in this region. A smaller-diameter implant may be necessary in this site. As a result, a distal cantilever may need to be reduced compared with when a regular or larger-diameter implant is used. This is also the area in which the anterior loop of the mandibular canal may be present. To its benefit, the bone angulation in this area is more vertical in relationship to the occlusal plane.

An osteoplasty of the crest of the ridge is often indicated, especially in Division B bone. The osteoplasty eliminates any fibrous tissue on the crest and ensures adequate width for implant placement. A bone caliper is used to measure the amount of bone to be removed in height. This caliper may also ensure there is adequate height to place implants after the osteoplasty in Division B bone. The osteoplasty may be accomplished with a bone rongeur or a high-torque hand piece with a surgical 703 bur or a crestal reduction bur under copious cooled saline irrigation, until greater than 6-mm width of bone is obtained. If several millimeters of bone must be removed to obtain the necessary width, additional resorption is probable during healing. Countersinking the crest module of the implant slightly below the remaining bone crest is considered in these instances to account for this resorption and to prevent premature loading of the implant through the soft tissue–borne interim prosthesis during healing.

The prosthesis must replace the vertical bone height removed during the osteoplasty. If a fixed prosthesis is the final prosthesis, care is taken to carefully evaluate the amount of bone reduced. Whenever an osteoplasty

WWW.HIGHDENT.IR

is performed, the bone is harvested and stored in sterile saline in case an autogenous graft is required during a later step of the surgical procedure.

When an osteoplasty is performed, the complete anterior section of the mandibular crest is reduced, not just the implant locations. The soft tissue will not follow abrupt contour changes on the crest. Instead, the gingival tissues form a thicker tissue in these regions. If the osteoplasty is done irregularly, when the abutment is placed, a greater sulcus depth occurs in the thicker tissue regions with the related complications of the increased risk of anaerobic bacteria. In addition, if a cantilever is planned for the final restoration, the osteoplasty should extend beyond the anterior ridge, under the cantilever length. Otherwise, the superstructure will need to be raised above the higher bone contour in the posterior region, which may compromise artificial tooth or attachment positions over the bar.

Implant Site Preparation

Rotary instruments are used to prepare the implant site. Regardless of implant design or manufacturer, several surgical concepts are crucial for initial rigid fixation. A primary criterion is to limit thermal or mechanical trauma to the surrounding hard tissues (Box 31-3).[40-55] Bone is very susceptible to heat.[40] Heat is generated by the drill during the implant osteotomy. A major method to reduce the bone temperature during drilling is the use of cooled irrigation.[41,42] A secondary method to reduce heat and thermal trauma is related to the amount of bone removed with each drill.[41,45] Therefore sequencing drill diameters should prepare the final osteotomy site. The drill speed and time during which the bone is prepared is one of the major factors related to bone trauma.[48,49] Additional factors such as internal versus externally cooled drills are less important.[48,55] A detailed discussion of these factors is presented in the bone density—a surgical approach (see Chapter 29).

Implant Site Location

Most often, five implant sites are planned between the mental foramina for mandibular overdentures or full-arch fixed prosthesis, and are labeled A, B, C, D, and E, because they do not always related to tooth positions. Regardless of the number of implants (i.e., 2 to 8) being placed at the initial surgery, all five sites are determined. This permits insertion of additional implants in the future (if needed) to upgrade a prosthesis type or to solve complications. A No. 6 round bur or sharp pilot drill marks the initial implant site location in the midline (C position), unless an FP-1 prosthesis is planned. If the intended prosthesis is FP-1, the center implant is placed off the midline toward the most distal foramen by 2.5 to 3 mm.

Box 31-3 Factors Critical to Minimal Heat During Osteotomy Preparation

- Cooling fluid
- Bone quality
- Minimum volume of bone removed by each drill (incremental drill diameter)
- Drill: sharpness, design
- Frequency/time of contact
- Depth
- Pressure
- Speed

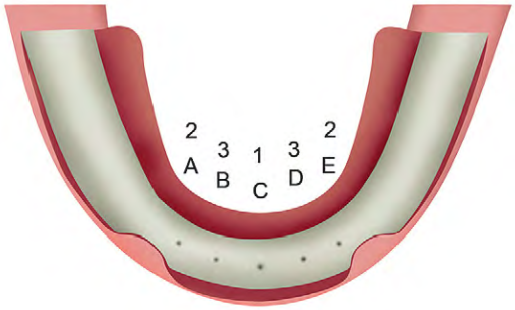


Figure 31-12 After the anterior mandible is reflected and the crest prepared for implants, five implant sites are planned. The first site (C) is usually in the midline, the sites A and E are positioned 4 mm in from of the foramen, then the B and D positions are selected. Each site should be at least 7 mm apart from center to center.

After the midline implant site (C) is determined, the boundary score lines on the crest of the ridge that correspond to the anterior limit of the mandibular canal are identified. A pilot hole is made with a No. 6 round bur or pilot drill placed 4 mm medial to each distal score line (if the implants are 4 mm in diameter) to determine the center of the most distal A and E implants. This provides 2 mm of surgical error away from the boundary that identifies the mandibular nerve during bone preparation and implant placement. The distance between the midline pilot hole and distal pilot holes are then equally divided for the B and D implants. They also can be mapped with a measuring tool. The distance between each implant center site should be at least 7 mm, or 3 mm between the 4-mm-diameter implant bodies. Larger-diameter implants require more than 7 mm between each center pilot hole (i.e., 8 mm for 5-mm-diameter implants) (Figure 31-12).

The initial pilot drill is used at a depth of 5 mm and evaluates the density and thickness of cortical bone on the crest of the ridge, if any remains after the osteoplasty. It can also be used for initial bone preparation in the narrow ridge. This end-cutting bur can prevent lateral perforations because it will roll away under light pressure from the harder inner cortical plate and stay within the softer trabecular bone. A No. 6 bur

WWW.HIGHDENT.IR

or a Lindemann drill may also stretch a hole in any direction to improve its final position. For example, if the initial bur hole is too lingual, the #6 or Lindemann bur can be used in the initial site to create an oblong hole more facial. As long as the initial hole is less than 2 mm from the ideal site, the final osteotomy and implant will completely obtund the initial erroneous pilot hole. In this way, the ideal location is finalized before the initial osteotomy is begun.

The surgical guide template is placed in position, and the faciolingual location and ideal angulation for the implants are determined. Ideal angulation is perpendicular to the plane of occlusion and corresponds to the cingulum of the teeth for a screw-retained bar or prosthesis, or to the incisal edge for cement-retained fixed prostheses. The template is then removed so that the facial and lingual contours of bone may be observed during site preparation.

The center implant site (C) is prepared first with the 2.0-mm-diameter pilot drill for the initial depth of bone preparation of 9 mm (Figure 31-13). This position should be perpendicular to the occlusal plane. If one marginal crest of bone is higher than the other (usually the lingual), the osteotomy depth is measured from the most inferior edge such that the implant body is positioned slightly below the lingual crest of the ridge.

A direction indicator (parallel pin) is placed into the center implant site (Figure 31-14). Angulation is checked in a labiolingual and mesiodistal direction with the surgical template, which may be facilitated with guidance from an assistant (preferably standing over the midline at the foot of the chair) (Figure 31-15). The osteotomy is often not prepared while the template is in place. The implant site must have adequate bone on the labial and lingual aspects. This requires direct vision and careful observation of both cortical plates while preparing the bone. A pumping motion is used to prepare the site to the radiographically determined depth; however, this dimension is reevaluated and may be changed at this time. The bone density is noted during the initial osteotomy preparation. The procedures described in this chapter are for D2 bone density.

Threaded root form implants are provided in several preestablished heights, dependent on the specific system. The ideal length of a threaded implant should be between 12 and 15 mm long in D2 bone. Because most stresses occur at the crest in favorable bone density, after an optimum implant length is obtained for each bone density, rigidly fixated implants do not appear to gain further clinical advantage when adding only length. In natural teeth, the periodontal ligament allows stress dissipation away from the crest, yet their length does not increase from the front to the back of the mouth (even though greater forces are generated in the posterior regions). Similarly, the need for longer implants or implants that engage the opposing cortical plate with an implant in a two-stage healing process is not supported in the literature.

After the initial sites are prepared with the pilot drill, small-diameter (2 mm) direction indicators are used in the initial implant preparations to evaluate the need for any minor adjustments (Figure 31-16). The surgical template is inserted to evaluate implant

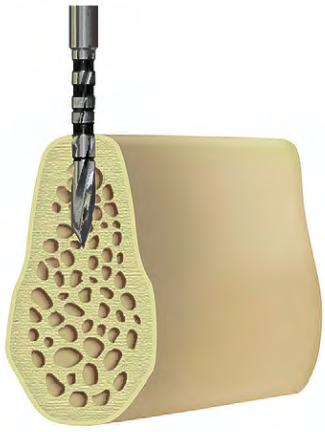


Figure 31-13 A 2-mm-diameter pilot drill is used to prepare the center implant site for 9 mm, perpendicular to the occlusal plane.

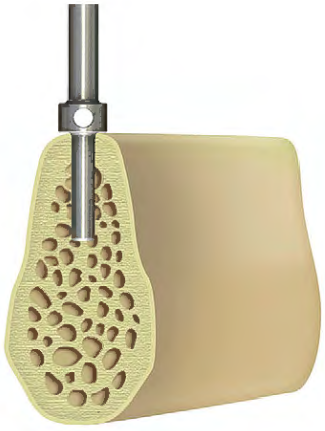


Figure 31-14 A direction indicator (parallel pin) is inserted into the center implant site.

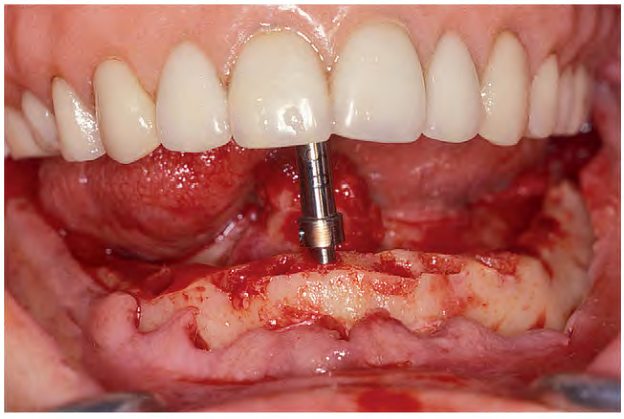


Figure 31-15 The direction indicator is evaluated for angulation and proper position.

WWW.HIGHDENT.IR

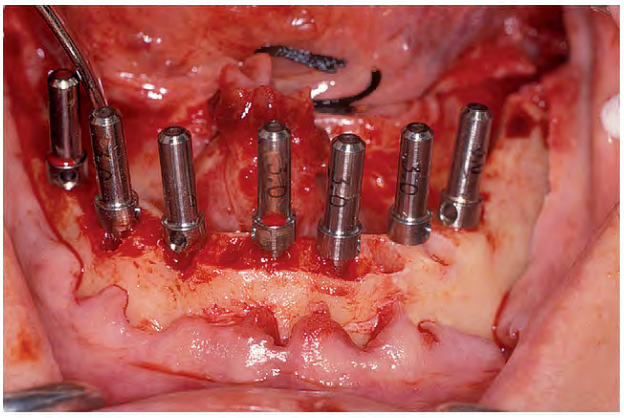


Figure 31-16 The pilot drill is used to prepare the initial sites, starting with the center, then A and E, then B and D, using the center parallel pin as a guide.

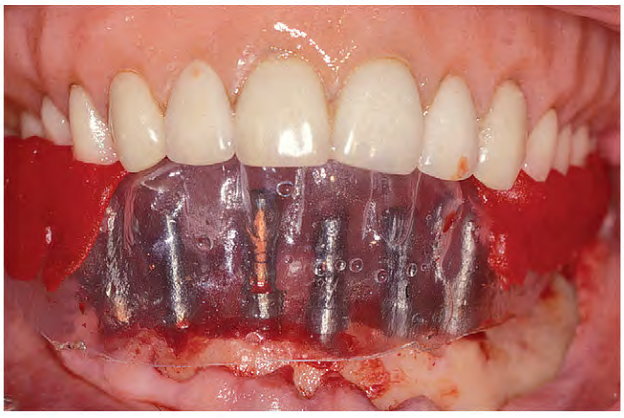


Figure 31-17 Small-diameter direction indicators are placed in the initial sites. A surgical template is inserted with the parallel guide pins in place to evaluate the future implant angulation and position.

angulation and position (Figure 31-17). If the angulation is incorrect by less than 20 degrees, it can usually be corrected with the next drill in the sequence provided by the manufacturer. If the angulation is to be modified by more than 20 degrees, a side-cutting drill (e.g., Lindemann) rather than end cutting is most efficient. These side-cutting drills also can stretch the initial osteotomy laterally to improve the implant position. If the angulation is beyond 30 degrees to an axis perpendicular to the occlusal plane, or if the osteotomy is too lingual or facial in relation to the incisal edge, correction of the initial osteotomy should precede any further bone preparation. The oblong osteotomy prepared to correct the problem of poor position or angulation is obliterated and made round again with successive drills of incremental diameter.

After the direction indicator confirms proper implant position, the osteotomy is made to the full depth of the planed implant size (Figure 31-18). It is usually better to drill 1 mm deeper than needed (i.e., 13 mm for a 12-mm implant) when the opposing landmark permits. If the opposing landmark is perforated before obtaining the desired length of the osteotomy, the osteotomy length before perforation is noted, and a shorter-length implant is selected. Ideally, the implant should not perforate the mandible to avoid irritation of the soft tissue, especially for a sharper apex implant design, where even slight perforation may irritate the tissues. If the implant has an open basket design, a perforation may trigger fibrous tissue ingrowth within the basket, rather than bone.

On occasion, bleeding from the osteotomy site may be copious, especially in the midline site, even though no bone perforation has occurred. An artery enters the lingual plate below the genial tubercles in the midline, and an anterior branch of the mandibular neurovascular canal continues beyond the foramen. The surgeon should confirm that no perforation through the lingual plate or inferior border has occurred. The majority of

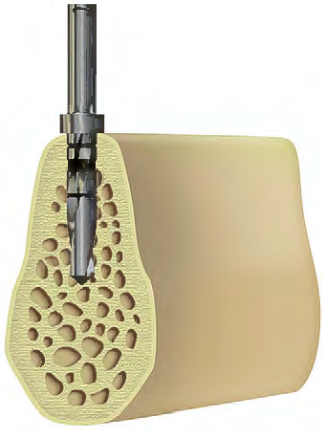


Figure 31-18 After the position and angulation is acceptable, the osteotomy sites may be made to the full depth of the desired implant size.

time, this bleeding is of no surgical consequence. It is also not associated with any soft tissue paresthesia after surgery. If the bleeding is causing concern for vision or total blood loss, a collagen sponge may be placed into the site, along with a drill or direction indicator to help obtund the site. Elevation of the head and pulling the tongue out to reduce flood flow to the lingual artery is also of benefit until the platelet plug forms and reduces the bleeding. If the osteotomy is completed, an implant may also be introduced into the site and will crush the walls of bone and halt the bleeding process.

The anterior mandible may present a significant undercut on the lingual aspect between the foramina. Life-threatening hemorrhage has been reported when a drill perforates the lingual plate of the sublingual region of the mandible and injures a sublingual or submental artery, especially in the canine region.[56-59] If perforation of the lingual cortical plate is associated with arterial bleeding, it is critical to identify its origin (Table 31-1). Bleeding in the floor of the anterior region of the mouth can originate from the lingual artery, facial

WWW.HIGHDENT.IR

Table 31-1 Treatment of Hemorrhage at Implant Osteotomy Site

BLEEDING SITE DURING IMPLANT OSTEOTOMY	ARTERIES	TREATMENT
Posterior mandible	Mylohyoid	Finger pressure at the site
Middle lingual of mandible	Submental	Surgical ligation of facial and lingual arteries
Anterior lingual of mandible	Terminal branch of sublingual or submental	Compression, vasoconstriction, cautery, or ligation
Invading the mandibular canal	Inferior alveolar artery	Bone graft

artery, or one of its branches. The submental artery originates from the facial artery, runs along the inferior border of the mandible, and meets at the midline. Pressure over the genial angle notch (just anterior to the masseter muscle) stops this type of bleeding. If the injured artery stems from the lingual artery, the bleeding will be reduced when the tongue is pulled out, thus placing pressure against the lingual artery where it crosses over the hyoid bone. Elevating the head is also of benefit. After the bleeding is reduced, it may be stopped by collagen sponges or hemostasis after clot formation, or rarely by surgical ties or electrocautery (see Complications).

A critical step in obtaining rigid osseous fixation after initial implant placement is to maintain a rigid bone-implant interface without micromovement between the time of original placement and the second stage of surgery. The implant does not have to be submerged below the bone to achieve this result. The implant does not even need to be below the soft tissue to obtain this type of interface.[60,61] However, the clinician should recognize situations such that the higher the implant is placed above the bone, the greater the risk factors for trauma that may result in movement at the interface during initial healing. As a result, when an overlying soft tissue–borne prosthesis is worn, it may be advantageous to countersink the implant below the bone. On the other hand, when a prosthesis abutment is added to the implant crest module below the bone, approximately 0.5 mm of soft tissue forms below the abutment-to-implant connection.[62] Therefore, if the crest module of the implant was below the crestal bone at the time of surgical placement, crestal bone loss will occur when the abutment is added.

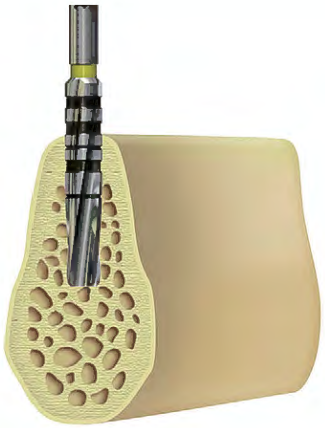
The implant surgeon should select the crest module position of the implant in relationship to the crestal bone on an individual basis. If the cortical bone and the overlying tissue is thick, and the patient has no parafunctional habits to apply stresses to the implant during healing, the implant and healing cover height are less critical and the platform of the crest module is often placed level with the crestal bone in more dense bone. Most often, the implant crestal module–abutment connection region should be placed 0 to 0.5 mm below the bone level in D2 bone, rather than countersinking the implant several millimeters below crestal bone. As a result, for most external hex systems, the hex connection and healing cover remain slightly above the bone. If the overlying tissue is thin, the superior cortical plate was removed during an osteoplasty, or the remaining bone is of softer quality such as fine trabecular (D4), the implant and healing cover should be placed below the bone crest. As a result, the osteotomy must be 1 mm deeper before implant insertion to permit the crest module to be below the bone. As a result, the final bone osteotomy depth is variable by ±1 mm for each patient condition.

After the center site is initially prepared, the most distal implant sites are prepared next, using the direction indicator in the center osteotomy for parallelism in all planes. Direction indicators may then also be placed in the distal sites, and the implant surgeon proceeds to the intermediary implant site locations in a similar fashion. Direction indicators may also be used to tamponade profuse bleeding from an implant bone drilling site until the implant is placed. This technique is useful in maintaining adequate vision and decreasing surgical blood loss.

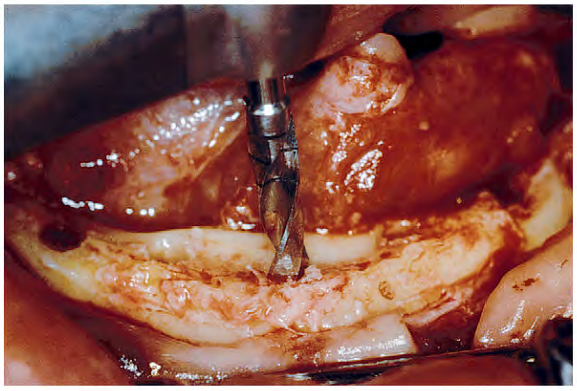
If a bone drill becomes locked in the bone during preparation, the hand piece should not be wiggled back and forth to disengage the drill. This may increase the size of the bone preparation, cause injury and necrosis to the bone, or separate the drill above or below the bone. Instead, the rotation of the drill in the hand piece is reversed, or the drill is disengaged from the engine and rotated counterclockwise with a forceps.

Drills of intermediate diameter are used next for the implant bone preparation. Gradual increases in drill diameter reduce the amount of pressure and heat transmitted to the bone, especially in presence of dense and thick cortical bone. Lavelle has shown the heat generated from a drill can be transmitted more than 3 mm away from the osteotomy site and reach temperatures higher than 50° C under copious irrigation.[50] The 2.0-mm initial drill is followed by a 2.5-mm drill, and then a 3.0- or 3.4-mm drill (Figure 31-19). Some manufacturers do not use an intermediate drill; however, a decrease in the heat and trauma generated is found with the intermediate drill.[53] The softer the bone, the fewer the number of intermediate drills

WWW.HIGHDENT.IR



A



B

Figure 31-19 A sequence of osteotomy drill preparation is dependent on implant diameter and design, but often uses a 2.0 mm initial drill, followed by 2.5 mm, 3.0 mm and 3.4 mm as a final dimension.

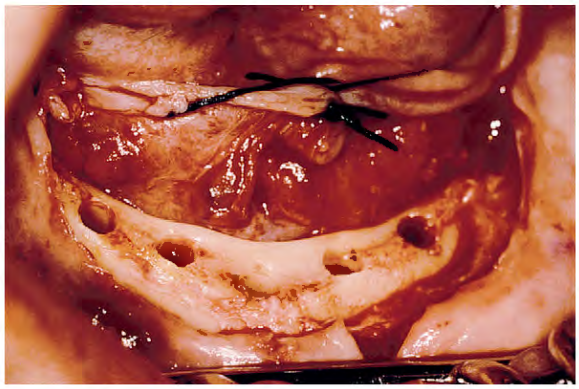


Figure 31-20 The final drill diameter is the most critical for the osteotomy, because the bone next to this drill will be in direct initial contact with the implants.

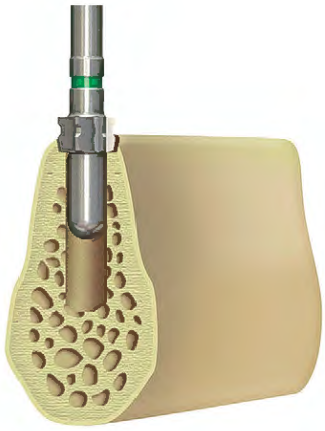


Figure 31-21 A crest module drill is often used in the anterior mandible, because cortical bone is present on the crest.

needed. Direction indicators are placed in bone sites medial or lateral to implant sites being prepared. Minor corrections in angulation should be accomplished during these intermediate drilling steps.

The final drill diameter is used to prepare the implant receptor site under copious irrigation (Figure 31-20). This step is the most critical for initial implant insertion and healing. The bone surrounding this drill will be in direct contact with the implant. When the final drill preparation is not precise, the implant-bone region may be incongruent and gaps may decrease initial stability, which may lead to early failure. A lesser initial contact with the host bone also decreases the percentage of new bone-implant contact formation.[54] Therefore a constant pressure and direction is used with the final drill to ensure a precise, round osteotomy is prepared along with direction indicators in adjacent sites to maintain the proper angulation. A summary of potential problems during implant osteotomy preparation and suggested solutions is presented in Table 31-2.

The crestal collar region of any system, regardless of dimension, should be placed in contact with crestal cortical bone. Many implants present a wider crest module design than the implant body. When the implant is placed below the bone, these systems may need an additional step in the bone preparation, with a crestal countersink drill to increase the osteotomy diameter only in this region (Figure 31-21). If there is no marginal cortical bone, and especially if the trabecular bone is soft, the implant is inserted without a crest countersink drill procedure. This compresses the marginal rim of bone and adds rigidity to the implant during initial healing.[63]

If a threaded implant system has been selected to support the prosthesis, the clinician should decide whether or not to tap the osteotomy before implant insertion. Very dense bone (D1) requires a bone tap for the entire implant height. D2 bone may also require a bone tap, especially if the implant is made of a softer grade of titanium. With thick inferior cortical plates at the apex of the osteotomy, a bone tap is also required for the entire height. If only the superior cortical plate is dense and provides initial rigidity of the implant, the bone tap is used only in this section. Studies concentrating on threaded implants inserted with and without a bone tap have shown that implants with precut threads in dense bone achieved a higher

WWW.HIGHDENT.IR

Table 31-2 Possible Problems Encountered during Root Form Surgery and Suggested Solutions

PROBLEMS	SOLUTIONS
Improper pilot hole/location	Create a new pilot hole with round bur No. 6 or stretch the existing hole with a No. 6 drill or a side-cutting drill (e.g., Lindemann).
Improper drilling angulation	Start a new direction with the pilot drill and 1.5-mm drill and continue with remaining procedures. Use a side-cutting drill to redirect the osteotomy. Use the assistant/doctor force direction indicators or template to regularly check direction during the osteotomy preparation.
Elliptical/eccentric preparation	Continue the surgical procedures and try to use the planned implant. If the implant is loose on seating, use a wider-diameter implant if possible. If not, pack the osteotomy with autogenous graft, compress it, and place implant again. If the angle with the adjacent implant is greater than 30 degrees, remove the implant and allow the surgical site to heal for approximately 6 months. Place the implant at a later date.
D1 bone density	Prepare osteotomy with a constant pumping motion to avoid overheating and use higher torque and speed. Use new drills and profuse irrigation with incremental drill sequence. Use bone tap with hand ratchet.
D3-D4 bone	Push the drill as it rotates at decreased speed. Do not bone tap. Insert implant with hand piece.
Perforated buccal or lingual bone plates	Stop drilling as soon as less than 1.0 mm of bone is lateral to the drill. Use an osteotome with a tapered end to expand the osteotomy, rather than removing the bone with a drill.
Threads exposed at crest of ridge	If implant threads are exposed coronally, graft autogenous bone harvested intraorally with rongeurs or a trephine bur over the region.

Figure 31-22 A bone tap is used at less than 30 rpm with a high torque hand piece or hand ratchet and copious irrigation in D1 or D2 bone before implant insertion. The bone tap may be used selectively only in the crestal region, when the underlying trabecular bone is soft and the implant does not engage cortical bone at the apical region of the implant.

percentage of bone-implant contact during the healing phase,[64] and greater bone trauma was identified with the self-tapping implant. In the cases of a fine trabecular bone, especially in the absence of crestal cortical plate, a bone tap is contraindicated because this decreases the initial stability of the implant.[63]

Whenever the bone tap is required, it should be used to prepare the bone at very slow speed (less than 30 rpm) with copious amounts of irrigant. This may be accomplished with a hand ratchet or a slow-speed, high-torque, contra-angle hand piece (40:1 to 100:1 reduction) (Figure 31-22). It is imperative that the angulation and light vertical pressure used on the ratchet or hand piece follow the same path as the final drill preparation for the entire depth. Otherwise, the tap may inadvertently stray in the trabecular bone and compromise the final implant placement. The direction indicators in adjacent sites are very beneficial in helping to maintain the correct angulation. A hand ratchet rather than a high-torque hand piece should be used in D1 bone to prevent hand piece breakage or wear.

The hand position of the surgeon is important in maintaining constant force and direction on the hand ratchet or hand piece during the threading process. When using a ratchet, one hand holds the ratchet while the thumb of the other hand is placed directly over the tap, the index finger retracts the lip for improved access and vision, and the middle finger is placed under the mandible in the direct path of the osteotomy. Care must be taken to let the handle of the ratchet rotate around the insertion pin, not pull or lever the pin and connected tap as it rotates. Using a hand piece to tap the implant site in D2 bone or the apical end in a D3 site presents some advantages, such as a relative ease of constant direction, especially in softer bone. The bone tap should be cleaned with saline before each new site is prepared.

WWW.HIGHDENT.IR

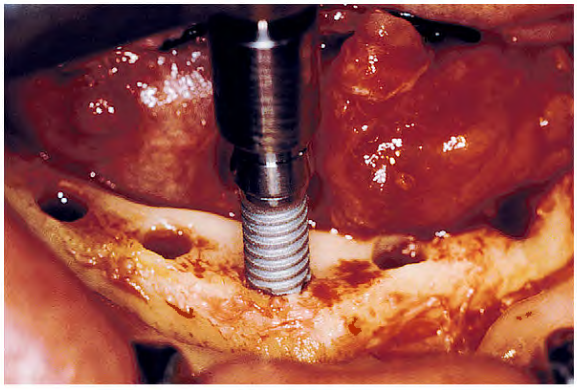


Figure 31-23 The high-torque hand piece is used to thread the implant into the anterior mandible at less than 30 rpm.

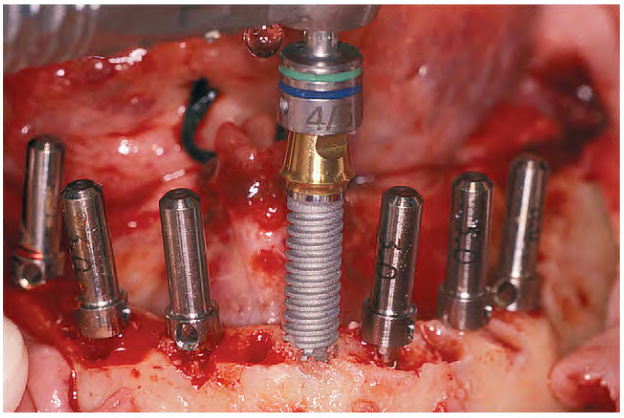


Figure 31-24 The hand piece insertion of the implant occurs after the osteotomy has been suctioned, to remove any debris or fluid, which may create hydrostatic pressure or friction during the insertion process.

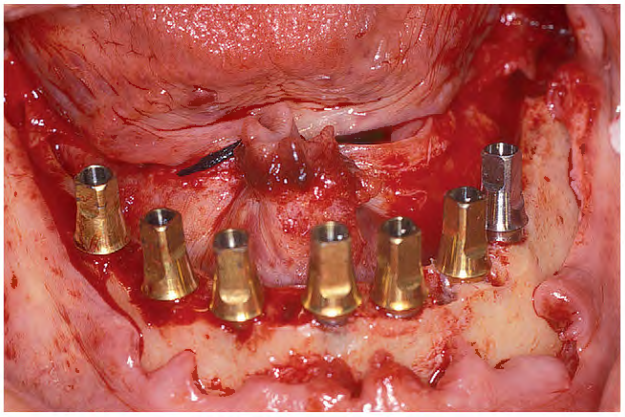


Figure 31-25 Five anterior implants have been threaded into position between the mental foramina, and two implants are inserted over the top of the foramen.

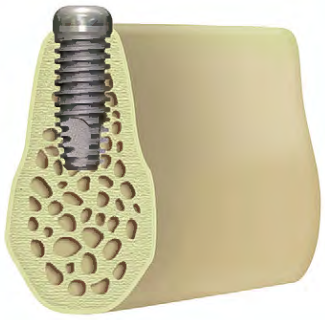


Figure 31-26 The first-stage cover screw is inserted into the implant body. This prevents soft or hard tissue growth over the implant platform during healing and protects the antirotational hex for abutment connection.

Implant Placement

The implant sites are flushed with saline to remove any debris and suctioned to reduce hydrostatic pressure during implant insertion. The implants are now ready to be threaded into position at a speed similar to the bone-tapping procedure (Figures 31-23, 31-24). The direction-indicator pins may still be needed in adjacent sites to guide the implants in the correct angulation in soft trabecular bone. The implant surface has been prepared by the manufacturer to be clean and uncontaminated. Handling the implant body is poor protocol and may alter the surface chemistry. Instead, the implant is placed directly into the implant site by means of a preattached insertion mount. Some manufacturers require the surgical team to attach the implant mounts before placement, but this procedure complicates the process with no benefit.

After the first threads of the implant body engage the bone, the same speed used to tap the bone is used to insert the implant. The surgeon's hand position is identical to that maintained during the bone tapping procedure. Sterile saline may be used after the implant has begun its descent into the implant osteotomy, but is not suggested. The patient may attempt to swallow as a result of the irrigant and during this process may close and change the angulation of the implant. The hand ratchet is used at the end of implant insertion to check the final stability of the implant body. The surgeon should not overtighten the implant in place. Excessive tightening of the implant in its final position may cause microfractures and compromise the entire implant thread-to-bone profile and interface development. When in doubt, the implant should be slightly unthreaded so as to decrease pressure at the developing interface. The marginal cortical bone should seal the implant periphery without voids or cracks. If any defects are present, the bone chips harvested from the drills or from an osteoplasty are used to fill any deficit (Figures 31-25, 31-26).

On rare occasions, the implant may not be rigid after implant placement. A mobile implant may not heal predictably with a direct bone interface. The implant may be fixated by inserting it deeper in the osteotomy or by the placement of a larger-diameter implant. In addition, the lateral cortical plates may be compressed with a mallet and blunt instrument to compress the bone against the implant. The surgical decision to

WWW.HIGHDENT.IR

obtain rigid fixation is based on the width and height of bone available.

The internal aspects of the implant bodies are thoroughly irrigated to remove any blood or debris before the placement of the cover screw. The first-stage cover screw is then inserted into the implant body. An antibiotic paste may be placed on the threads of the cover screws before their insertion. In theory, this paste helps seal the cover screw–implant body connection and decrease the risk of bacteria growth within the implant body during healing. However, no clinical study is available to confirm this hypothesis. The cover screw is threaded to final position, then loosened and tightened again. The implant should not rotate during this process. When the cover screw is threaded into position, care is taken not to overtighten it. The stress transmitted may rotate the implant body and also complicate the cover screw removal at the second stage of surgery.

Soft Tissue Adaptation

The crestal bone is evaluated for any sharp edges, and a rongeur or bone file is used as indicated. The surrounding bone is also evaluated, because final soft tissue contour is directly dependent upon its topography. The surgical site is thoroughly irrigated with saline to remove any debris or bone fragments. The lingual retraction sutures are cut and removed. The tissues are approximated without tension, and a 3-0, resorbable, horizontal mattress interrupted suture is placed at the midline. An interrupted horizontal mattress or a continuous suture is placed on the distal aspect of the midline suture. It is important that the labial periosteum be joined to the lingual periosteum. Surface epithelium should not contact the periosteum along the suture line, because this may result in delayed healing and incision line opening. The sutures should be placed approximately 3 to 5 mm from the incision line, and 3 to 5 mm apart.

A firm pressure is applied to the reflected soft tissue for 4 minutes after the suturing is completed. This helps prevent bleeding under the soft tissue flaps and helps the tissue adhere to the bone, because fibrin released from the blood is natural tissue glue. A postoperative panoramic radiograph is taken at the conclusion of the procedure.

Because the anterior implants and primary healing covers are often placed after an osteoplasty, the labial flange of a removable prosthesis is overextended and needs to be shortened. The internal surface of the prosthesis is aggressively relieved 3 to 5 mm. A soft tissue conditioner replaces the acrylic. The tissue conditioner is also relieved over the implant sites so that the prosthesis is not in contact with the soft tissue directly over the implant sites. The patient may wear the denture during the healing process; however, the less it is worn, the less likely premature loading of the implant will occur. Incision line opening is a possible consequence of wearing the denture during the first 2 postoperative weeks; however, this is rarely a problem if the denture is properly modified and the patient wears it primarily for social encounters. The patient should not use denture adhesives or other products that may enter the incision line. The denture should not be worn at night, and the patient is instructed to coat it with petroleum jelly or soak it in water to maintain the pliable nature of the conditioner.

SURGERY IN THE POSTERIOR MANDIBLE–DIVISION A

There are 20 million Americans missing all of their mandibular teeth, and 40 million partially edentulous people are missing all their posterior teeth in one or more quadrants (Figure 31-27). Therefore 60 million people are candidates for implants in the posterior mandible. A primary indication for root form implants is the Division A posterior mandible. Dental implants are rarely inserted in the mandibular second or third molar location, and the first premolar is anterior to the mental foramen. Therefore the posterior mandible is primarily concerned with the second premolar and first molar region.

On occasion, surgery in the posterior region of the mouth is complicated by a small crown height space, especially when opposing teeth are present. The implant drills and hand piece require a space of at least 20 mm to the bony crest, so the osteotomies may be prepared with proper angulation. In addition, the implant and insertion tools often require at least 20 to 25 mm of space. This may be most difficult in the second molar region because the amount of space decreases posteriorly. If opposing teeth have extruded, the occlusal plane should be corrected before implant placement to increase the crown height space for surgery. On

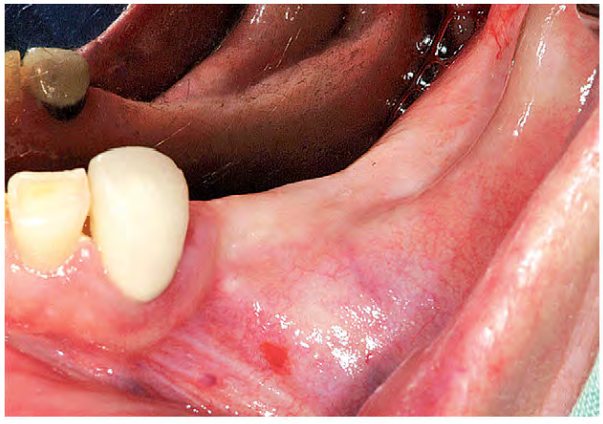


Figure 31-27 The posterior mandible is often missing multiple adjacent teeth.

WWW.HIGHDENT.IR

occasion, a shorter-length implant may be indicated to reduce the crown height space needs of the surgery.

The posterior mandible has greater initial width than the anterior mandible. As such, Division A bone is more commonly found early after tooth loss. The Division A bone has a height measurement that is determined from the crest of the edentulous ridge to the opposing landmark of the mental foramen or mandibular inferior alveolar nerve, artery, and vein. The ideal bone height includes 2 mm of surgical "safe zone" above the landmarks to decrease the risk of paresthesia by inadvertent violation of the structures. Therefore a 12-mm-long implant requires 14 mm of available bone height and a 9-mm-long implant requires 11 mm of bone height. The ideal length of an implant in the posterior regions is 12 mm or more; however, 9 mm has proven predictable when implant width, number, or design is also considered (Figure 31-28).[65] After the resorption of the edentulous ridge has evolved into Division B, it may last for more than a decade, so this condition is also often observed. Unlike the anterior mandible, the posterior mandibular Division B bone is less likely to be modified to Division A by osteoplasty, because the mandibular canal is on average 12 mm above the inferior border of the mandible and rarely permits enough crestal bone reduction to increase the width and still have enough height of bone for the Division A criteria. Therefore bone augmentation in Division B posterior mandibles is often warranted.

Many aspects of the surgical approach are similar to root form surgery in the anterior mandibular region. However, because the mandibular canal is the opposing landmark, rather than a thick cortical plate such as in the anterior regions, attention is specifically brought to the neurovascular complex.

The study models of the patient are duplicated, and a diagnostic wax-up of the final prosthesis is made. This step is important in determining the position of the abutment posts for the prosthesis (including the plane of occlusion, the height of the abutment for cement, and whether it is adequate for long-term cement retention), the need for pontics, the angulation of the post in relation to the abutment teeth, and the mesiodistal position of the implant. The study models, diagnostic wax-up, panoramic radiograph, and other diagnostic images are all available at the time of surgery.

Figure 31-28 A panoramic image of an edentulous posterior mandible, missing both premolars and molar. There is abundant bone height above the mandibular canal. Most often, the position of the mandibular canal may be observed in the first molar site.

The patient is premedicated, as suggested for any implant surgery, and anesthetized using an Akinosi block with both lidocaine 2% with 1:100,000 epinephrine and a long-acting anesthetic. Postoperative discomfort is reduced with long-acting anesthetic agents. The patient is prepared for surgery with a preoperative intraoral and extraoral Betadine or chlorhexidine scrub. The surgical team also scrubs and gowns in the standard aseptic method.

SOFT TISSUE REFLECTION

The surgical region is checked for regional anesthesia with local infiltration of 2% lidocaine with 1:100,000 epinephrine. Local infiltration also provides hemostasis and improved visibility during the soft tissue incision. In addition, when the local anesthetic is injected on the crest of the ridge and under the periosteum, a hydroelevation of the tissue may help the initial tissue reflection.

The primary incision is made on the crest of the ridge from the retromolar papilla (at the base of the pad) to the anterior tooth abutment. If there is 3 mm or more of attached gingiva on the crest of the edentulous ridge, the incision bisects this tissue. This places half of the attached gingiva width on each side of the cover screws or low-profile permucosal extensions. If there is less than 3 mm of attached keratinized tissue on the crest, the incision is made more lingually so at least 1.5 mm of the attached tissue is placed to the facial aspect of the implant abutments. In this way, the buccinator muscle is separated from the incision line and does apply tension and cause opening of the incision line during the initial healing, and gingival complications are reduced over the long term. The crestal incision is often continued in the sulcus and interproximal of the posterior teeth to the ipsilateral canine (Figure 31-29).

A secondary incision is made lateral to the retromolar pad and up the ascending ramus, to the height of the occlusal plane, and just below the two attachments of the temporalis muscle (Figure 31-30). On rare occasion, the buccal artery crosses over the ramus in this region and may be severed during the vertical release incision. This artery is a branch of the maxillary artery. Therefore a hemostat is readily available and clamps the soft tissue on the lingual aspect of the flap if excessive bleeding is noted. After 10 minutes, the hemostat may

WWW.HIGHDENT.IR

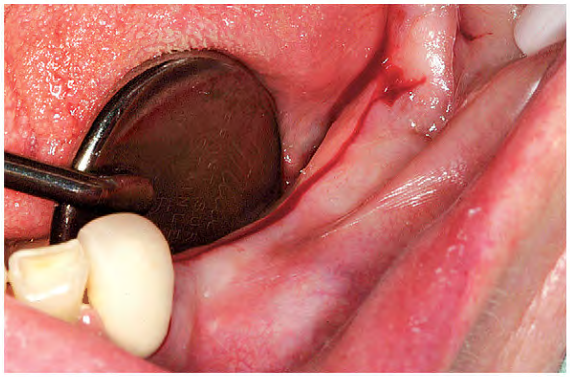


Figure 31-29 The posterior incision line is made on the crest of the edentulous ridge and often extends to the distal of the canine.

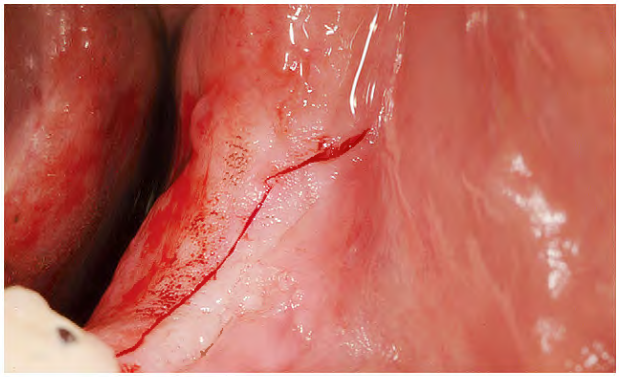
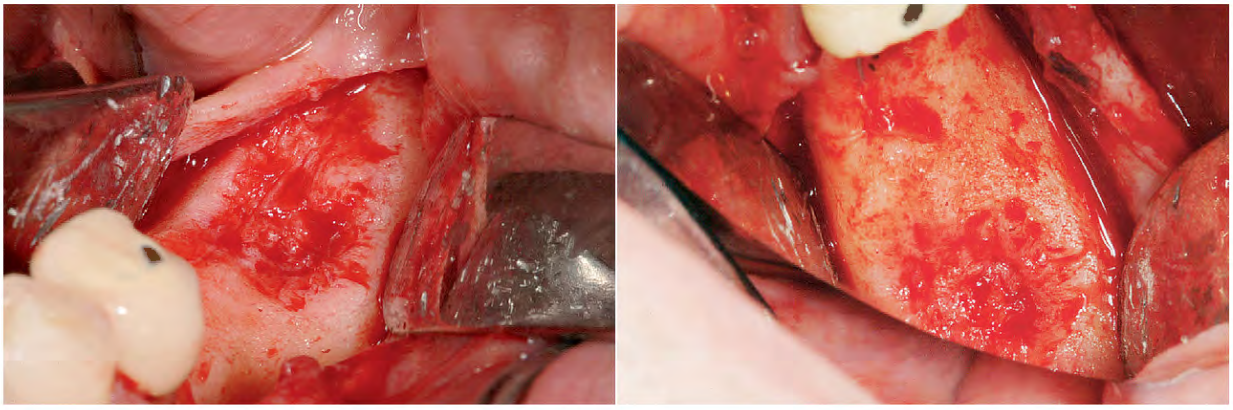


Figure 31-30 The distal release incision extends lateral to the retromolar pad, along the ascending ramus.



A B

Figure 31-31 **A,** The lingual flap is first reflected off the crest. **B,** The facial flap is reflected and, when a computed tomography scan is not available, usually exposes the superior aspect of the mental foramen.

be released and the bleeding will be arrested. Usually a secondary vertical release incision is also made just distal to the mandibular canine (which includes the distal papilla), which is always anterior to the mental foramen.

The lingual flap is first reflected only a few millimeters beyond the lingual crest of the ridge. Almost all concavities in bone of the posterior mandible occur on the lingual aspect in conjunction with the submandibular fossa. Therefore the region should be palpated and evaluated with a CT scan when available before osteotomy preparation. A full-thickness periosteal flap is then reflected on the facial, exposing the underlying crest and lateral surface of the edentulous mandible in the region of the foramen (Figure 31-31). When a CT scan of the region is not available and intraoral radiography is not easily present, the facial reflection should expose several millimeters of the lateral aspect of the edentulous ridge and the superior portion of the mental foramen. By exposing this area, the implant surgeon is less likely to inadvertently perforate the bone, which would result in fibrous tissue formation next to the implant in the region. In addition, the mental foramen is almost always identified in mandibular posterior surgery to determine the zone of safety above the mandibular canal when a CT scan is not available before surgery.[32]

The retromolar papilla should be elevated to the lingual to expose the underlying bone when implants may be placed in the mesial aspect of the second molar. The bone under the papilla in this area is usually more than 5 mm wide and 5 mm in length. The region is palpated just inferior to the retromolar pad to identify the submandibular anatomic landmarks. Severe undercuts inferior to this region are typical. As a result, root form implants are rarely placed under the papilla, but its reflection improves vision and access in the mesial half of the second molar area.

MANDIBULAR NERVE LOCATION: CLINICAL ZONE OF SAFETY

The risk of mandibular canal penetration during endosteal implant placement in the posterior mandible is a serious concern. At the time of surgery, intrusion into the mandibular canal results in an increased risk of hemorrhage, visibility impairment, and increased

WWW.HIGHDENT.IR

potential of fibrous tissue formation at the surface of the implant. More important, the patient experiences altered nerve sensation in the form of anesthesia, paresthesia, or hyperesthesia. This may affect the patient's lifestyle during eating, kissing, and applying lipstick and is a common cause for litigious action against the implant dentist. Consequently the surgical boundary is often set conservatively 2 mm above the mandibular canal to establish a surgical zone of error.

Procedures to Identify the Mental Foramen

A common approach to identify the existing available bone above the inferior alveolar canal is to start at the foramen, because this is the location in the posterior mandible where the reduction of the vertical height of bone begins. Compared with the anterior mandible, where the opposing landmark is the inferior border of the mandible, the posterior mandible is limited by the foramen and inferior alveolar complex.

The anteroposterior position of the mental foramen is variable and may correlate as far forward as the apex of the first premolar to as far distal as below the mesial root of the first molar. In a study by Cutright et al., the mental foramen location variance was different for white and black populations (Figure 31-32).[27] Mandibles for whites had the foramen farther forward, with 5 of 76 mandibles being under the first premolar (6.6%), 24 between the premolars (31.6%), 39 below the second premolars (51.1%), and 8 between the second premolar and first molar root (10.5%). Mandibles of black people had the foramen between the premolar 11 of 78 times (14.1%), below the second premolar 41 times (52.6%), between the second premolar and first molar 25 times (32%), and below the mesial root of the molar 1 time (1.3%). Therefore the average foramen of white people was below or in front of the second premolar (89.3%) and the average foramen in blacks was below or distal to the second premolar (85.9%).

The mental foramen vertical position is usually found more coronal and facial than the mandibular canal (Figure 31-33). Agthong et al. stated the foramen was 14 to 15 mm from the inferior border.[66] Neiva stated the position was 12 mm (range, 9 to 15 mm) from the lower cortex of the mandible.[36] Fishel et al. noted in a white population, the foramen was coronal to the apex of the first premolars 38.6% of the time, 15.4% at the apex, and 46% apical to the apex position. The position of the foramen in relation to the second premolars was 24.5% coronal, 13.9% at the apex and 61.6% apical.[29] Therefore the vertical position of the foramen is variable. On occasion, more than one mental foramen may be present. However, this appeared to occur less than 2% of the time in whites and 5.7% in blacks.[67]

Jacobs et al. evaluated panoramic radiographs and could observe the foramen 94% of the time, but only 49% were clearly detectable.[68] Fishel et al. found that

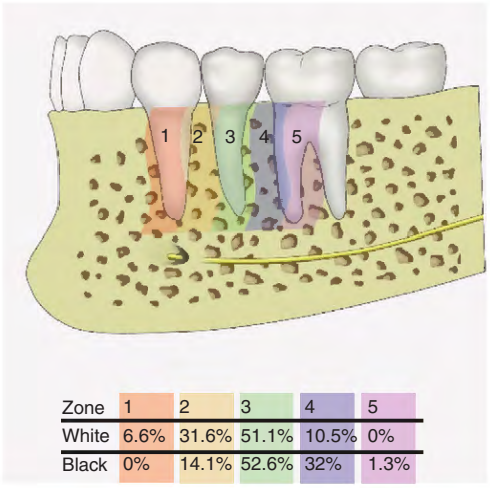


Figure 31-32 The mandibular foramen may extend as far forward as the first premolar to as far distal as between the second premolar and first molar roots. It is often correlated to sex and race.

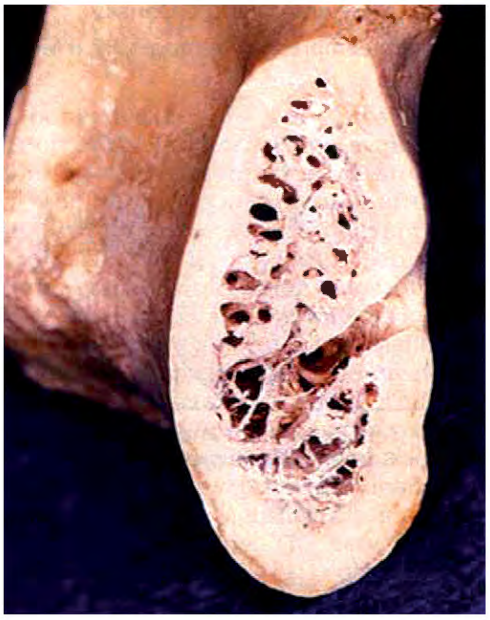


Figure 31-33 The mandibular foramen is usually coronal and facial to the position of the mandibular canal. The foramen is cone shaped, with the outer rim larger than the inner dimension of the foramen.

even the mental foramen was only visible on approximately 50% of periapical radiographs.[29] On the other hand, Yosue and Brooks[69] showed that the mental foramen could be identified on a panoramic radiograph 87.5% of the time. However, only 64% of the radiographs evaluated clearly showed the accurate position of the foramen. These same authors evaluated a panoramic x-ray and the actual skull in four subjects. The panoramic radiographs illustrated the mental foramen in the correct position less than 50% of the time, and often it was significantly closer to the inferior border of the mandible than its actual position. In addition, the appearance of the foramen varied with the positioning

WWW.HIGHDENT.IR

of the mandible in relationship to the focal length used while obtaining the radiograph.[69] Conventional tomography and CT are usually more diagnostic as to the position of the mental foramen.[28,70,71,72]

Procedures to Identify the Mandibular Canal

The available bone height over the mandibular canal is critical to determine whether adequate bone is present to place root form implants. The most common methods to assess this dimension are periapical radiographs, panoramic radiography and CT images with or without reformatted images.

Several clinical approaches to the posterior mandible have been proposed using radiographic techniques. Usually the inferior alveolar nerve proceeds anteriorly, inferiorly, then horizontally and laterally below the apices of posterior teeth and curves superiorly to exit at the mental foramen. It is reported to never transverse lower than 6 mm below the height of the foramen.[73] In the region of the first mandibular molar, the mandibular canal is often difficult to identify on a periapical or panoramic radiograph. A cortical lining of the canal is not always present in the molar region.

The angulation of the bone in the posterior mandible progressively evolves from almost vertical in the premolar region to 15 degrees in the first molar and 25 degrees in the third molar region. A periapical radiograph cannot be parallel to all three of these planes. In addition, the floor of the mouth prevents the periapical x-ray from being ideally positioned in relation to the edentulous ridge. As such, foreshortening or elongation of the image is expected.

Because the x-ray beam of a panoramic radiograph originates from below the patient's mandible, the location of the canal in reference to the crest of the ridge is dependent on the buccolingual position of the mandibular canal. If the nerve canal proceeds along the lingual aspect of the mandibular body, the canal will be projected more superior toward the crest. When the nerve canal proceeds toward the foramen on the buccal of the mandibular body, it will be projected more inferior in relationship to the crest. In other words, the distance from the crest to the mandibular canal may be the same in two different patients, but appear on the panoramic films as two different positions (Figure 31-34).

Despite obvious limitations, a panoramic radiograph is often used as the initial method to determine the height of the bone available over the mandibular canal. Most often, a panoramic radiograph can identify the entry of the mandibular inferior alveolar nerve into the ramus, because the lingula is cortical bone and the canal has a cortical lining, surrounded by softer trabecular bone. The mental foramen is also able to be identified on most films. Although a bifid canal has been observed, its occurrence is less than 1%.[74]

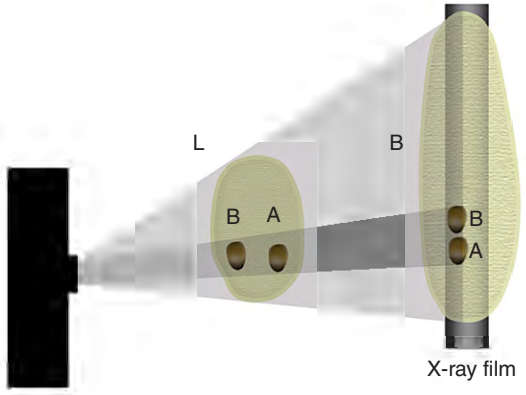


Figure 31-34 Although the distance from the crest of the ridge is similar in both of these nerve locations, when the canal resides more buccal in the mandibular body, it appears more apical on the x-ray film.

When present, the canal that exits the mental foramen is the opposing landmark, because sensory nerves that do not innovate soft tissue are of little concern for paresthesia.

After the panoramic radiograph is observed and the bone height over the radiographic canal directly measured on the radiograph, the patient may be placed in one of three categories: (1) there is obviously enough radiographic bone height over the canal and foramen (>15 mm), (2) there is obviously not enough height over the landmarks (<9 mm), or (3) the adequate bone height is questionable. The magnification of the radiograph is initially ignored to determine in which of the three categories the available bone height is placed.

A 25% manufacturer-supplied magnified image of the implant body may be placed over the radiograph to help select the desired size of implant to be inserted in the abutment positions. This magnified image is not accurate, but may help to determine whether the patient obviously has enough bone. These magnified images often prove imprecise in marginal cases because panoramic radiographs do not have a uniform magnification rate. Additional variables such as the patient's head position may significantly alter magnification by as much as 55%. In addition, at the time of surgery, the crest of the edentulous ridge is often modified by osteoplasty to increase the width of crestal bone. The premeasured bone height is reduced by a dimension that may be difficult to assess.

A study by Sonick et al. compared the location of the mandibular canal with panoramic and CT radiographs of a cadaver mandible.[72] The average distortion of the panoramic image was 3.0 mm (range, 0.5 to 7.5 mm) and the CT average was 0.2 mm (range, 0 to 0.5 mm). Therefore the panoramic method of mandibular nerve location would be limited to obvious extremes of abundant or inadequate bone.

The cortical lining of the mandibular canal is often absent in the first molar region and even when present may be confused with a vascular region surrounded by

WWW.HIGHDENT.IR

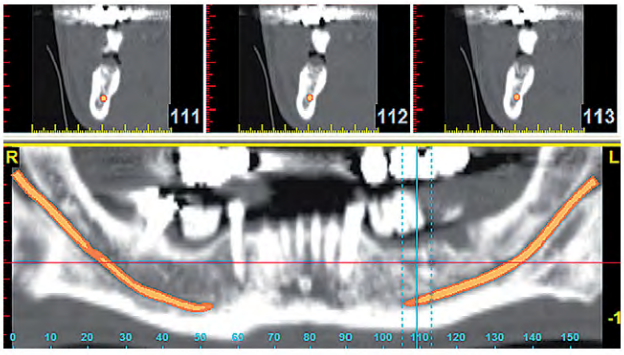


Figure 31-35 A computed tomography–reformatted image of a mandible allows the identification of the mandibular canal in several planes. Therefore the width of available bone above the nerve, the height of available bone, and the angulation of the bone may be determined.

trabecular bone. As such, tomograms or an individual CT image may not easily determine the position of the mandibular canal. A computer-generated tomogram series of reformatted images represents an improved diagnostic tool for identifying bone width, height, and mandibular canal location (Figure 31-35). Therefore when the diagnosis is unclear, the use of a CT scan and the reformatted images into a computer software package (i.e., Materialise, Implant Logics) is a considerable advancement in diagnostics for the location of the mandibular canal. Using a CT scan is most indicated when the panoramic radiograph gives an uncertain answer relative to the available bone in the region. When questionable bone height is present, the CT scan is indicated to determine a more exact canal position and whether an implant may be inserted above the mandibular canal.

A surgical approach without block anesthesia has been suggested to allow implants placement above the canal in questionable ridges.[75] In this technique, local anesthesia is achieved by infiltration into the surrounding soft tissues. The bone is then prepared without anesthesia, because few sensory nerve fibers exist in this region, except those of the mandibular canal. The patient is instructed to notify the surgeon if sensation or discomfort is experienced, which theoretically correlates to the rotary instrument approaching the mandibular nerve. However, the infiltration surgical approach presents several disadvantages. With this approach, sedation must be minimal to enable the patient to communicate intelligently during the procedure. The misinterpretation of a pressure phenomenon or bur vibration raises the patient's and doctor's anxiety regarding nerve encroachment. Additional sensory mandibular nerve pathways are often present within the bone. These nerves innervate teeth but little or no soft tissue of the face. Apprehension or misinterpretation by the doctor or patient with this technique can result in the selection of a short endosteal implant of lesser bone-implant surface area. This condition may lead to greater bone loss and failure and is not recommended.

The placement of endosteal implants medial to the mandibular canal has been reported.[76] However, the position of the canal in a transverse plane is not reliable. The inferior alveolar nerve enters the medial aspect of the ramus above the lingula and exits on the lateral aspect of the mandibular body at the mental foramen. The crossover from medial to lateral aspects usually occurs in the third to second molar region, but this is highly variable. Attempting to place an implant medial or lateral to the canal as a result of an average anatomic location or, more recently, with information from the CT image has been suggested.[76-78] This technique requires precise control of the angulation of penetration at the crest of the edentulous ridge. Surgical error of a few degrees may result in perforation of the canal. Visualization of a few degrees is most difficult in the posterior regions of the mouth when observing the mandible from the lateral aspect of the patient. As a result, the general rule is to place the implant above the mandibular canal.

ZONE OF SAFETY

The zone of safety is defined as an area within the bone that can safely support implants without fear of impingement on the mandibular neurovascular bundle. A zone of safety for the placement of posterior mandibular endosteal implants was established by Misch in 1980 by the evaluation of 530 panoramic radiographs of partially edentulous patients. In 1989 this evaluation was confirmed by Misch and Crawford with an additional 324 consecutive panoramic radiographs.[32]

The zone of safety is a clinical method to assess a safe zone to insert endosteal implants. It is used when a CT image does not confirm the presence of adequate available bone height. Therefore when periapical or panoramic images are the only diagnostic tool to assess available height and abundant height seems present (>15 mm), two treatment methods may be used:

1. A 2-mm pilot drill prepares an implant site 9 mm deep. A 9-mm-long force direction indicator is positioned in the osteotomy, and a periapical radiograph is made. The indicator is evaluated in light of the opposing landmark, and a decision is made to remain at 9 mm or reprepare the site to 12 mm deep.
2. The mental foramen is reflected and the safe zone is established before drilling the initial osteotomy depth.

The zone of safety is determined either on panoramic radiographs or clinically during surgery as follows. Line A is drawn parallel to the posterior plane of occlusion, at the level of the residual crestal ridge and at the sites of implant placement. Line B is drawn at the most superior aspect of the mental foramen parallel to line

WWW.HIGHDENT.IR

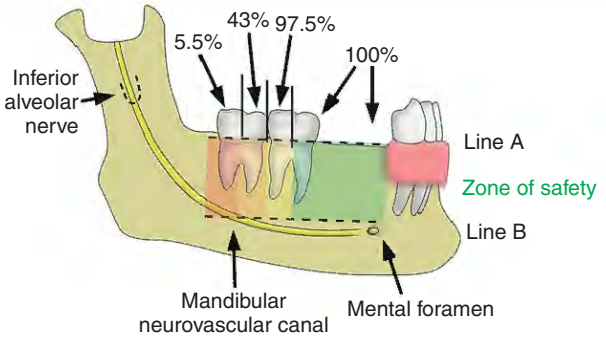


Figure 31-36 Mandibular zone of safety. Draw an imaginary line parallel to the plane of occlusion at crest of the posterior ridge **(A)** and another parallel line at the top of the foramen **(B).** The height of bone between these lines is the zone of safety to the mid first molar region.

A. Lines A and B are joined with a perpendicular line C. The length of line C is the safe zone measurement to the mesial half of the first molar (Figure 31-36).

The results of studies by Misch and Crawford[32] indicated the prevalence of the mandibular canal below the second line (B) on radiographs, using the landmark of the mental foramen. A zone of safety was observed 100% of the time mesial to the middle of the mandibular first molar. The most common position of the mandibular canal was 2 mm or more inferior to line B (drawn from the top of the foramen). Therefore the area within the zone of safety is above the mandibular canal and allows a surgical safety zone that most often approximates 2 mm. In the region of the distal half of the first molar, the mandibular canal was below line B (within the safe zone) in 97.5% of radiographs. In the region of the mesial half of the second molar, the mandibular canal was below line B in 43% of the radiographs; in the distal half of the second molar, it was only 5.5% (Table 31-3). Because implants are not often placed in the second molar area, the zone of safety has proven a useful clinical tool.

The position of the foramen is also an anatomical landmark for mandibular implant placement in the premolar region. Because the foramen is most often found distal to the first premolar, implants are often considered safe to place in the first premolar region, just anterior to the foramen. An anterior loop of the mandibular canal occurs when the mandibular nerve proceeds inferior and anterior to the position of the foramen, then loops superior and distal to reach the foramen. An anterior loop was observed in 12% of the panoramic radiographs and up to 3 mm in length anterior to the foramen.[32] Therefore when this condition exists, first premolar implants should usually be placed above the mental foramen position.

Safe Zone Procedure

The zone-of-safety technique is used before and during surgery for endosteal implant placement in the posterior mandible, when a CT scan was not used to determine the height of available bone and when a panoramic radiograph clearly indicates enough bone is present above the canal (>15 mm). First, radiographic estimates of vertical bone height are made by measuring the distance between lines A and B in the safe zone. Correction of this measurement (from magnification) may be accomplished by using calibrated balls or wires and using ratios of magnification or an arbitrary 25% magnification. The approximation of the available bone height is used to develop the treatment plan. An implant height may be initially determined, with the understanding that the safe zone is actually measured at the time of surgery and may be different because of osteoplasty or incorrect magnification allowance. If the final estimates of available bone height are less than 15 mm to the canal, it is suggested a CT scan be obtained before surgery to determine precise mandibular nerve position in this region.

Bone Preparation

If the edentulous ridge is narrower than 6 mm, an osteoplasty is performed under copious irrigation, as long as the remaining height of bone is greater than 12 mm above the foramen position. As a result of the osteoplasty, the crest width is increased, but the anatomic landmark of the mandibular canal is now closer to the remaining crest of bone.

The ideal implant position is level with both cortical plates. Bone remodels to the height of the lowest cortical plate. The height of the available bone is then determined, forward of the mesial half of the first molar. An imaginary line A is drawn parallel to the

Table 31-3 Mandibular Canal Location: Posterior Mandible*

LOCATION	MESIAL FIRST MOLAR	DISTAL FIRST MOLAR	MESIAL SECOND MOLAR	DISTAL SECOND MOLAR
+	324 (100%)	316 (97.5%)	149 (43%)	18 (5.5%)
0	0	6 (1.8%)	166 (51%)	34 (41%)
–	0	2 (<1%)	8 (5.5%)	172 (53%)

+, Within zone of safety; *0*, below zone of safety but not touching canal; –, touching or below top of canal.
* Data from 324 consecutive panographic radiograph measurements.

WWW.HIGHDENT.IR

occlusal plane on the crest of the residual ridge after osteoplasty. Another parallel imaginary line B is established at the superior aspect of the mental foramen. The height of the available bone in the second premolar and mid-first molar region corresponds to the distance between these lines and represents the maximum height of the endosteal implant in this region (Figure 31-37).

The radiograph is then evaluated to confirm and extend the zone of safety beyond the mid-first molar region. If the canal appears to remain at the same distance as the zone of safety in the distal of the first molar region (97.5% of the time), the safe zone measurement is expanded to the distal root region. If the canal appears to rise 2 mm or more in the mesial of the second molar region (about 60%), the safe zone measurement is reduced accordingly in this region. The distal half of the second molar region is almost always reduced from the safe zone measurement (94% of the time) and usually is at least 4 mm less than the safe zone height. The osteotomy preparation may be started if adequate bone height is present. It is recommended to err on the side of safety in the region past the mesial half of the first molar, using the panoramic radiograph as a guide. This clinical approach has been used successfully over two decades in thousands of surgical procedures without paresthesia.

The premolar site implant osteotomy may proceed directly over the foramen to the same safe zone measurement without the implant osteotomy impinging on the nerve, based on three anatomical considerations. Although the crossover position of the mandibular canal from the medial aspect of the ramus to the lateral area of the body of the mandible is variable and may occur anterior to the second molar region, the mandibular nerve always exits the mandible facially at the foramen. Therefore the implant osteotomy is lingual to the canal and its contents in the foramen region. In addition, the mandibular foramen is cone shaped, with the base of the cone oriented facially and its summit lingually (see Figure 31-33). Therefore, when the height measurement is made from the top of the external portion of the foramen, this represents the more superior portion of the cone-shaped foramen. In addition, the nerve exits from a path inferior to the foramen, so the nerve approaches below the height of the external measurement. Thus the nerve is apical and lateral to the area of the implant osteotomy, when it is directly over the foramen and at the height of the mental foramen.

After the measurement is made for available bone height, the implant osteotomy locations may be established every 7 mm (for 4-mm-diameter implants), starting 3.5 to 4 mm distal from the last abutment tooth (Figure 31-38). An abutment location point is made on the midcrestal bone of the ridge with a round No. 6 or pilot bur (Figure 31-39). A measuring tool may be used to measure the interimplant distance on one side and the tooth-to-implant position on the other side. If the pilot holes for the implant osteotomies are positioned too close to each other, it may be separated by an

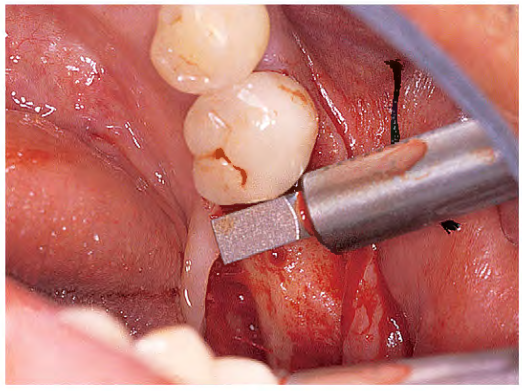


Figure 31-38 A measuring tool is placed on the distal of the tooth, so that the center of the implant site is 3.5 to 4 mm distal to the tooth. In this way, the 4-mm diameter implant will be 1.5 to 2 mm distal to the tooth.

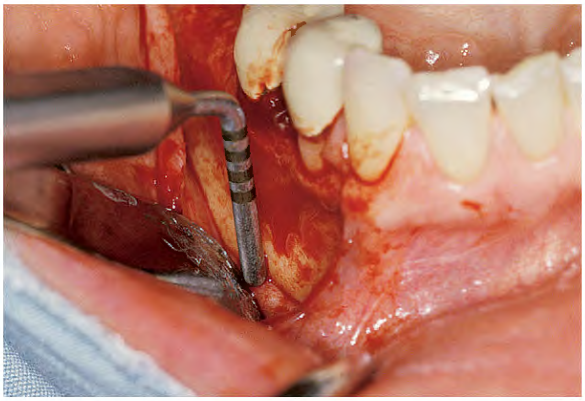


Figure 31-37 A probe is positioned over the mental foramen and measures the vertical distance to the crest of the edentulous ridge. This measurement is compared with the preoperative x-ray and determines the zone of safety for posterior implant length.

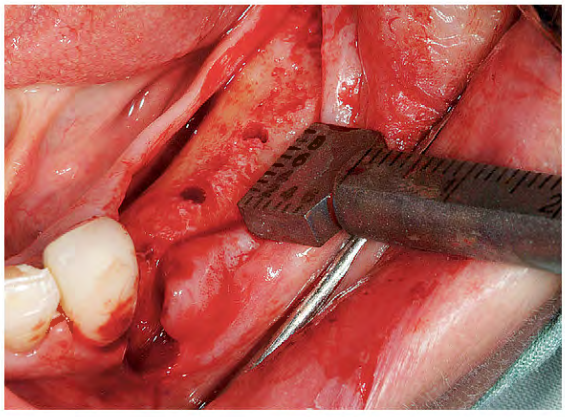


Figure 31-39 The implant sites are at least 7 mm apart in the posterior mandible so that the implants will have 3 mm of bone between them.

WWW.HIGHDENT.IR

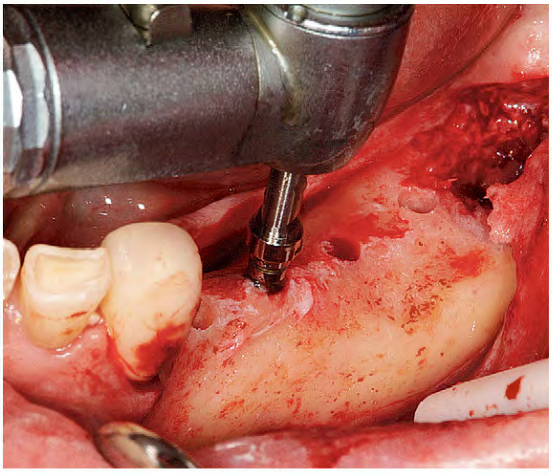


Figure 31-40 A 2-mm end-cutting drill used with copious amounts of saline prepares the osteotomies to 9 mm (when more than this height is available).

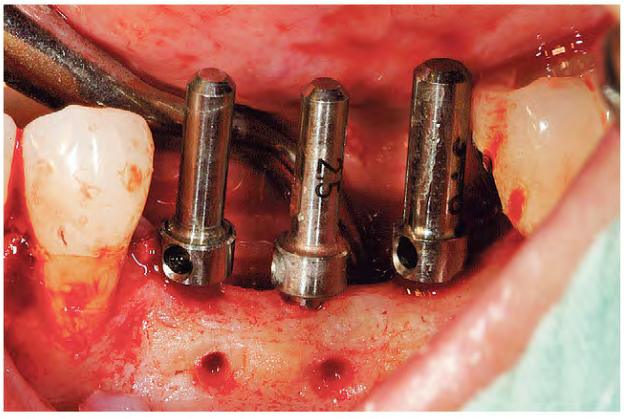


Figure 31-41 The force direction indicator is positioned into the osteotomy and evaluates implant position.

additional 1 mm by "stretching" the hole away from the problematic implant or tooth with a side-cutting Lindemann drill. This same technique is used if the osteotomy is too facial or lingual.

A 2-mm end-cutting drill with copious amounts of saline then prepares the initial osteotomy at 9 mm deep (when more than this height is available) (Figure 31-40). After the implant osteotomy is 2 mm in diameter, a force direction indicator (parallel pin) is inserted (Figure 31-41). The patient gently closes into occlusion, and the direction of force is noted. The indicator should be positioned between the central fossa and palatal cusp of the maxillary teeth. This permits the occlusal loads to be ideally directed onto the implant body.

The surgical template is fitted over the remaining teeth and compared with the pilot hole positions. A periapical radiograph may be made at this time to verify the position away from the adjacent roots and confirm the available bone height (Figure 31-42). If the preselected implant length needs to be modified from

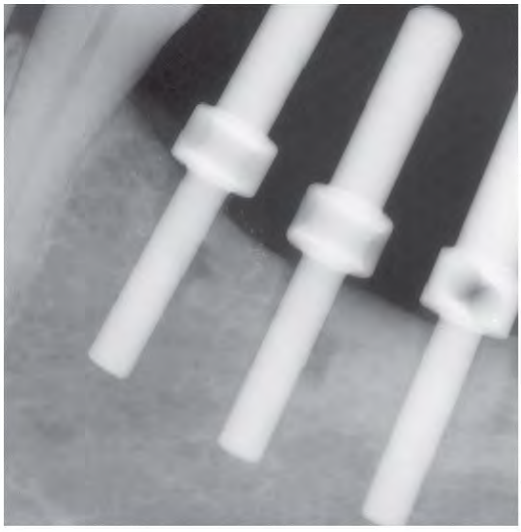
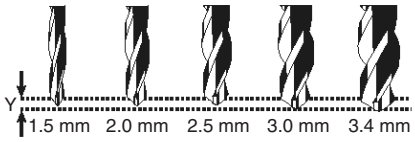


Figure 31-42 A periapical radiograph is taken with the direction indicator in place to confirm the implant position in relation to the adjacent roots and mandibular canal.

that planned on the basis of discrepancy between the initial radiographic estimations and present clinical measurements, a different-length implant is selected at this time. The available bone and implant should be at least 9 to 10 mm in height from the crest of the ridge to the inferior portion of the implant body. Larger-diameter implants (or two regular size implants) are used in the molar regions when possible to account for the greater force magnitude in the posterior regions of the mouth.

The depth of the millimeter gauge lines inscribed on the shank of the drills most often do not include the cutting edge of the drill and are not to the actual depth of the drill. Most drills have a sharp V-shaped apical portion to improve their cutting efficiency. The V-shaped apical portion of the drill (called a *Y dimension* in engineering) is often not included in the depth measurements of the commercial drills and may measure as much as 1.5 mm on larger drill diameters. As a result, implant companies often provide a caution statement included in the implant company surgical manual (drills prepare sites deeper than the depth gauge lines indicate) (Figure 31-43).[79-81] In addition, some companies include the healing cap height along with the implant depth gauge lines. Some systems make the drill depth gauge 1 mm longer than the implant (a 13-mm implant has a corresponding line at 14 mm on the drill). Other systems make the actual implant 1 mm shorter than the corresponding drill gauge (a 13-mm implant is really 12 mm, and the drill line is 13 mm). Some manufacturers make the depth line of the drill the same as the implant length. This variance in corresponding depths becomes critical when preparing bone sites over the mandibular canal or when exact measurements are required. Therefore it is necessary to evaluate the manufacture's drills and compare them

WWW.HIGHDENT.IR



Drill diameter	Y dimension
1.5 mm	0.43 mm
2.0 mm	0.58 mm
2.5 mm	0.74 mm
3.0 mm	0.86 mm
3.2 mm	0.94 mm
3.4 mm	0.99 mm
3.7 mm	1.07 mm
4.0 mm	1.17 mm
4.2 mm	1.22 mm
4.4 mm	1.27 mm
4.7 mm	1.35 mm

Figure 31-43 The Y dimension or cutting edge of a drill is often not included on the depth lines of a drill. Therefore the 9-mm depth line on a 4.7-mm-wide drill may actually represent 10.35 mm of cutting depth.

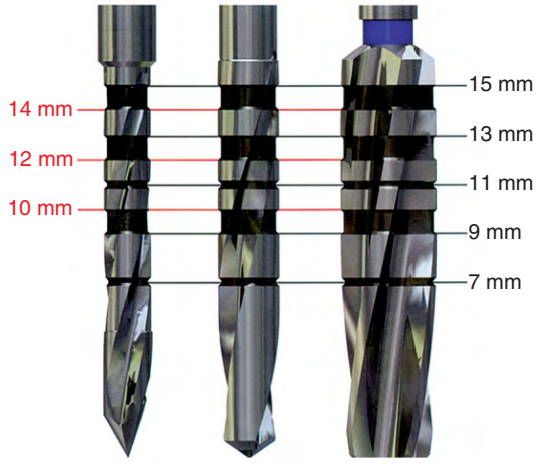


Figure 31-44 The depth lines on these drills include the Y dimension. The larger-diameter drills have no V-shaped end and therefore no Y dimension (BioHorizons Dental Implants, Birmingham, Ala.).

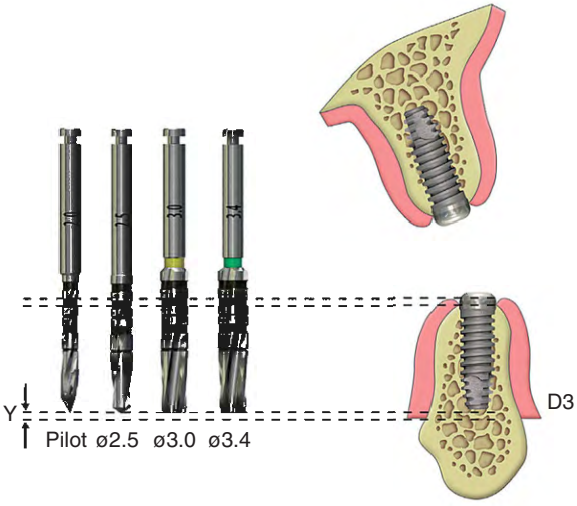


Figure 31-45 The drill sequence protocol is related to the bone density. The 2.0-mm pilot drill is most often followed by the 2.5- and 3.0-mm intermediate drills. The 3.4-mm final drill may be followed by implant insertion in D3 bone.

with the length of the implant before preparing a posterior osteotomy above the mandibular canal.

The apex drill design was modified by Strong et al.[82] to eliminate the Y dimension of a drill (Figure 31-44). The flat end in the larger-diameter drills also acts as a stop, because it does not proceed deeper than the osteotomy depth of the pilot drill. This drill design is of most benefit in the posterior mandible, when the implant approaches the mandibular nerve location.

Osteotomy preparation proceeds following the surgical protocol adapted to the bone density (Figure 31-45). A 2.5-mm end-cutting drill is followed by a 3-mm drill and the final drill dimension for the system employed. A countersink drill and a bone tap are only used for D1 bone. In the posterior mandible, D2 bone most often uses a countersink drill for a larger crest module, but does not use a bone tap. D3 bone does not use a crest module drill or a bone tap. A posterior mandible that has been edentulous for several years may present a fine trabecular pattern under a thin crestal cortical plate. The less dense trabecular bone does not require the entire osteotomy depth to be prepared before implant placement. When the available bone height is marginal and the trabecular bone is less dense, the drill may be stopped 2 mm shorter than the full osteotomy depth. In this way, the risk of nerve injury is significantly reduced. A countersink drill is not indicated under these conditions. The implant is inserted to the complete depth by compression of the trabecular bone around the apical 2 mm of the implant. The crest module of the implant compresses the thin cortical plate and increases initial fixation. When the trabecular bone is fine, countersinking the implant may place it below the crestal cortical bone, causing decreased stability during healing. The posterior mandible may exhibit greater flexure and torsion compared with the anterior mandible as a result of parafunction, and the crestal bone may be required to stabilize the implant during healing. On occasion, the lingual cortical plate at the end of the osteotomy may prevent complete seating of the implant, as a result of the submandibular fossa in the region. In these cases, the depth of the osteotomy must be completely prepared, as in the anterior mandible.

The surgical sites are thoroughly rinsed to remove any debris or contaminants before inserting the implant. Otherwise, the implant may compress the contents of the site beyond the apex and place pressure against the mandibular nerve. This may cause a transitional paresthesia. The bone tap (in D1 bone) and implant insertion is performed at a reduced speed of less than 30 rpm with a high-torque, slow-speed hand piece (Figure 31-46). Implant insertion most often occurs without irrigation. The crest module is often placed

WWW.HIGHDENT.IR

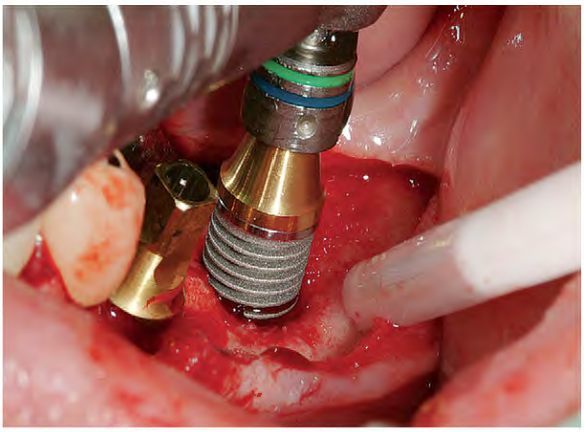


Figure 31-46 A low-speed hand piece threads the implant into position, usually without irrigation.

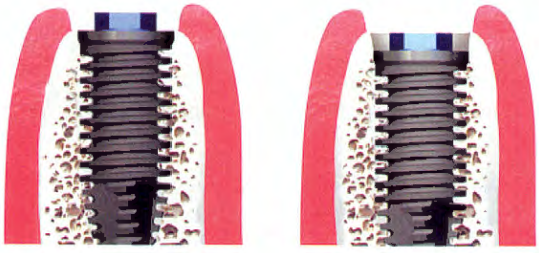


Figure 31-47 **A,** For external hex designs, the implant platform may be placed even with the crest of bone as suggested for D2 bone and when the implant is less at risk of movement during healing. **B,** In D4 bone, the implant is more at risk of movement during healing. As a result, it is countersunk below the crest of bone, so the implant cover screw is level with the crestal bone. The location of the implant platform determines the depth of implant osteotomy.

level with the crest of the ridge in soft bone and slightly above the crest in good-quality bone (Figure 31-47).

The first-stage cover screw is inserted for a two-stage surgical approach, or a permucosal healing extension (PME) is inserted for a one-stage surgical approach. A one-stage approach is used when the patient does not wear a soft tissue–borne partial denture. This approach eliminates the second-stage surgery and allows the tissue to be mature at the time of prosthetic fabrication (Figures 31-48 and 31-49).

The tissues are approximated and sutured with 3-0 or 4-0 polyglycolic acid PGA or Vicryl suture. The accessory anterior incisions are approximated first to ensure proper tissue placement after reflection. Most often, the suture is positioned within the mid-papilla in the distal of the canine from the facial. The suture then picks up the lingual aspect of the papilla and proceeds to the medial of the canine. After it is looped around the canine, it is tied to the suture end and the distal papilla is pulled medial up against the distal of the canine. An interrupted suture is placed below in the vertical release incision to allow primary closure of the soft tissue in the correct position. A continuous nonlocking suture or interrupted sutures may be placed

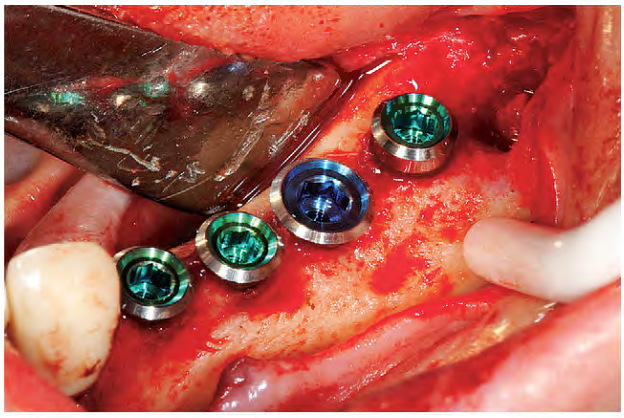


Figure 31-48 A one-stage implant surgical approach is often used in good-quality bone and when the patient does not wear a soft tissue–borne transitional prosthesis.

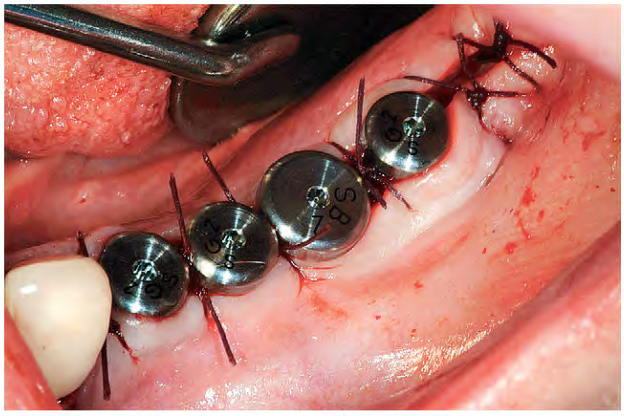


Figure 31-49 The cover screw or permucosal extensions are exposed in the one-stage surgical approach. The tissues are approximated with a resorbable suture (PGA or Vicryl).

along the crest of the ridge. Pressure is then applied to the soft tissue over the surgical site for 4 minutes to arrest any bleeding and help adapt the periosteum to the bone.

IMMEDIATE POSTOPERATIVE PROCEDURES AND INSTRUCTIONS

After surgery for the anterior mandible, the patient's mouth is rinsed with saline, and moist gauge sponges are rolled and inserted over the surgical site. Several other 4 × 4-inch gauze sponges are rolled together and placed intraorally between the arches. The patient bites on the gauze packs for approximately 1 hour. A panoramic radiograph is taken at the conclusion of surgery while the transitional prosthesis is modified (Figure 31-50). The radiograph is closely evaluated to ensure proper implant placement in relationship to the opposing landmarks or surrounding structures. If inadequate placement is observed or infringement on the mandibular canal is evident, the implant

WWW.HIGHDENT.IR

Figure 31-50 A panoramic radiograph is taken at the conclusion of the surgery to confirm the implant position away from the mandibular nerve and adjacent teeth.

placement should be corrected at this surgical visit. On rare occasions it has been necessary to remove the sutures and reposition an implant after analysis of the panoramic radiograph. The threaded implant may be unthreaded one to two revolutions to lift an implant away from a neurovascular canal when necessary. Decompression of the nerve complex decreases the risk of paresthesia. It is far better to perform this correction surgery while the patient is still anesthetized and the soft tissue is not healed than to wait for a future appointment.

In the posterior partially edentulous patient, the posterior region most often is out of the esthetic zone and does not require a transitional prosthesis during hard and soft tissue healing. The usual course of events after the surgical procedure is uneventful. If the patient needs to wear a removable partial denture with a metal framework, it is recommended to make holes through the metal mesh in the saddle area corresponding to implant placement before relining with a soft tissue conditioner. Removing only the acrylic under the metal framework on the saddle area does not provide adequate relief for the surgical site, because the acrylic is usually 0.8 mm thick.

Occasionally edema develops in the floor of the mouth, accompanied by minor hematoma formation. However, the edema may extend extraorally and include the chin, especially if the inferior cortical plates were perforated. An occasional short-term paresthesia of the lip is possible because of the edema. Analgesics and corticosteroids are recommended the first few days following the surgery, and the protocol outlined in Chapter 21 is implemented.

The patient is provided with written postoperative instructions highlighting areas such as rest, application of ice and pressure with gauze packs, and steps to follow in case of bleeding, suture line opening, and pain. Prescription of chlorhexidine gluconate rinses and the use of salt water rinses are also recommended. Usual postoperative instructions similar to those after oral surgery procedures are implemented. The patient returns in 10 to 14 days for suture removal and observation, even though resorbable sutures are used. Polyglycolic resorbable sutures require 6 to 8 weeks to resorb, and on rare occasion small fistulae form in this time frame. Any fistula, regardless of cause, affects the pH in the area and can cause bone loss; therefore the sutures are removed at the postoperative visit.

Tissue conditioners are selected for the immediate postoperative period because they are able to change dimension during the first 24 to 36 hours under pressure. As a result, excess force from swelling or occlusion on the tissue in any region will cause modification of the material. However, tissue conditioners become more rigid faster than the soft tissue liners. In addition, silicone-based products harbor more bacteria and promote yeast formation; therefore the tissue conditioner is removed at the suture removal appointment. The internal surface of the denture is then relieved, and a soft tissue liner is placed, which is also relieved over the implants in the surgical region. The patient is scheduled for an additional soft tissue checkup in 3 weeks to confirm full maturation of the soft tissue.

COMPLICATIONS

Nerve Injury

One of the most troublesome complications—short and long term—of bone harvesting (i.e., ramus block graft), third molar extractions, and implant surgery in the posterior mandible is nerve injury (Figure 31-51). The two most common nerves of concern are the inferior alveolar nerve (IAN) and the lingual nerve. The trauma from surgery or administration of local anesthetic to branches of the fifth cranial nerve may lead to loss or altered sensation (paresthesia) or, worse, to painful symptoms (dysesthesia). A thorough presentation of this topic is beyond the scope of this text; however, the clinician should know how to evaluate and refer when appropriate when associated symptoms

Figure 31-51 Implant surgery in the posterior mandible is a primary cause of mandibular nerve injury.

WWW.HIGHDENT.IR

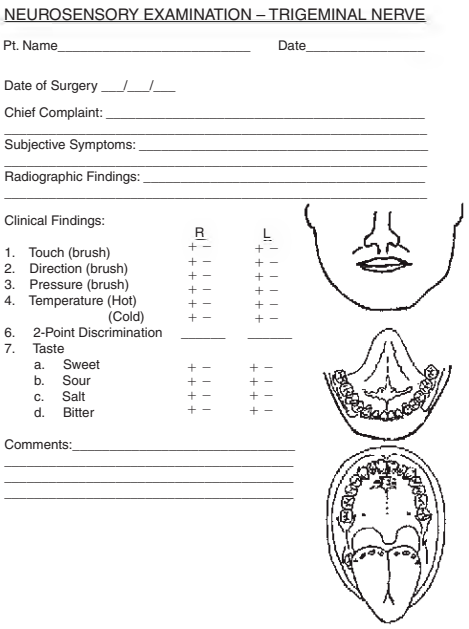
present, or when a patient complains of the subjective or objective difference in feeling after an implant surgery.

It is beneficial to examine the patient as soon as practical as to the extent of the symptoms and to determine the classification of nerve injury (Table 31-4) (Figure 31-52). The goal of the examination is to ascertain (1) the character of the sensory disturbance, (2) the subjective degree and changes to the sensory loss, (3) the objective consequences, and (4) the start and duration. There are two steps to this process: the patient interview and sensory testing.

Character of Altered Sensation

The character of the sensory disturbance may be described in many ways. When the patient is first examined, the subjective chief complaints are identified in the patient's own words. Numerous unpleasant sensations may be described by the patient, including: (1) numbness, (2) a crawling feeling, (3) sharp and constant or periodic pain, (4) itching, (5) tingling, (6) hypersensitivity, (7) burning, (8) throbbing, (9) pins and needles, (10) prickling, and (11) warm or cold. In implant dentistry, these sensations more often affect the mandibular lip, chin, and lower anterior gingiva. Less often, the tongue may also have altered sensations. Patients with tongue symptoms may also experience loss of taste. As a consequence of these symptoms, lip or tongue biting and difficulty in shaving or applying lipstick, drinking, kissing, and even speaking have been reported. Patients affected with any of these sensory disorders of the face may complain of depression and difficulty in personal relationships.

The goal of the interview is to ascertain (1) the character of the sensory disturbance, (2) the start and duration, (3) the subjective degree and change to the sensory loss, and (4) the objective consequences (Box 31-4).


NEUROSENSORY EXAMINATION – TRIGEMINAL NERVE

Pt. Name_____ Date_____

Date of Surgery ___/___/___

Chief Complaint: _____

Subjective Symptoms: _____

Radiographic Findings: _____

Clinical Findings:

	R	L
1. Touch (brush)	+ –	+ –
2. Direction (brush)	+ –	+ –
3. Pressure (brush)	+ –	+ –
4. Temperature (Hot)	+ –	+ –
(Cold)	+ –	+ –
6. 2-Point Discrimination	_____	_____
7. Taste		
a. Sweet	+ –	+ –
b. Sour	+ –	+ –
c. Salt	+ –	+ –
d. Bitter	+ –	+ –

Comments:_____

Figure 31-52 Neurosensory examination for the evaluation of trigeminal nerve injury.

Degree and Changes of Altered Sensation Forms

The degree of subjective sensory loss and changes at subsequent appointments is noted at the patient interviews and examinations. The sensations may worsen over the following months (i.e., paresthesia evolving into dysesthesia), but most often the symptoms begin to resolve over the next few months. It is unusual for the character of the altered feeling to change during this time frame. For example, before the myelin sheath (which acts as a coating on an electrical wire) grows over the new axons, a hyperesthetic state may be present.

The second part of the patient examination at each appointment includes sensory tests. There are several

Table 31-4 Neurosensory Examination and Treatment

POSTSURGERY PERIOD	DOCUMENTATION	PHARMACOLOGIC INTERVENTION	TREATMENT	REFERRAL
48 hours	Radiographic examination Neurosensory examination	Corticosteroids	Implant evaluation: removal or reposition if impingement within the mandibular canal	None, unless unfamiliar with neurosensory testing
1 week	Neurosensory examination (testing should be continued every 2 weeks)	High-dose nonsteroidal anti-inflammatory drugs (600-800 mg TID) for 3 weeks	Palliative	Refer to microneurosurgeon if: known nerve transaction, dysesthesia, or complete anesthesia
8 weeks	Neurosensory examination	As needed	Palliative	If no sign of improvement, refer to microneurosurgeon

WWW.HIGHDENT.IR

Box 31-4 Altered Sensation Evaluation

- Character of disturbance
- Start and duration: monitor first month, then every 4 weeks for 3 months
- Subjected degree and change to sensory loss
- Objective consequences

Box 31-5 Sensory Testing (Bilaterally)

- Sharp needle test (tingle or painful)
- Shortest distance between indentation
- Mapping area of altered feeling
- Blunt cotton swab test (tingle or painful or none)
- Pulp testing teeth
- Temperatures test (cold, warmth) optional

simple tests that may be conducted within the office (Box 31-5).

1. A sharp needle test with the end of a 27-gauge needle indents the skin and mucosal surfaces (but should not penetrate) while the patient describes the sensation with their eyes closed (i.e., tingling, painful).
2. The shortest distance the patient can determine between two indentation points with the needle should also be identified. A normal distance between two indention points varies from 5 to 15 mm from one person to another. The same two procedures are performed on the unaffected side of the mouth/face.
3. An eyeliner pencil is used to map out the area of altered feeling and a photograph or a drawing is made of the region.
4. After the sharp needle test, the blunt end of a cotton swab may be used over the affected and unaffected regions while the patient describes the sensation.
5. Pulp testing of the teeth is an effective method to test sensory function of the inferior alveolar nerve.
6. When temperature complaints have been rendered by the patient, another sensory test may be the application of cold (e.g., ice) or warmth to the area and have the patient describe the sensation.

Painful stimuli are often the first function of the nerve to return after altered nerve sensation. However, when painful stimuli are an initial finding and continue for more than 1 month, the painful stimuli should be reevaluated after block anesthesia. Failure to relieve pain after a mandibular block indicates a central-sympathetic or psychologic problem rather than a peripheral cause.

Duration of Altered Sensation

The start of the sensory disturbance may be immediately after surgery (and the local anesthetic failed to "wear off"), or it may have begun several days after the surgical procedure. The former case most likely is from the surgery or local anesthetic injection, whereas the latter is usually related to postoperative swelling. On rare occasions, a fractured jaw may occur even weeks after a ramus block harvest or implant surgery, especially if the inferior border of the mandible was perforated. This is more likely in a patient with parafunction, and the flexure or torsion of the mandible may fracture the jaw through the unhealed implant site or block bone harvest, because the mandible is initially weakest at these sites. The presence and duration of the altered nerve sensations are noted at each patient evaluation, during the first month and every 4 weeks after the initial evaluation during the next few months. After 3 months, a decision should be made as to referral of the patient.

Box 31-6 Neurapraxia

1. Mild injury or retraction of nerve
   No violation of nerve trunk
2. No axonal degeneration
3. Pressure from retractor
   Hydraulic pressure
   Postoperative bleeding
   Traumatic soft tissue reflection
   Invasion of canal space during surgery
   Anesthetic needle
4. Management: corticosteroids
   Spontaneous recovery: 1 to 4 weeks
5. Most common

Classification of Nerve Injury

The goal of the patient examination is to determine the classification of nerve injury and therefore the chance of recovery. There are several nerve injury classifications, and each has particular advantages, especially for practitioners treating this disorder. The Seddon classification is user friendly for the occasional problem encountered by most dentists and will be presented in this text.[83]

Three types of nerve injury are described in the Seddon classification: neurapraxia, axonotmesis, and neurotmesis. Neurapraxia (Box 31-6) is a mild injury caused by compression injury to the nerve or retraction of the nerve. Examples of compression injuries include: pressure from a retractor over the mental foramen, hydraulic pressure from saline or blood from the implant site while the implant was threaded into position, postoperative bleeding within the bone or around the mental foramen, an implant inserted into the mandibular canal, a piece of bone that invaded the canal during the site preparation or implant insertion, or even a tie-back suture on the facial or lingual flap. Retraction injury of a nerve is usually from reflection of

WWW.HIGHDENT.IR

the soft tissue flap and the use of a retractor to maintain visibility or protect the flap from trauma during the use of rotary drills.

In neurapraxia, there is no axonal degeneration distal to the point of nerve injury, but there is a temporary conduction block while nerve recovery occurs. Spontaneous recovery of the altered sensations most often occurs within 1 to 4 weeks from this injury type. Fortunately this is the most common type of nerve injury observed, when there is no obvious violation of the nerve trunk.

When the patient is seen within 2 days of surgery with symptoms of nerve injury, an oral dose of corticosteroid (i.e., Decadron 8 mg) decreases inflammation and swelling in the region. If, during surgery, the nerve trunk is compressed or retracted beyond usual protocol, the intravenous form of corticosteroid (i.e., 1 to 2 mL of Decadron 4 mg/mL may be applied (not injected) to the injured area for 1 to 2 minutes. This direct application will decrease the risk of the formation of Nissl body disintegration, which causes the paresthesia.[84]

Axonotmesis describes a nerve injury with a loss of axonal continuity; however, the general structure of the nerve remains intact (the endoneurium is preserved) (Box 31-7). These injuries are more significant and may result in dysesthesia or less than normal nerve recovery. A stretch nerve injury from reflection of a soft tissue flap may result in this type of disorder. A nerve has a 20% elongation for the elastic limit to remain normal and 30% elongation causes structural failure. When the nerve complex is small in diameter as it exits the mental foramen, this injury is more prevalent. A slower stretching of the nerve as the flap is retracted is better tolerated by the axons of the nerve trunk. An implant drill may proceed through the top of the neurovascular canal and also cause this condition. When a postoperative radiograph reveals an implant may have slightly violated the canal space, it is prudent to unthread the implant and introduce Decadron 4 mg/mL into the osteotomy. Corticosteroids have been used with success in neurosurgery after trauma to a nerve. The decrease in inflammation increases the rate of recovery. No infection risks have been demonstrated with this protocol. After a 2- to 3-minute wait, it is prudent to place a shorter implant into the site. In addition, a corticosteroid (Decadron) is given orally for 3 to 5 days. The usual dosage is 8 to 12 mg in the morning of the first day, 4 to 6 mg the morning of the second day, and 2 to 3 mg the morning of the third day. Initial signs or symptoms of nerve recovery in this type of injury do not occur until 1 to 3 months after the injury. This injury type has symptoms and signs of nerve injury immediately after the surgery and is rarely due to swelling of the soft tissue, unless excessive.

It is possible that an anesthetic needle which penetrates the nerve trunk may cause this type of injury, although more often, neuropraxis results. Injury to the lingual nerve during inspection is more frequent than is the alveolar nerve. Women seem to be more frequently affected than men, in a 2:1 ratio. When the lingual nerve is affected, 80% of patients have an altered gustatory perception and the vast majority did not show improvement 9 to 19 months after the injury. Because the lingual nerve is smaller in diameter than the IAN, the penetration with a needle may cause more damage. In 1995, Haas et al. reported the risk of paresthesia after local anesthesia administration was 1:783,000 cases. The risk was slightly higher for drugs in greater concentration (i.e., septocaine 4% versus lidocaine 2%). An injection of anesthetic fluid into the nerve trunk may cause axonotmesis.[85,86]

Neurotmesis describes nerve injury a complete severance of the nerve trunk. When this occurs, all axons distal to the injury undergo Wallerian degeneration; anesthesia of the soft tissue that is innervated by the affected nerve is a consequence of this condition. When a discontinuity or gap is present between the nerve ends, scar tissue forms between the structures and axonal sprouts from the proximal aspect of the nerve are prevented from penetrating the endoneurial tubules (Box 31-8). Therefore no recovery of nerve sensations is expected without neurosurgical procedures. Neurotmesis is suspected when anesthesia is present or hypesthesia has lasted more than 3 months. A triggering sign that gives an electrical sensation from the injured site with palpation is suspect. A 200% increase in sensation to sharp stimuli 3 months after injury may also be a sign of neurotmesis. Fortunately most nerve injuries are not of this category, and recovery within 3 to 4 months is expected in most patients.

Box 31-7 Axonotmesis

1. Nerve injury with loss of axon continuity, but general structure intact
2. Wallerian degeneration
3. "Stretch" nerve injury from reflection
   Implant drill to top of canal
   Implant violating the canal
   Anesthetic needle
4. Management: remove offending element
   Corticosteroids
   Recovery:1 to 3 months

Box 31-8 Neurotmesis

1. Complete severance of nerve trunk
2. Wallerian degeneration
3. Complete anesthesia for more than 3 months; may have triggering signs or increase in sensation to sharp stimuli

WWW.HIGHDENT.IR

The lingual nerve may be severed during the reflection or incision to soft tissue lingual to the retromolar pad.[87] The usual location of this structure is 2 ± 1 mm medial to the lingual plate and 3 ± 0.4 mm below the crest of the lingual plate. However, according to Behnia et al., in 14% of mandibles the lingual nerve was in the soft tissue above the lingual crest of the mandible, and in 0.15% the nerve was actually residing in the retromolar pad. Therefore all incisions in the posterior mandible should be lateral to the retromolar pad. A careful reflection of the periosteum and gentle retraction of the lingual tissue, when necessary, is warranted to maintain the integrity of the lingual nerve within the lingual flap of tissue.

Lingual nerve damage is reported more often than IAN damage in third molar extraction reports when the tooth is removed through the lingual plate. However, in the usual surgical approach, the incidence of IAN injury is 2% to 7%, whereas lingual nerve involvement is less than 1% of impacted third molar extractions. In implant dentistry, the more common complication of altered nerve sensation is related to the inferior alveolar nerve.

According to Girard et al., the inferior alveolar nerve may have the potential for recovery after an injury for as long as 2 years.[88] On the other hand, Sunderland estimates 75% to 90% of the distal nerve atrophies and is irreparable after 1 year of altered feeling.[89]

Rather than waiting and decreasing the chance microsurgery may correct or improve the condition,[90] referral to a neurosurgical repair team is suggested when (1) transection or complete compression of the nerve is observed at surgery or on a CT scan, (2) painful dysesthesia exists, (3) anesthesia or unacceptable hypoesthesia from a patient's perspective has lasted for 3 months, and (4) triggering or radiating pain over the suspected site of injury is present.

It should be noted that a panoramic or periapical radiograph may not be diagnostic for an implant impingement on the mandibular canal. The implant may be buccal or lingual to the nerve location. A CT scan is warranted before diagnosis of nerve encroachment.

Life-Threatening Hemorrhage

The surgical insertion of dental implants has been shown to be a relatively safe procedure. However, since 1984 there have been at least 11 case reports of life-threatening hemorrhagic episodes as a consequence of placing implants in the mandible.[59,91-102] Although it may be considered rare and most dentists will never observe such a significant complication, it is prudent to be aware the risk exists and knowledgeable about treatment.

The cause of life-threatening hemorrhage is from significant internal bleeding in the floor of the mouth, usually caused by a perforation of the lingual cortical plate and a related swelling of the floor of the mouth and tongue, which causes respiratory obstruction (Figure 31-53). The significant hemorrhage usually begins during the surgery related to insult to the blood vessels, but may even occur 1 to 6 hours after the initial bleeding has been arrested. The hematomas that form in the lingual, sublingual, submandibular, and submental spaces displace or enlarge the tongue and the floor of the mouth, which may completely obstruct the airway. This complication has been reported secondary to several surgical procedures, including tooth extraction, local lingual anesthesia, use of sharp rotary instrument, periodontal procedures, and vestibuloplasties.

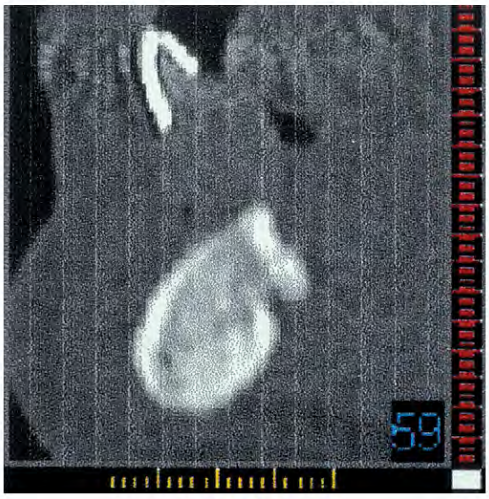


Figure 31-53 Lingual hemorrhage may be the result of implant osteotomy preparation and perforation of the lingual cortex in the anterior mandible. Careful evaluation of the symphyseal anatomy may avoid this serious complication.

There are primarily two major arteries that supply the floor of the mouth and are related to life-threatening hemorrhage: the lingual artery and the facial artery. It is important to determine the original source of the bleeding to establish a method to control the situation.

The lingual artery is usually the second anterior branch (third overall) of the external carotid artery, which crosses over the hyoid bone and is the major vessel to the tongue. This is important to know, because when the bleeding is suspected from this source, pulling out the tongue compresses the lingual artery against the hyoid bone and decreases the flow of blood to this vessel.

The lingual artery also has a significant branch, 2 mm in diameter, called the *sublingual artery*. It supplies blood to the lingual gingiva and the lingual aspect of the anterior cortical plate of the mandible, as well as other structures. Smaller branches of this vessel enter the lingual cortical plate through the lingual foramina. Typically, the largest foramina is found in the midline of the lingual cortical plate, just below the geniotubercles, with a mean distance of 6 mm from the inferior border of the mandible; however, accessory foramina in the premolar regions are also often observed.

WWW.HIGHDENT.IR

The facial artery is usually the third anterior branch (fourth overall) of the external carotid artery. This artery loops under the bottom of the mandible (in the region of the second molar), then laterally near the anterior border of the masseter muscle (antegonial notch) to supply portions of the face. The submental artery (also 2 mm in diameter) branches from the facial artery just before it crosses over the inferior border and courses along the interior and inferior aspect of the mandible. If this artery is suspected in the hemorrhage event, pressure against the interior and lingual aspect of the mandibular notch, just in front of the masseter muscle attachment, will significantly reduce the blood flow to this vessel. Bavitz et al. observed the sublingual artery was absent or small and unimportant in 53% of mandibles.[37] However, it has also been reported to be a significant or principal source of blood supply to the floor of the mouth in 29% to 59% of cases.[37,93,94]

The sublingual fossa on the lingual aspect of the mandible extends above the mylohyoid muscle attachment from the lateral incisor to the first premolar region, and is highly variable in depth from one patient to another.

In addition, the anterior mandible is often angled to the lingual, after the initial labial resorption of the bone or, even more often, after resorption of the residual ridge in a C–h mandible. An attempt to place implants in the long axis of the missing teeth often results in a lingual perforation of the mandible, especially in the canine region. In a review of the literature by Kalpidis and Setayesh, the most common site for life-threatening hemorrhage was during implant osteotomy or insertion in the canine position after the perforation of the lingual plate. Other causes included a tear in the lingual periosteum during elevation or handling of the flap.[102]

The signs or symptoms of life-threatening hemorrhage of the floor of the mouth include: (1) swelling and elevation of the floor of the mouth; (2) an increase in tongue size, which may even protrude from the mouth; (3) difficulty in swallowing or speech; and (4) pulsating or profuse bleeding from the osteotomy site or floor of the mouth.

The suggested methods to treat life-threatening hemorrhage, when suspected, may include:

1. Bimanual compression, with one finger intraorally over the site and one finger extraorally, compressing the two fingers together.
2. Pull out the tongue and observe whether a decrease in bleeding occurs (lingual artery blood flow reduced). Place deep pressure along the inner and inferior aspect of the body of the mandible at the antegonial notch region and observe whether the bleeding is reduced (facial artery blood flow reduced).
3. Elevate the head. This may decrease blood flow to the region by almost 30%.
4. Place an oropharyngeal airway behind the tongue before it enlarges and makes the insertion of this device almost impossible.
5. Place hemostatic agents in the osteotomy on or in the lingual periosteal tissues.
6. Push with firm pressure against the transverse process of the fourth cervical vertebra in the neck, on the side of the bleeding. (This reduces the blood flow to the external carotid artery.)
7. Transport the patient to the hospital if these attempts to halt the signs and symptoms of life-threatening hemorrhage are not received or if these problems develop after several hours. Do not hesitate to transport the patient to the hospital. NOTE: If complete airway obstruction prevents a patient from breathing, an emergency cricothyroidotomy may be a last resort until intubation may be performed.

Procedures in the hospital to treat this condition have included: (1) ligation of the afflicted vessels, (2) endotracheal intubation (oral or nasal), or (3) emergency tracheotomy. The duration of hospitalization in the cases reported in the literature has ranged from 1 to 11 days.

Although less reported in the literature, there are cases in which the site of life-threatening hemorrhage is the preparation of an implant site in the second molar region. The perforation of the lingual plate occurs because of the significant undercut under the mylohyoid muscle. In addition to the sublingual fossa, a submandibular fossa, which is of more significance, is found below the mylohyoid muscle attachment in the first molar to the third molar region. This facial artery and its lateral loop are found in this depression. The residual bone may be wide enough for an implant, but the angulation of the implant for proper prosthetic abutment support places the implant directly over the submandibular fossa and facial artery. When the dentist drills into the bone, the dense lingual plate is encountered. A firm pressure on the drill may result in penetration of the lingual plate, and because a firm pressure is required for the osteotomy, after it penetrates, the drill proceeds quickly several millimeters beyond the lingual cortex. When an artery is partially cut, rather than constricting and forming a platelet plug, the bleeding may continue and fill the submandibular space. A bulging area may be observed below the mandible extraorally because it is below the mylohyoid muscle. Pressure on the antegonial notch is not effective in this situation, because the artery laceration is before the bony landmark.

Pressure against the transverse process of the fourth vertebra and elevation of the head to reduce blood flow are primary methods to reduce blood flow from the site. Firm external pressure to the posterior floor and intraoral pressure with a finger over the site is also of benefit. If the borders of the surrounding region continue

WWW.HIGHDENT.IR

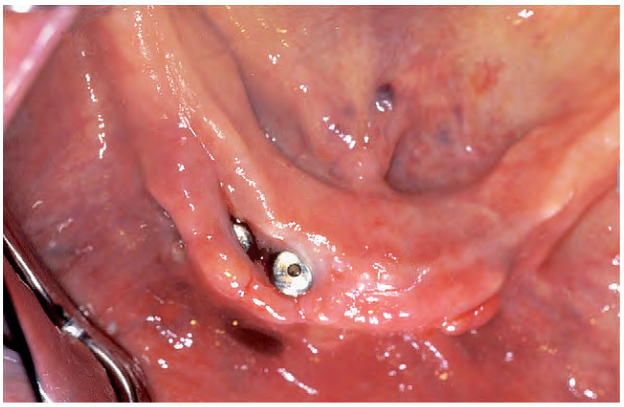


Figure 31-54 Implant premature exposure should not be resutured again to approximate the tissues. Instead, the implant should be completely uncovered and the permucosal extension inserted.

to expand, a nasotracheal or orotracheal airway and transportation to the hospital are appropriate.

Prevention is the key to handling life-threatening hemorrhage. The awareness of bone undercuts may be appreciated by a CT scan and reformatted images or by palpation of the lingual plate of the mandible at the time of surgery. A *stop* on the dental drill at the desired depth, so it does not penetrate several millimeters after the lingual plate is perforated, is also of benefit A shorter implant that reduces the need to engage the opposing cortical plate is often appropriate.

Incision Line Opening

The most common postoperative complication is incision line opening. If a minor opening in the incision line is observed, the cause of the incision opening should be determined. If the design of the removable interim prosthesis is involved, it is corrected. The patient is instructed to rinse three to three times daily with chlorhexidine and gently debride the incision line with a moist sponge or soft toothbrush. Within a few days to weeks, the soft tissue will granulate into the opening without compromise to the implants. Resuturing the area should not be attempted because it would further increase the problem.

If the granulation process to close the incision line extends more than 2 weeks, it may be encouraged by trimming the epithelial margins of the wound with a high-speed hand piece and coarse diamonds. This will cause minor bleeding and allow the granulation process to continue. This minor debridement is generally performed with only topical anesthetic.

Implant Exposure

If the implants become exposed during the healing period of a two-stage surgery, no attempt should be made to cover them with tissue. Implant exposure alone does not alter the success of rigid fixation. The soft tissue over the top of the implant has little to do with the bone forming on the surface of the implant (Figure 31-54). However, if the cause of the exposure was pressure necrosis of the soft tissue over the implant from the prosthesis, this is a problem that may cause micromovement of the implant. Therefore, whenever an implant becomes exposed through the tissue, external forces over the implant should be eliminated. The denture is relieved aggressively over the area with implant exposure.

Barboza et al.[103] and Tal et al.[104] have observed that partial exposure of a dental implant is more detrimental than complete exposure. Pinpoint to three-quarter exposures of the implant are more often associated with exudate or chronic inflammatory tissue around the crestal region and marginal bone loss. The incidence of anaerobic bacteria also increases. As a consequence, it is highly recommended the partial exposure of the implant not be ignored or treated lightly.

The protocol of partial exposure, unassociated with exudate, includes the following: (1) complete exposure of the implant cover screw, removal of the healing cover, flushing the implant with chlorhexidine and insertion of a permucosal extension (PME); (2) oral hygiene with a soft toothbrush morning and evening; and (3) chlorhexidine application over the area twice each day.

An implant with a partial exposure or exudate requires more aggressive therapy. The implant should be completely uncovered. The cover screw should be removed and the implant irrigated with chlorhexidine. In addition, the amount of marginal bone loss should be ascertained. Reflection of the tissue and exposure of the implant is in order. The granulation tissue should be curetted from the surrounding bone. The implant surface area may be mechanically cleaned with a diamond bur or air abrasion with baking soda and the implant surface may be treated with tetracycline or citric acid. A bone graft and a membrane may also be necessary when the bone loss is several millimeters. When a bone graft is placed, the tissue should completely cover the membrane.

In regions of limited bone loss, a PME may be inserted and the tissue approximated around the extension. When a membrane is used, usually primary closure is in order to increase the success rate. Antibiotics for 5 days and daily rinses of chlorhexidine are also indicated. The same regimen is implemented for nonsubmerged or a one-stage healing protocol.

Implant Failure

Mobility of the implant during healing is unusual but may occur. This complication is rarely accompanied by pain or infection, but it is often accompanied by a radiolucent zone around the implant. Whatever the cause (bone necrosis, implant movement, or infection), the implant should be removed, followed

WWW.HIGHDENT.IR

#1 Cone Socket Mirror Handle	Kelly-Rankin Hemostat 6.5 inch
#5 Mouth Mirror #6 Satin Steel Handle	#2 Kelly Hemostat-Curved
#17 Dressing Pliers	#18 Iris Scissors, Curved
#2 DE Explorer, Satin Steel	#13 Suture Scissors
#12 Colorvue PTS 3-6-9-12, 12	Cottle Chisel Cvd 5 mm
#11-1/2 SE Root Canal Plugger	Beyer Rongeurs
#43 Adson-Brown Tissue Pliers	#2 Mead Mallet-Fiber Facings
Brown Tissue Forceps 9 × 9T 6”	Columbia Cheek Retractor (2)
Allison Tissue Forceps	Markel Mouth Prop (Adult)
Anesthetic Syringe (2)	Markel Mouth Prop (Child)
#3 Scalpel Handle (2)	3-0 Violet PGA Suture 27”,C-9 Needle 23 mm,
#5 Straight Round Scalpel Handle	1/2 Circle Reverse Cut
#2/4 Molt DE Curette	4-0 Violet PGA Suture 18, C-6 Needle
#4 Molt Curette	18.7mm, 3/8 Circle Reverse Cut
#6 Potts Elevator	Colorvue Probe Handle #6
#7 Potts Elevator	#4 Mott Surgical Curette-Right
Buser Periosteal Elevator	#4 Mott Surgical Curette-Left
#23 Seldin Periosteal (2)	Sinus Retractor Misch 10 mm
#87 Lucas DE Surgical Curette	Sinus Retractor Misch 15 mm
#U-15/30 DE Scale, Satin	Surgical Trays with clips (2)
#99C Kells Forceps	Misch Periosteal, right & left
#21 Miller Bone File	Complete Misch Surgery Kit
Crille-Wood Perma Sharp Needle Holder	

Figure 31-55 Surgical tray and basic armamentarium

8 to 10 weeks later by a graft with autogenous or bone substitutes, and allowed to regenerate bone before the next implant placement. On rare occasion, an early implant failure may be replaced with a larger-diameter implant. This is contraindicated in the presence of infection.

SUMMARY

Root form implants placed in an anterior mandible are the most common indication of the completely edentulous patient. It is an excellent region to begin a learning curve in implant surgery because the landmarks are clear, access is good, and bone density often permits more flexibility in technique. The step-by-step approach described in this chapter allows excellent results for implant-supported prostheses. The placement of root form implants in the posterior mandible is also a common procedure in the partially edentulous patient and is described with a clinical technique to assess the zone of safety after the placement of implants safely above the mandibular canal. Figure 31-55 illustrates the surgical tray and basic armamentarium.

References

1. Lill W, Lembacher J, Schuster G et al: Experimentelle untersuchungen zur kongruenz und passgenauigkeit von implantat und implantatbett. Eine in-vitro-studie, *Z Zahnarztl Implantol* 6:240-246, 1990.
2. Brunski JB: The influence of functional use of endosseous dental implants on the tissue-implant interface: clinical aspects, *J Dent Res* 58:1953, 1979.
3. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 6:387-416, 1981.
4. Misch CE: Implant overdenture relieves discomfort for the edentulous, *Dentist* 37-39, Jan/Feb 1989.
5. Malamed SF: *Handbook of local anesthesia*, ed 3, St Louis, 1990, Mosby.
6. Akinosi JO: A new approach to the mandibular nerve block, *Br J Oral Surg* 15:83-87, 1977.
7. Brånemark PI, Zarb GA, Albrektsson T: *Tissue-integrated prostheses, osseointegration in clinical dentistry*, Chicago, 1985, Quintessence.
8. Friberg B: Sterile operating conditions for the placement of intraoral implants, *J Oral Maxillofac Surg* 54:1334-1336, 1996.
9. Kraut RA: Clean operating conditions for the placement of intraoral implants, *J Oral Maxillofac Surg* 54:1337-1338, 1996.

WWW.HIGHDENT.IR

10. Peterson LJ: Microbiology of the head and neck, *Oral Maxillofac Surg Clin North Am* 3:247, 1991.
11. Pogrel MA: Infection management, *Oral Maxillofac Surg Clin North Am* 5:127, 1993.
12. Scharf DR, Tarnow DP: Success rates of osseointegration for implants placed under sterile versus clean conditions, *J Periodontol* 64:954-956, 1993.
13. Gristina AG: Biomaterial centered infection, microbial adhesion versus tissue integration, *Science* 237:1588, 1987.
14. Gristina AG, Naylor P, Myrvik CL: Infections from biomaterials and implants: a race for the surface, *Med Prog Technol* 14:205, 1988.
15. Gristina AG: Implant failure and the immuno-incompetent libra inflammatory zone, *Clin Orthop* 298:106, 1994.
16. Dahlen G: Effect of antimicrobial mouth rinses on salivary microflora in healthy subjects, *Scand J Dent Res* 92:38, 1994.
17. Lambert PM, Morris HF, Ochi S: The influence of 0.12% chlorhexidine digluconate rinses on the incidence of infectious complications and implant success, *J Oral Maxillofac Surg* 55(suppl 5):25-30, 1997.
18. de Albuquerque RF Jr, Head TW, Mian H et al: Reduction of salivary *S. aureus* and mutans group streptococci by a preprocedural chlorhexidine rinse and maximal inhibitory dilutions of chlorhexidine and cetylpyridinium, *Quintessence Int* 35:635-60, 2004.
19. Arata T, Murukami T, Hirai Y: Evaluation of povidone iodine alcoholic solution for operative site disinfection, *Postgrad Med J* 69(suppl 3):S93-S96, 1993.
20. Peterson LJ: Antibiotic prophylaxis against wound infections in oral and maxillofacial surgery, *J Oral Maxillofac Surg* 48:617-620, 1990.
21. Dent CD, Olsor JW, Farish SE et al: The influence of preoperative antibiotics on success of endosseous implants up to and including stage II surgery: a study of 2641 implants, *J Oral Maxillofac Surg* 55(suppl 5):16-24, 1997.
22. Laskin DM, Dent CD, Morris HF et al: The influence of preoperative antibiotics on success of endosseous implants at 36 months, *Ann Periodontol* 5:166-174, 2000.
23. Timmenga N, Raghoebar GM, Boering G et al: Maxillary sinus infection after sinus lifts for the insertion of dental implants, *J Oral Maxillofac Surg* 55:936-939, 1997.
24. Barone A, Santini S, Sbordone L et al: A clinical study of the outcomes and complications associated with maxillary sinus augmentation, *Int J Oral Maxillofac Implants* 21:81-85, 2006.
25. Kleinheinz J, Buchter A, Kruse-Losier B et al: Incision design in implant dentistry based on vascularization of the mucosa, *Clin Oral Implants Res* 16:518-523, 2005.
26. Roberts EW: Bone physiology and metabolism, *J Calif Dent Assoc* 15:54-61, 1987.
27. Cutright B, Quillopa N, Schubert W: An anthropometric analysis of the key foramina for maxillofacial surgery, *J Oral Maxillofac Surg* 61:354-357, 2003.
28. Greenstein G, Marnow D: The mental foramen and nerve: clinical and anatomical factors related to dental implant placement: a literature review, *J Periodontol* 77:1933-1943, 2006.
29. Fishel D, Buchner A, Hershkowith A et al: Roentgenologic study of the mental foramen, *Oral Surg Med Oral Pathol* 41:682-686, 1976.
30. Wang, TM, Shih C, Liu JC et al: A clinical and anatomical study of the location of the mental foramen in adult Chinese mandibles, *Acta Anat Basel* 126:22-29, 1986.
31. Bergman RA, Afifi AK, Miyauchi R: Mental foramen. In *Illustrated encyclopedia of human anatomic variation: opus V: skeletal systems.* Available at http://www Anatomy atlases.org/AnatomicVariants/SkelatalSystem/Images/127.shtml. Accessed June 21, 2006.
32. Misch CE, Crawford E: Predictable mandibular nerve location—a clinical zone of safety, *Int J Oral Implant* 7:37-40, 1990.
33. Rosenquist B: Is there an anterior loop of the inferior alveolar nerve, *Int J Perio Rest Dent* 16:40-45, 1996.
34. Arouzman MJ, Otis L, Kipnis V: Observations of the anterior loop of the inferior alveolar canal, *Int J Oral Maxillofac Implants* 8:295, 1993.
35. Solar P, Ulm C, Frey G et al: A classification of the intraosseous paths of the mental nerve, *Int J Oral Maxillofac Implants* 9:339-344, 1994.
36. Neiva RF, Gapski R, Wang HL: Morphometric analysis of implant-related anatomy in Caucasian skulls, *J Periodontol* 75:1061-1067, 2004.
37. Bavitz JB, Harn SD, Hansen CA et al: An anatomical study of mental neurvovascular bundle-implant relationships, *Int J Oral Maxillofac Implants* 8:563-567, 1993.
38. Kuzmanovic DV, Payne AG, Kieser JA et al : Anterior loop of the mental nerve: a morphological and radiographic study, *Clin Oral Implants Res* 14:464-471, 2003.
39. Mardinger O, Chaushu G, Arensburg B et al: Anterior loop of the mental canal: an anatomical-radiologic study, *Implant Dent* 9:120-125, 2000.
40. Eriksson RA, Albrektsson T: Temperature threshold levels for heat-induced bone tissue injury: a vital-microscopic study in the rabbit, *J Prosthet Dent* 50:101-107, 1983.
41. Yacker M, Klein M: The effect of irrigation on osteotomy depth and bur diameter, *Int J Oral Maxillofac Implants* 11:635-638, 1996.
42. Schroeder A: Preparation of the implant bed. In Schroeder A, Sutter F, Buser D et al, editors: *Oral implantology,* New York, 1996, Thieme.
43. Marx RE: Principles of hard and soft tissue reconstruction of the jaws, abstract ML 315, American Association of Oral and Maxillofacial Surgeons, New Orleans, 1990.
44. Misch CE, Strong T: Bone temperature and its effect on drill size and sequence: a pilot study (personal communication), 2003.
45. Rafel SS: Temperature changes during high-speed drilling on bone, *J Oral Surg Anesth Hosp Dent Serv* 20:475-477, 1962.
46. Babbush CA: The endosteal blade vent implant—the histology of animal studies and scanning election microscope observations. In Babbush CA, editor: *Surgical atlas of dental implant techniques,* Philadelphia, 1980, WB Saunders.
47. Matthews J, Hirsh C: Temperatures measured in human cortical bone when drilling, *J Bone Joint Surg* 45A:297-308, 1972.
48. Hobkirk J, Rusiniak K: Investigation of variable factors in drilling bone, *J Oral Surg* 35:968-973, 1977.
49. Brisman DL: The effect of speed, pressure, and time on bone temperature during the drilling of implant sites, *Int J Oral Maxillofac Implants* 11:35-37, 1996.
50. Lavelle CH, Wedgewood D: Effect of internal irrigations on frictional heat generated from bone drilling, *J Oral Surg* 38:499, 1980.
51. Sharawy M, Misch CE, Weller N et al: Heat generation during implant drilling: the significance of motor speed, *J Oral Maxillofac Surg* 60:1160-1169, 2002.

WWW.HIGHDENT.IR

52. Eriksson R, Adell R: Temperatures during drilling for the placement of implants using the osseointegration technique, *J Oral Maxillofac Surg* 44:4-7, 1986.
53. Misch CE, Hoar JB, Beck G et al: A bone quality based implant system: a preliminary report of stage I and stage II, *Implant Dent* 7:35-42, 1998.
54. Watzek G, Haider R, Gitsch M et al: *Influence of drill cooling and bone structure on primary implant fixation* [abstract], Boston, 1991, American Academy of Osseointegration.
55. Haider R, Watzek G, Plenk H Jr: Influences of drill cooling and bone structure on primary implant fixation, *Int J Oral Maxillofac Implants* 8:83-91, 1993.
56. Ten BruggenKate CM, Krekeler G, Kraaijenhagen HA et al: Hemorrhage of the floor of the mouth resulting from lingual perforation during implant placement, *Int J Oral Maxillofac Implants* 8:329-334, 1993.
57. Laboda G: Life threatening hemorrhage after placement of an endosseous implant. Report of a case, *J Am Dent Assoc* 121:559-600, 1990.
58. Mordenfeld A, Andersson L, Bergstrom B: Hemorrhage in the floor of the mouth during implant placement in the edentulous mandible, a case report, *Int J Oral Maxillofac Implants* 12:558-661, 1997.
59. Kalpidis CE, Konstantinidis AB: Critical hemorrhage in the floor of the mouth during implant placement in the first mandibular premolar position: a case report, *Implant Dent* 14:117-124, 2005.
60. Watzek G: Preprosthetic and peri-implant surgery—general principles. In Watzek G, editor: *Endosseous implants: scientific and clinical aspects,* Chicago, 1996, Quintessence.
61. Buser D, Weber HP, Lang NP: Tissue integration of non submerged implants: one-year results of a prospective study with 100 ITI hollow cylinder and hollow screw implants, *Clin Oral Implant Res* 1:33-40, 1990.
62. Berglundh T, Lindhe J, Ericsson I et al: The soft tissue barrier at implants and teeth, *Clin Oral Implant Res* 2:81-90, 1991.
63. Misch CE: Bone density: its effect on surgery, healing and progressive bone loading, *Int J Oral Implant* 6:23-31, 1990.
64. Watzek G, Danhel-Mayhauser M, Matejka M et al: Experimental comparison of Brånemark and TPS dental implants in sheep. Abstracts of the UCLA Symposium: Implants in the partially edentulous patient, 50. Palm Springs, Calif, 1990.
65. Misch CE, Steigenga J, Barboza E et al: Short dental implants in posterior partial edentulism: a multicenter retrospective 6-year case series study, *J Periodontol* 77:1340-1347, 2006.
66. Agthong S, Huanmanop T, Chentanez V: Anatomical variations of the supraorbital, infraorbital and mental foramina related to gender and side, *J Oral Maxillofac Surg* 63:800-804, 2005.
67. Sawyer DR, Kiely ML, Pyle MA: The frequency of accessory mental foramina in four ethnic groups, *Arch Oral Biol* 43:417-420, 1998.
68. Jacobs R, Mraiwa N, van Steenberghe D et al: Appearance, location, course and morphology of the mandibular incisive canal: an assessment on spiral CT scan, *Dentomaxillofac Radiol* 31:322-327, 2002.
69. Yosue T, Brooks SL: The appearance of mental foramina on panoramic and periapical radiographs, *Oral Surg Oral Med Oral Pathol* 68:488-492, 1989.
70. Klinge B, Petersson A, Maly P: Location of the mandibular canal. Comparison of macroscopic findings conventional radiography and computed tomography, *Int J Oral Maxillofac Implants* 4:327-332, 1989.
71. Lindh C, Petersson A: Radiologic examination for location of the mandibular canal. A comparison between panoramic radiography and conventional tomography, *Int J Oral Maxillofac Implants* 4:249-256, 1989.
72. Sonick M, Abrhams MD, Faiella RA: A comparison of the accuracy of periapical, panoramic, and computerized tomographic radiographs in locating the mandibular canal, *Int J Oral Maxillofac Implants* 9:455-460, 1994.
73. Ritter EF, Moelleken BR, Mathes SJ et al: The course of inferior alveolar neurovascular canal in relation to sliding genioplasty, *J Craniofac Surg* 30:20-24, 1992.
74. Sanchis JM, Penarrocha M, Soler F: Bifid mandibular canal, *J Oral Maxillofac Surg* 61;422-424, 2003.
75. Hahn J: Personal communication, 1986.
76. Watzek G: Surgical procedures for implant installation depending on the degree of maxillo mandibular atrophy in endosseous implants. In Watzek G, editor: *Endosseous implants: scientific and clinical aspects,* Chicago, 1996, Quintessence.
77. Linkow L: *Mandibular implants: a dynamic approach to oral implantology,* New Haven, Conn, 1978, Glarus.
78. Stella JP, Tharanon W: A precise radiographic method to determine the location of the inferior alveolar canal in the posterior edentulous mandible: implications for dental implants. Part 2: clinical application, *Int J Oral Maxillofac Implants* 5:23-30, 1990.
79. *Osseotite certain implant system surgical manual,* Palm Beach Gardens, Fla, 2003, 3i.
80. Shramme AG: *Surgical manual ITI solid screw*, Waldenburg, Switzerland, 2007, ITT Solid Screw Implants Institute.
81. *Screwvent surgical guide*, Carlsbad, Calif, 2006, Zimmer Dental.
82. Strong T, Misch CE, Beck G: BioHorizons dental drill system and method of use, US Patent 6,863,529, issued 2002.
83. Seddon HJ: Nerve injuries, *Med Bull* 31:4-10, 1965.
84. Galloway EB 3rd, Jensen RL, Dailey AT et al: Role of topical steroids in reducing dysfunction after nerve injury, *Laryngoscope* 110:1907-1910, 2000.
85. Haas DA, Leenon D: A 21-year retrospective study of reports of paresthesia following local anesthetic administration, *Can Dent Assoc* 61:319-330, 1995.
86. Hillerup S, Jensen R: Nerve injury caused by mandibular block analgesia, *Int J Oral Maxillofac Surg* 35:437-443, 2006.
87. Behnia H, Kheradvar A, Shahrokhi M: An anatomic study of the lingual nerve in the third molar region, *J Oral Maxillofac Surg* 58:649-651, 2000.
88. Girard KR, Jacobs R, Mraiwa N et al: Considerations in the management of damage to the mandibular nerve, *J Am Dent Assoc* 98:65-71, 1979.
89. Sunderland S: The anatomy and physiology of nerve injury, *Muscle Nerve* 13:771-784, 1990.
90. Kraut RA, Chahal O: Management of patients with trigeminal nerve injuries after mandibular implant placement, *J Am Dent Assoc* 133:1351-1354, 2002.
91. Mraiwa N, Jacobs R, van Steenberghe D et al: Clinical assessment and surgical implications of anatomic challenges in the anterior mandible, *Clin Implant Dent Relat Res* 5:219-225, 2003.
92. Flanagan D: Important arterial supply of the mandible, control of an arterial hemorrhage and report of a hemorrhagic incident, *J Oral Implantol* 29:165-173, 2003.

WWW.HIGHDENT.IR

93. Tepper G, Hofschneider UB, Gahleitner A et al: Computed tomographic diagnosis and localization of bone canals in the mandibular interforaminal region for prevention of bleeding complications during implant surgery, *Int J Oral Maxillofac Implants* 16:68-72, 2001.
94. Hofschneider U, Tepper G. Gahleitner A et al: Assessment of blood supply to the mental region for reduction of bleeding complications during implant surgery in the interforaminal region, *Int J Oral Maxillofac Implants* 14: 379-383, 1999.
95. Mason ME, Triplett RG, Alfonso WF: Life-threatening hemorrhage from placement of a dental implant, *J Oral Maxillofac Surg* 48:201-204, 1990.
96. Isaacson TJ: Sublingual hematoma formation during immediate placement of mandibular endosseous implants, *J Am Dent Assoc* 135:168-172, 2004.
97. Burk RH, Masch GL: Lingual artery hemorrhage, *Oral Surg Oral Men Oral Pathol* 62:258-261, 1986.
98. Niamtu J III: Near-fatal airway obstruction after routine implant placement, *Oral Surg Oral Men Oral Pathol* 92:597-600, 2001.
99. Darriba MA, Mendonca-Caridad JJ: Profuse bleeding and life-threatening airway obstruction after placement of mandibular dental implants, *J Oral Maxillofac Surg* 55:1328-1330, 1997.
100. Givol N, Chaushu G, Halamish-Shani T et al: Emergency tracheostomy following life-threatening hemorrhage in the floor of the mouth during implant placement in the mandibular canine region, *J Periodontol* 71:1893-1895, 2000.
101. Woo BM, Al-Bustani S, Ueeck BA: Floor of the mouth hemorrhage and life-threatening airway obstruction during immediate implant placement in the anterior mandible, *Int J Oral Maxillofac Surg* 35:961-964, 2006.
102. Kalpidis CD, Setayesh AM: Hemorrhaging associated with endosseus implant placement in the anterior mandible: a review of the literature, *J Periodontol* 75:631-645, 2004.
103. Barboza EP, Caula AL: Diagnoses, clinical classification and proposed treatment of spontaneous early exposure of submerged implants, *Implant Dent* 11:331-337, 2002.
104. Tal H: Spontaneous early exposure of submerged implants: classification and clinical observations, *J Periodontol* 70: 213-219, 1999.

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Chapter 32

Stage II Surgery: Uncovery and Treatment of Healing Complications

Carl E. Misch

The loss of implants from initial surgery to prosthetic loading has gradually decreased over the last decade and is often less than 5%.[1,2] These failures may be explained to the patient as caused by local conditions or intraoral healing responses, and they are usually well accepted by the patient. Failures from stage II uncovery until prosthesis connection have also been identified and range from 2.5% to 5.9%.[1-6] These early loading or "late failures" are more of a problem because the restoring dentist has begun the restoration, accompanied by laboratory and chair time costs. The patient often blames the restoring dentist for this complication, relating it to some clinical event such as the final impression or removal of the transitional prosthesis.

NOMENCLATURE FOR IMPLANT FAILURES

With the development of many different implant systems and the advent of such techniques as single-stage surgery, immediate loading, and direct implant placement of the prosthesis, implant failure terminology can be confusing. A review of the literature shows inconsistency in its classification of implant failures relative to time. In particular, the terms *late failure* or *later failure* have not been precisely defined in regard to the timing of the failure. *Late failure* may be used to describe early loading failures after stage II uncovery or complications after several years to decades after implant loading.

Misch and Jividen have published a classification for implant failure, based on the unusual events of an implant history.[7] The suggested terminology of failures relative to time includes *surgical failure*, which may be used to describe the inability to place the implant at the time of surgery (e.g., from fracture of bone, failure to obtain initial rigid fixation)(Table 32-1).

Osseous healing failure may describe the period from implant placement to abutment connection of the implant and is related to the healing ability of bone. *Early loading failure* describes the period from the abutment placement to the first year the implant serves as a prosthetic abutment. When immediate loading is performed, the osseous healing period and early loading time frame are similar. Early implant failure is more likely caused by inadequate surface area of the implant for the magnitude of the load or quality of bone that is not sufficient to support the load. *Intermediate implant failure* is the period after the first year of loading and includes up to the subsequent 5 years of function. *Late implant failure* is used to describe the condition after the implant and prosthesis have been loaded for longer than 5 years and shorter than 10 years. *Long-term failure* may be used to describe failures after 10 years. Peri-implant disease and fatigue fracture are more common causes of long-term failures. This nomenclature is therefore proposed to facilitate future research efforts and interpretation.

The initial surgery and healing process ideally result in a rigidly fixated implant, absence of crestal bone loss around the implant, adequate zones of nonmobile keratinized tissue (>2 mm), soft tissue thickness less than 4 mm, and an absence of tenderness or discomfort under vertical or lateral forces (Box 32-1). A second-stage implant (uncovery) surgery permits the direct evaluation of these criteria and the corrections necessary to lay

Table 32-1 Implant Failure Terminology

TYPE OF FAILURE	TIMING
Surgical	At surgery
Osseous healing failure	Between stages I and II
Early loading	Abutment placement to first year
Intermediate	First-year loading to 5 years
Late	5 to 10 years
Long term	>10 years

WWW.HIGHDENT.IR

Box 32-1 Criteria for Evaluation of Successful Implants at Stage II Uncovery

Rigid fixation
Absence of crestal bone loss
Absence of pain
Adequate zone of keratinized gingiva
Sulcus depth 4 mm
Absence of inflammation
Proper hard and soft tissue contour
Prosthetic abutment allows implant loading under physiologic conditions

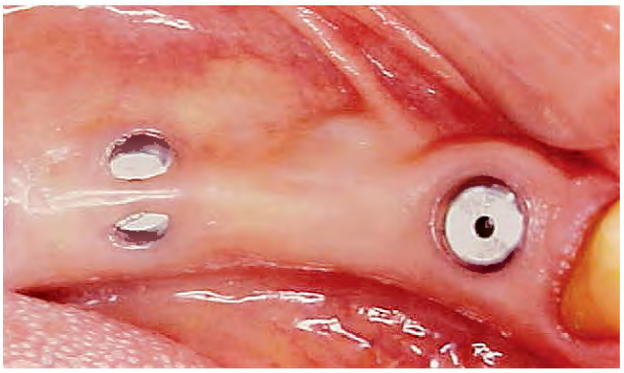


Figure 32-1 Spontaneous exposure of implants during the initial healing period occurs 5% to 15% of the time, depending on the hard and soft tissue conditions at the initial surgery.

the groundwork for long-term success. An opportunity exists at the stage II surgery to correct poor implant placement, inadequate crestal bone healing, soft tissue defects, or poor soft tissue relationships. The final restoration should not be placed in long-term jeopardy at the expense of compromised initial healing conditions.

A second-stage surgery most often is used by the profession for an implant that is completely covered by soft tissue during the initial healing period. However, even when the implant was inserted with a one-stage surgical approach or became exposed during the healing process, a second-stage surgery may be of benefit to evaluate the surrounding bone and soft tissue conditions.

The goal of a two-stage surgical approach, as presented by Brånemark in 1977, is to have the implant covered with soft tissue for 3 to 6 months while the bone remodels around the implant.[8] The primary reasons cited were to reduce the risk of infection and prevent the apical migration of tissue around the implant. In addition, the implant was countersunk below the bone to reduce the risk of loading or movement during initial healing. If the implant became prematurely exposed during the first 6 weeks, the soft tissue would be surgically repositioned to recover the implant once again.

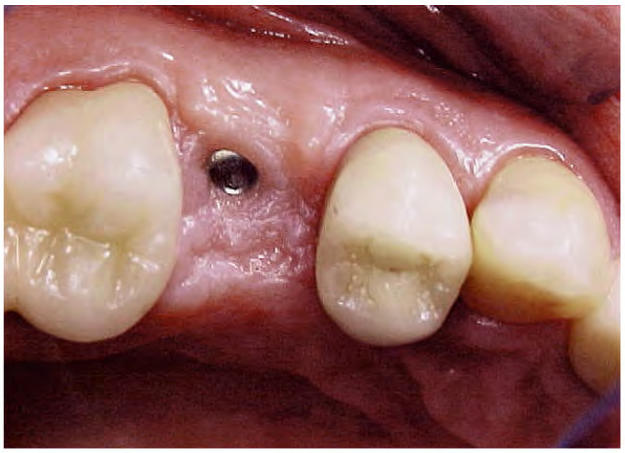


Figure 32-2 The epithelium over the implants of a spontaneous exposure, on occasion may be associated with chronic inflammatory cells.

SPONTANEOUS EARLY EXPOSURE

The implant covered with soft tissue may spontaneously become partially or completely exposed prior to stage II uncovery (Figure 32-1). The spontaneous early exposure of submerged implants has been found, on occasion, to be accompanied by early exudate, inflammation, and crestal bone loss. In a 15-year report by Adell et al., 4.6% of 2768 implants presented early perforations.[9] These implants had an external hex and were countersunk, level with the bone.

When Rosenquist and Grenthe[10] placed implants into extraction sockets, 11% of the implants had early exposures. Tal et al.[11-14] observed early exposure in 16.1% of external hex implants and 11% with internal hex implants. Therefore it appears the risks of early exposure occurs approximately 5% to 15% of the time and is dependent on the profile of the implant at surgery and the hard and soft tissue conditions.

When the implant has an early exposure, the epithelium may become hyperplastic, with cells exhibiting hyperparakeratosis and acanthosis (Figures 32-2, 32-3). The connective tissue may also be infiltrated by chronic inflammatory cells, mainly lymphocytes and plasma cells.[13] Toljanic reported 3.9 times more bone loss in premature exposures, with a mean bone loss of 2.71 mm (standard deviation [SD] ±1.78 mm) for prematurely exposed implants versus 0.43 mm (SD ±1.08 mm) for nonexposed implants. It may be argued that the exposure of the implant triggers the "biological width" phenomenon, which would occur regardless after stage II uncovery. In 1996, Ericsson et al. found in a dog study the amount of marginal bone loss initially was similar, but after 6 months of exposure, both the one-stage and two-stage implant bone levels were similar (Figures 32-4, 32-5).[16]

WWW.HIGHDENT.IR

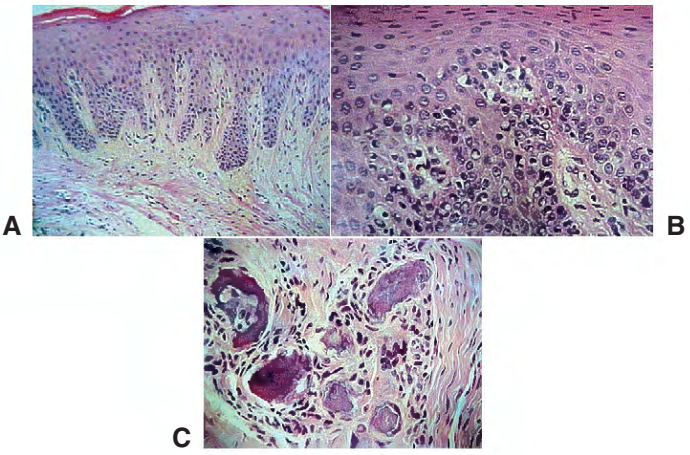


Figure 32-3 Histologic evaluation of the patient in Figure 32-2 **(A)** revealed connective tissue with infiltrated chronic inflammatory cells **(B,C).**

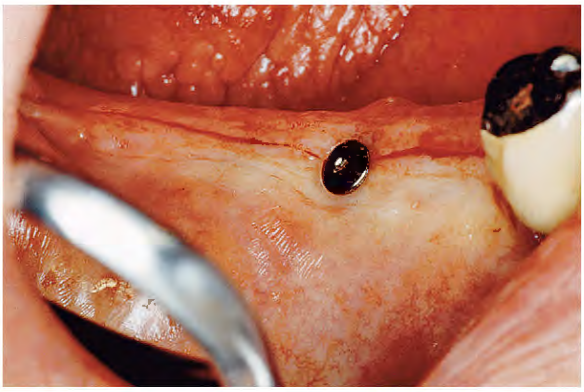


Figure 32-4 Spontaneous exposure during healing may occur to only one implant of multiple sites.

Tal et al. evaluated 115 early exposures of submerged endosteal implants.[14] Of this group, 11% had significant bone loss, with 2 implants >4 mm; 2 implants, 3 to 4 mm; and 10 implants, 2 mm of bone loss. Although most often premature exposure was not a complication, on occasion the condition may lead to significant bone loss. Barboza et al. compared crestal bone loss for one-stage surgery versus a two-stage surgery in 10 patients with 56 implants.[17] She found more crestal bone loss around exposed implants and also found a higher incidence of *Prevotella intermedia*, *Streptococcus* beta hemolysin, and *Fusobacterium* sp. The exposure of the implant cover screw seems to create a potential focus for bacterial plaque which may contribute to crestal bone loss (Figures 32-6, 32-7). Therefore it is indicated to monitor patients from stage I surgery to stage II surgery every 4 to 8 weeks to access and treat appropriately premature exposure of implants.

It has been observed by several authors that the risk of exudate or inflammation is increased when the implant is only partially exposed (Figure 32-8).[11-16] The

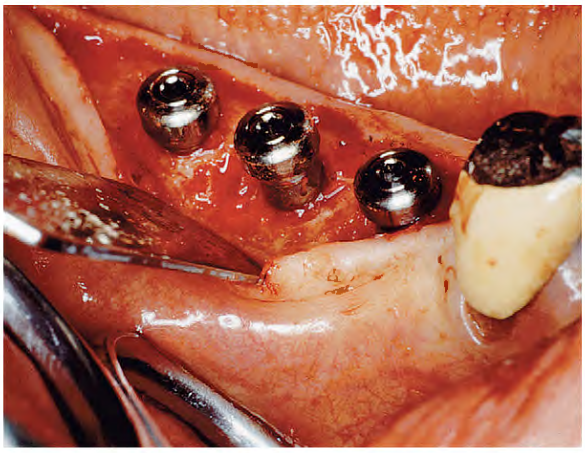


Figure 32-5 At the uncovery, the implant with premature exposure has more crestal bone loss than the implants completely covered during initial healing. This initial difference may be related to the "biological width" after the implant becomes exposed. Most often the overall crestal bone is similar during the next 6 months.

Figure 32-6 In approximately 10% of implants with spontaneous premature exposure, the implant has exudate or significant bone loss.

Figure 32-7 The periapical radiograph of the previous implant at implant placement **(A)** and 3 months later after spontaneous premature exposure **(B).** Note the 4 mm of marginal bone loss. *(Courtesy E. Barboza, São Paulo, Brazil.)*

contaminated crevice of the cover screw is more likely to be in a septic environment under these conditions. Therefore whenever the implant cover screw is partially exposed, the first action is to completely expose the implant (Figure 32-9). The cover screw is then removed

WWW.HIGHDENT.IR

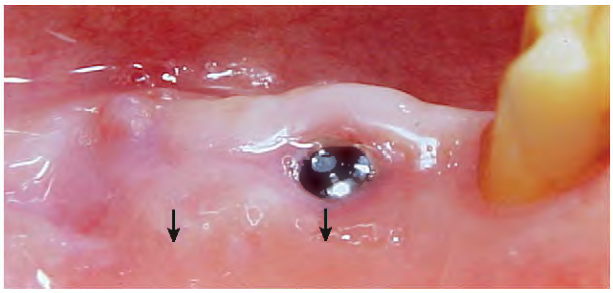


Figure 32-8 The two implants have become partially exposed during the initial healing process. The smaller the exposure (implant on left has pain point exposure), the greater the risk of marginal bone loss. *(Courtesy E. Barboza, São Paulo, Brazil.)*

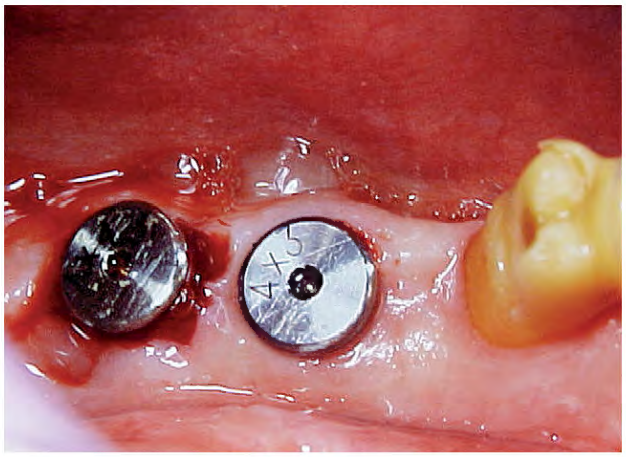


Figure 32-9 The implants in the previous picture have been completely exposed and flushed with chlorhexidine 0.12%, and the permucosal extension has been inserted. *(Courtesy E. Barboza, São Paulo, Brazil.)*

and the implant body irrigated with chlorhexidine 0.12%. A permucosal extension (PME) is then inserted into the implant. After the implant is completely exposed in the oral environment, a daily oral hygiene regimen is in order. This consists of twice-daily plaque removal with a soft toothbrush and the direct application of chlorhexidine 0.12%. If the patient is wearing an overlaying removable prosthesis, it should be modified so that no direct force is applied to the implant until after the bone interface is ready for occlusal loading.

There are most likely three reasons to explain the greater crestal bone loss around a prematurely exposed implant. The first is related to the microgap. Bone is able to grow over the cover screw crevice when covered by soft tissue. However, after the cover screw (or PME) is exposed in the oral environment, the oral epithelium most often repositions itself 0.5 to 1.0 mm below the crevice of the cover screw, usually within a few weeks. A second cause of marginal bone loss is related to the crest module design. When smooth metal is placed below the bone and the implant is exposed to the oral environment, the marginal bone loss extends apically until it reaches a rough surface or a groove (or thread) on the implant body. This process also occurs within the first month. As a consequence, submerged healing implants have less bone loss than implants, which becomes prematurely exposed.

It should be mentioned that after the implant is uncovered and used for prosthetic support, the phenomena described occur and bone loss becomes evident. Although submerged implants exhibit less marginal bone loss after initial healing, the overall marginal bone loss of two-stage and one-stage implants may be similar by the time the prosthesis is delivered to the patient.[17]

In addition to marginal bone loss from the microgap and the biological width, additional bone loss may occur from bacterial plaque. Rams and Link noted the bacterial environment becomes at greater risk of an anaerobic component when the probing depth is greater than 5 mm.[18] The anaerobic condition may be established around a prematurely exposed implant in two ways: A partial exposure of the implant has a greater risk of an anaerobic environment, and the cover screw is not tightened to 10 to 30 N-cm and may be loose, making the implant internal structure more likely anaerobic. Either of these conditions increases the risk of exudate or inflammation with greater marginal bone loss. It should be noted this cause of marginal bone loss could be corrected by uncovering the implant completely and flushing the region with chlorhexidine 0.12%. However, the bone loss that occurred before this event would not be corrected at this point.

Another factor to contribute greater marginal bone loss is that the implant may have become prematurely loaded through the soft tissue, causing tissue dehiscence. The premature load may cause the cover screw to loosen (increasing the anaerobic environment) and overload the developing crestal bone-implant interface.

UNCOVERY PROCEDURE

When more than three implants are uncovered, the patient is prepared for surgery in a manner similar to the original stage I procedure. In general, discomfort, swelling, and risk of infection occur to a lesser degree than at the original surgery. However, if corrective procedures are indicated during surgery, they should not be compromised by poor patient preparation. This chapter primarily addresses uncovery of multiple implants out of the primary esthetic zones. Maxillary anterior implants in patients with high lip lines are treated similar to anterior single tooth replacements, which are discussed in Chapter 33.

Soft Tissue Incision

The initial incision for stage II surgery is as important as in the first stage. When a one-stage surgical approach is used, attention to the soft tissue is similar to the following procedure. The incision is designed to place

WWW.HIGHDENT.IR

Figure 32-10 At stage II uncovery, the crestal incision bisects the keratinized tissue, ensuring that the facial tissue has more than 1.5 mm of attached tissue.

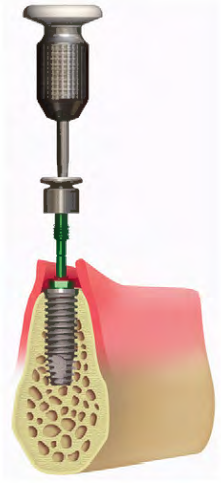


Figure 32-11 The soft tissue reflection is extensive enough to reposition the keratinized tissue around the permucosal extension of the implants or to correct any bone defect around the implants.

keratinized tissue on each side of the permucosal abutment. Therefore the full-thickness incision bisects the attached tissue on the crest of the ridge (Figure 32-10). The attached tissue and incision are usually more lingual than the actual implant site. As a result, a tissue punch is rarely used, because it usually eliminates the facial attached tissue around the implant site. The incision continues at least 5 to 10 mm distal to the last implant placed. A full-thickness envelope flap design permits reflection of the periosteum for direct implant-bone interface observation. A tissue punch may be used to uncover the implant when there is 1.5 mm or more of attached keratinized tissue facial to the implant site. This occurs more often in the posterior maxilla or when an implant was immediately inserted after the tooth was extracted.

The periosteal elevators should not be levered against the endosteal implant body or first-stage cover screw during this procedure. Instead, the lingual or palatal bone is used for leverage, and the facial tissues are

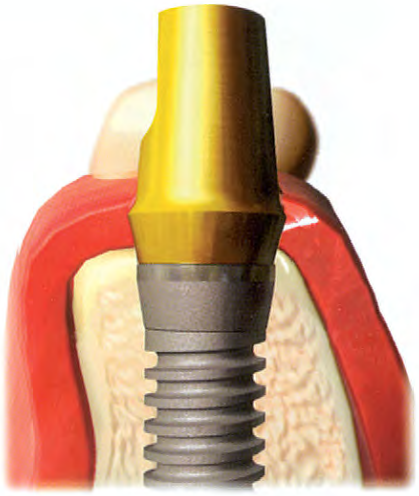


Figure 32-12 The abutment or permucosal extension may be larger in diameter than the crest module of the implant. As such, when bone is over or adjacent to the cover screw, the marginal bone must be recontoured to allow complete seating of the abutment.

gently pulled off the healed implant sites. Adequate reflection of the soft tissue completely exposes the crestal bone around the implant site and allows repositioning of the attached tissue at the conclusion of the procedure (Figure 32-11). If the healing cover screw became exposed during the healing phase, the primary crestal incision is made along the lingual aspect of the healing cover, and a sulcular incision is placed around the rest of the implant. A mucoperiosteal flap is then reflected in a fashion similar to that previously described. A lack of attached tissue is common on the facial aspect of the implant when it becomes exposed during initial healing and may require a tissue graft or acellular tissue graft on the facial to restore attached or immobile tissue.

Bone-to-Implant Interface Evaluation

After the soft tissues are reflected, the first-stage cover screws are identified and the surrounding area is closely evaluated. Many implant abutments are wider than the first-stage cover screw and may require as much as 1 mm of horizontal space around the implant platform on the crest module (Figure 32-12). Any bone growth on the cover screws or over the surrounding region is removed with surgical curettes or low-speed rotary uncovery burs designed to remove excess bone, accompanied by cooled sterile saline irrigation (Figure 32-13).

Bony Defects

On occasion, a vertical or horizontal defect may be revealed around the uncovered implant. The causes for this defect are often similar to those of premature implant exposure and include crestal bone trauma during surgery, excess torque from implant insertion (especially with wider crest modules), bone flexure

WWW.HIGHDENT.IR

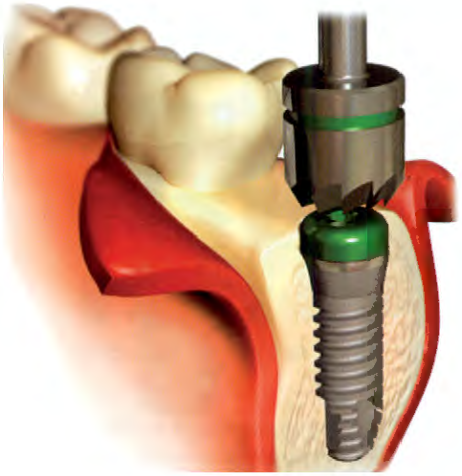


Figure 32-13 A bone profile drill in a low-speed hand piece, with copious irrigation, may remove the bone over or adjacent to the crest module and permit the abutment to be seated completely on the implants.

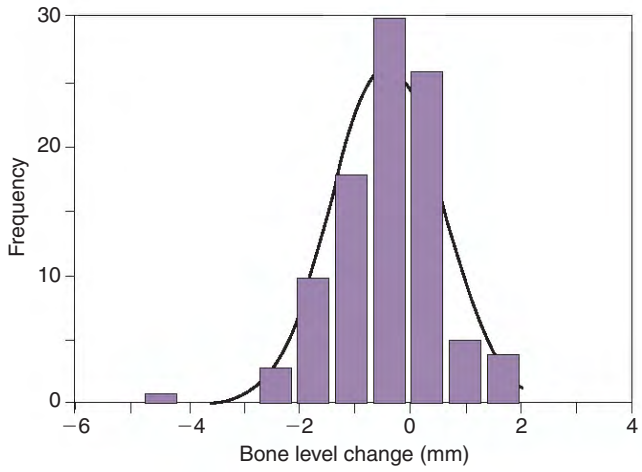


Figure 32-14 The range of bone loss in the Kline et al. study at stage II uncovery was from −5 mm to +2 mm. The average bone loss was 0.2 mm. (*Kline R, Hoar JE, Beck GH et al: A prospective multicenter clinical investigation of a bone quality-based dental implant system*, Implant Dent 11:332-334, 2002.)

or torsion in the posterior mandible, local patient habits that load the implants during healing, incision line opening, postoperative infection, implant surface contamination, idiopathic bone loss, or healing factors related to systemic disease. In a study by Kline et al., the average crestal bone loss was 0.2 mm at stage II uncovery, but the range was −5 mm to +2 mm (Figure 32-14) (Box 32-2).[2]

Vertical Defects

If a vertical defect filled with soft tissue is identified anywhere around the implant, a curette is used to eliminate it (Figure 32-15). When soft tissue is removed from around a tooth, the root is scraped because the tissue attaches to the cementum. The fibrous tissue in a vertical defect around an implant is not attached to the

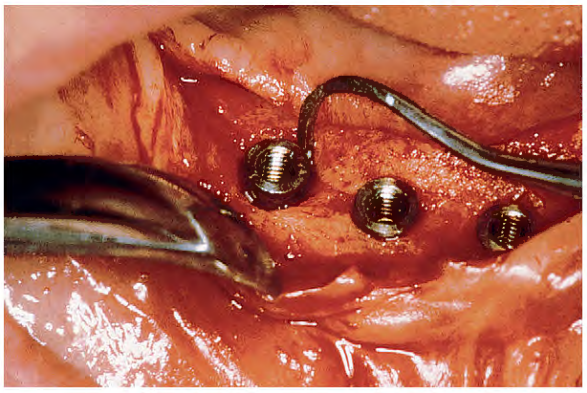


Figure 32-15 A soft tissue in a vertical defect is removed by using a curette that scrapes the surrounding bone, not the implants.

Box 32-2 Causes for Bone Loss at Stage II Uncovery

- Premature implant exposure
- Crestal bone trauma at surgery
- Excess torque at implant insertion
- Bone flexure/torsion in posterior mandible

implant. Therefore the bone is scraped, not the implant. This loosely bound and unorganized tissue is relatively easy to remove at this time. The implant surface should not be scratched or contaminated during this procedure. The extent of bone loss is assessed and should be less than 3 mm if the implant is to be uncovered at this appointment (Table 32-2).

If the vertical defect around the implant is of moderate depth (greater than 3 mm) for more than 25% of the circumference, a barrier membrane is placed over the grafted defect and the soft tissue is reapproximated. This prevents soft tissue ingrowth into the defect and provides an improved environment for the bone graft healing time against the implant surface. In this scenario, the second-stage uncovery is delayed for approximately 2 to 4 months, depending on the size of the defect.

When bone loss exposes the threads of the implant body, the ability to reform bone in the defect when the implant is uncovered and loaded is reduced. When the implant has not been exposed before the uncovery procedure, the implant body is usually not contaminated by microorganisms because it has been under soft tissue. The full-thickness reflection of the region has exposed areas of vital bone not involved in the implant support. This bone may be harvested and packed into the vertical defect (after the soft tissue in the defect has been thoroughly removed). The defect and surrounding area are overpacked. When the implant defect is larger than 3 mm, the most predictable method to correct the condition is with a particulate autologous bone graft covered by a resorbable membrane (i.e., Alloderm, Biomend), and the soft tissue is reapproximated over

WWW.HIGHDENT.IR

Table 32-2 Resolution of Bone Loss at Uncovery

DEFECT	MANAGEMENT	OPTIONS
Vertical <3 mm	Osteoplasty	Convert into horizontal defect by leveling off bone
Vertical >3 mm	Less than half of implant height	Graft with autogenous bone wedge or membrane if deep defect
	More than half of implant height	Remove implant
Horizontal	Less than half of implant height	Position soft tissue apically; graft autogenous bone
	More than half of implant height	Remove implant

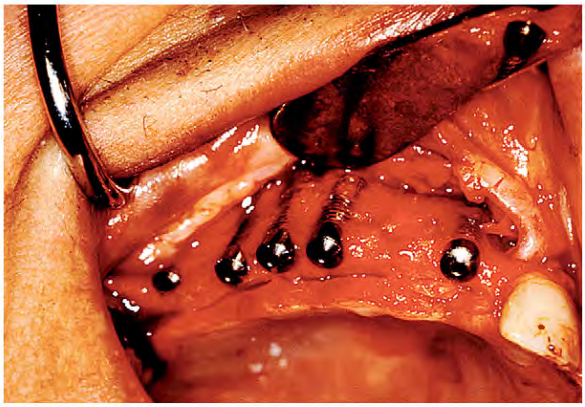


Figure 32-16 The defect at implant uncovery is greater than 3 mm and threads are exposed on the implant body. A particulate autograft covered with a membrane and primary closure is suggested.

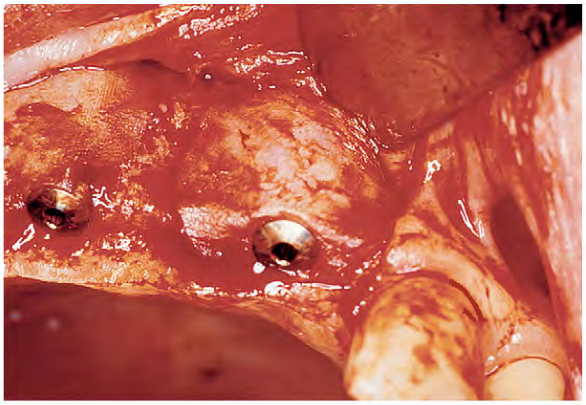


Figure 32-17 At the uncovery 8 to 12 weeks later, bone is now present over the threads of the implants.

the membrane, bone graft, and implant for an additional 8 to 12 weeks of healing (Figures 32-16, 32-17). A vertical defect greater than 3 mm is usually grafted, unless it represents half or more of the total implant height, in which case the implant should be removed (see Table 32-2).

When the vertical bone defect around an implant is less than 3 mm, the implant may be uncovered and used in the current condition for the prosthetic abutment. After the soft tissue is removed from a defect, the surrounding bone is again evaluated. In the case of a vertical bone defect of less than 3 mm, there are three surgical options. An osteoplasty may eliminate

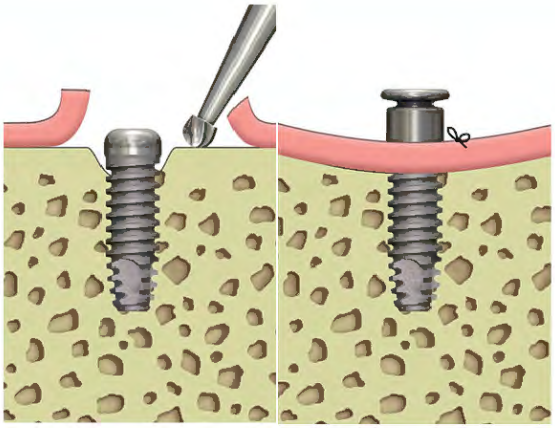


Figure 32-18 When the defect is less than 3 mm **(A)**, the implant may be uncovered. An osteoplasty to eliminate the defect **(B)** is the most predictable method, but modifies it into a horizontal defect. This may be inappropriate in the esthetic zone.

the vertical defect when the reduced bone-implant interface does not compromise the prosthetic support or esthetics. The PME is placed at the same appointment (Figure 32-18). A second method to correct a vertical defect less than 3 mm is to curette the defect and overfill the region with an autograft (Figure 32-19). The PME may be added at the same appointment and the tissue approximated around the site (Figure 32-20). When there is a desire to have thicker soft tissue around the site, a barrier membrane (e.g., Alloderm) may be used over the implant site and covered with soft tissue.

A third alternative to correct a vertical defect on the mesial and distal region is to drive a wedge into the bone several millimeters away from the implant body. Tapping a wedge-shaped osteotome into the distal bone compresses vital bone up against the implant body (Figures 32-21, 32-22). The wedge-shaped defect created in the ridge as a result of the osteotome is several millimeters away from the implant and surrounded by bone; therefore this defect will heal without consequence. A facial or lingual vertical defect may be corrected by taking a blunt instrument with a mallet and compressing the facial or lingual bone against the implant body. This technique also places living bone adjacent to the implant body (Figure 32-23). The PME is placed at this appointment.

WWW.HIGHDENT.IR

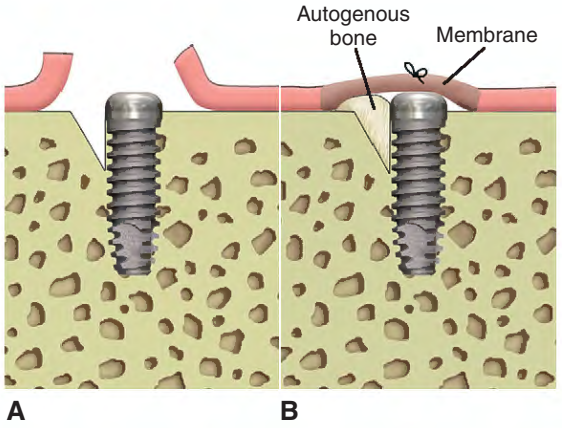


Figure 32-19 A second method to correct a vertical defect less than 3 mm **(A)** is to curette the defect and fill the region with an autograft **(B).** The implant may be exposed with a permucosal extension or covered by a membrane when additional soft tissue thickness is desired.

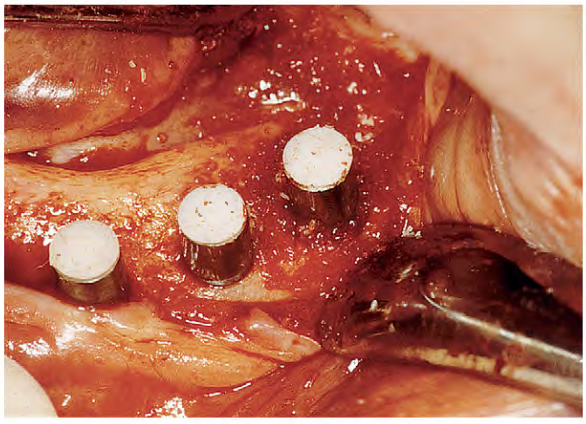


Figure 32-20 The implant on the right has a vertical defect less than 3 mm. The permucosal extension is added to the implant; autograft is collected from the surrounding area and overfills the defect. The implant may then be uncovered.

Horizontal Defects

A horizontal bony defect of bone around the implant body may also be treated in several ways. When horizontal bone loss around an implant is more than half of the implant body, the implant should be surgically removed and the site grafted at the uncovery.

The most predictable method to treat horizontal bone loss that is less than 50% of the implant body is to reduce the soft tissue thickness to decrease the probing depth around the implant. The soft tissue may be apically repositioned, exposing a portion of the implant body into the oral cavity. If threads or a rough surface are present above the bone, an aluminum oxide ("white") stone and rubber wheel are used under copious irrigation to smooth the region and limit plaque accumulation. Cement-retained prosthesis on an implant with horizontal bone loss may be placed on the implant body in esthetic regions.

Another option to address horizontal bone loss is to grow bone above the defect, and this method is

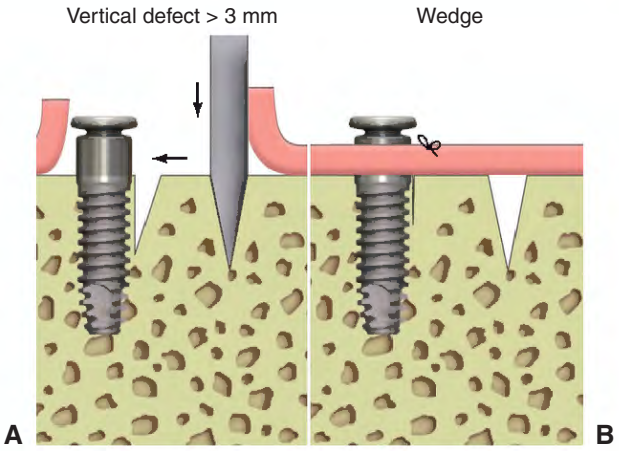


Figure 32-21 **A,** The implant on the left has a vertical defect less than 3 mm deep. **B,** A wedge is driven into the bone in the appropriate side, and vital bone is pushed up against the implant.

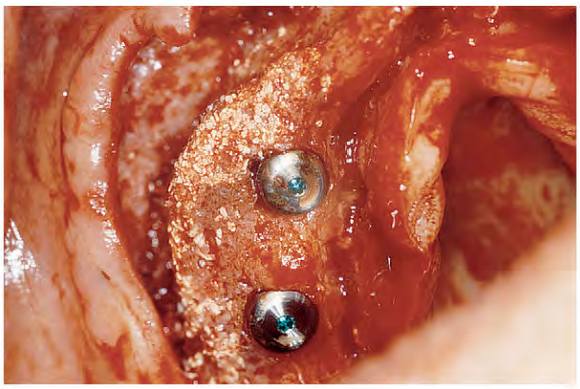
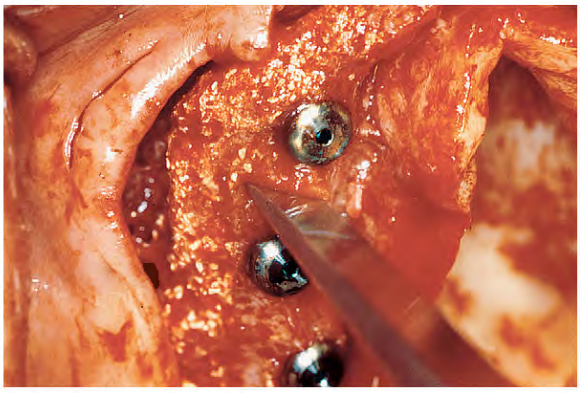


Figure 32-22 An implant in the canine region has a defect on the facial and distal aspects.

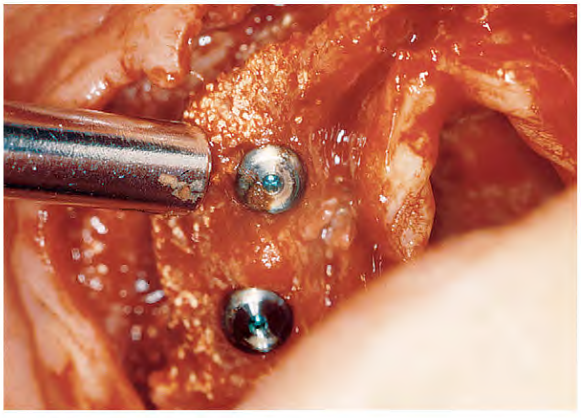
used when the final prosthesis is FP-1 or additional bone-implant interface is required to withstand the forces exerted on the prosthesis. To improve the amount of bone formation, several steps may be taken. The first is to use autogenous bone for the graft. In most cases, bone is harvested and placed on the crest, after the region has been curetted to increase blood supply and increase the regional acceleratory phenomenon. A barrier membrane is also placed over the site to prevent fibrous tissue ingrowth into the region. Anaerobic bacteria are often growing on the implant body when bone loss is present and should be mechanically removed. In addition, before grafting, the first-stage cover screws are removed, and the internal cavity of the implant body is thoroughly flushed with chlorhexidine 0.12% before replacing the screws into the implant body. The tissues are reapproximated over the bone graft and membrane with primary closure. The second-stage uncovery is delayed for approximately 3 to 4 months, depending on the size of the horizontal defect and bone graft.

On occasion, the facial aspect of the implant is exposed for several millimeters. This more often occurs when the implant was placed in the maxilla and the

WWW.HIGHDENT.IR



A



B

Figure 32-23 **A,** A wedge is created on the distal aspect of the implant and tapped into place. The distal wedge bone is pressed against the distal defect of the implant. **B,** A blunt instrument placed on the facial aspect is tapped with a mallet and compresses the facial bone against the implant, which eliminates the defect.

ridge was less than 5 mm in width or bone expansion was performed to insert the implant. Although the implant has rigid fixation, the lack of facial bone places the implant at an increased risk. The implant site may be improved at the uncovery surgery by performing an onlay graft. Most often, the easier method to accomplish the augmentation is the layered, particulate, membrane graft technique described in Chapter 37. The ridge is decorticated on the facial, mesial and distal aspects to the implant. Autologous bone is harvested with a trephine bur from above and lateral to the implant site. The autograph is particulated in 3 × 3-mm pieces and laid over the facial aspect of the implant. A mixture of 30% demineralized, freeze-dried bone cortical fibers (Grafton) and 70% mineralized, freeze-dried bone allograft (MinerOss) is mixed with the platelet-rich plasma from 10 to 20 mL of whole blood. A resorbable membrane (i.e., Biomend) or acellular tissue (AlloDerm) covers the graft.

Because the implant is already integrated, a PME may be placed into the implant body and the tissue approximated around the implant. Before soft tissue closure, the labial flap should be expanded with tissue

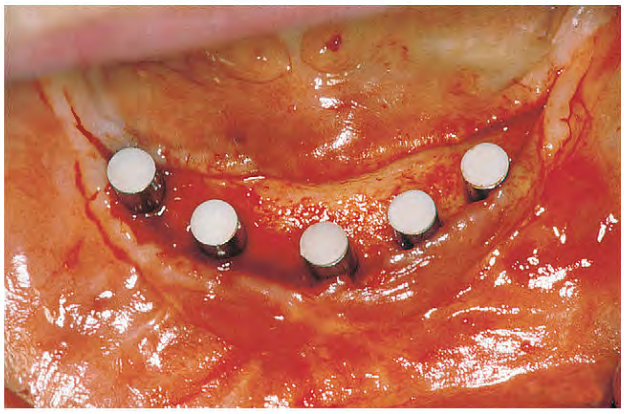


Figure 32-24 If the bone levels are acceptable, a 3- to 5-mm-high permucosal extension is threaded into the implant body.

scissors (i.e., Metzenbaum scissors). The final prosthesis should be delayed for more than 3 months when this procedure was performed at the uncovery appointment to allow maturation of the tissues.

RIGID FIXATION DETERMINATION

If the bone level is acceptable around the crest module of the implant, the first-stage cover screws are removed, the internal chamber of the implant body is thoroughly flushed, and the top of the crest module is closely evaluated to make sure it is free of any bone or soft tissue over the edges. A 3- to 5-mm-high, second-stage PME is then seated with finger pressure into the implant body (Figure 32-24).

The rigid fixation of the implant body is then tested for resistance to the torque required to place the permucosal extension with 10 N-cm torque (less than half the final force for the abutment). This may be accomplished by freehand pressure and a short insertion tool. The clinician must use caution when tightening the PME because the torque force required to remove the permucosal extension at the next appointment is similar to the force needed to tighten it. Reverse torquing is more critical when applied to the implant body in D4 bone at this stage of healing, because it may traumatize the bone-implant interface.

REVERSE TORQUE TESTING

Reports in the literature have suggested reverse torque testing (RTT) at stage II surgery or to assess an implant suitability for immediate loading.[19] Two alleged advantages are its use as a biomechanical measure of initial stability and its use as a definitive verification of initial osteointegration.

RTT has been used as a research tool for many years. Several research studies have been performed to ascertain the nature and strength of the bone-implant interface

WWW.HIGHDENT.IR

using both push-out and RTT techniques.[20-28] These findings have been useful for the development of implant body designs and surface coatings. A strong and stable bone-implant interface has always been intuitively desirable. Interest in resistance of bone-implant interface to torsional forces has been heightened as a result of the increased awareness that specific (and higher than previously believed necessary) torquing preload forces are needed to reduce the incidence of abutment screw loosening. From a clinical standpoint, it is desirable to determine whether the bone-implant interface can withstand the preload torque forces needed to tighten abutment screws.

Several reports[3-5,29] have reported on the phenomenon of early loading or late failures, whose incidence ranges from 2.5% to 5.9%. The term *late failure* in these reports refers to implants that have failed in the time between an apparently successful stage II surgery and before the completion of prosthetic restoration. In the Misch and Jividen classification, this is an *early loading failure.* The rationale for RTT is based on the premise that most, if not all, late failures are the result of an inconspicuous nonintegration, which is a condition whereby an implant passes current assessments for the evaluation of rigid fixation (manual mobility, radiographs) at uncovery, yet it is insufficiently integrated to indefinitely remain in that state. These implants are said to have a subclinical micromovement that is not visible or detectable with existing techniques. RTT was suggested to subject implants to early objective testing before undergoing prosthetic reconstruction.

RTT, used as verification for rigid fixation, involves placing a defined reverse torque (counterclockwise) to the implant (via mount) at stage II uncovery surgery. The level of applied torque ranges from 10 to 20 N-cm. In a study using a primate model, Carr et al.[24] found that 5.4% of the metallic implants could not withstand a reverse torque level of 35 N-cm. Implants failing such testing are presumed to be nonintegrated, likely to become early loading failures, and are therefore not recommended for use as prosthetic abutments.

It has been suggested that RTT is not advisable or necessary in D1 or D2 bone.[19] Concerns of stripping of the external hex in implants made of grade I titanium, because of the higher bone strength and presumed lower incidence of early failures lessen or obviate the need for RTT for these bone types. However, a multicenter clinical trial involving 2131 implants in 707 patients found the highest stage II failure rate was actually for the most dense bone.[30] As a result, it appears that implants in all quality bone would also have high enough incidence of early failures to justify the evaluation of every implant. In addition, if the same N-cm torque is used in all bone densities, the hex components are at similar risk.

The strength of trabecular bone before fracture in the jaws is directly related to its density. The densest bone (D1) is 10 times stronger than the least dense (D4). The RTT is suggested to be most beneficial in less dense bone, yet this density bone is most at risk of failure during the RTT evaluation. In addition, bone is often only 60% mineralized at stage II uncovery after the surgical trauma from stage I surgery. According to computer densitometry reports, bone may be more dense and the bone interface stronger on the day of surgery, compared with the stage II uncovery date.[8,31,32] It takes 52 weeks after initial surgery for bone to be completely mineralized. The mineralization of bone is related to its strength. Therefore using RTT at stage II uncovery evaluates the interface when it is weak and more at risk to fracture from overload. In addition, at 4 months, the bone is often still histologically woven bone, rather than lamellar bone. Woven bone is unorganized and weaker than the load-bearing lamellar bone, which is more desirable at the implant interface.[33] An RTT at uncovery does not permit this improved interface to develop before testing. RTT of implants too early in the healing process (relative to bone density) is more likely to lead to the removal of implants that would otherwise be integrated.

RTT protocol requires a clinician to detect even small micromovements that would indicate failure. Sullivan states that "on initial activation of the electronic torque control unit, the illusion of movement can be ascertained when slack is taken out of the system."[6,19] This movement is subtle enough that the use of magnifying loops is recommended. The amount of implant movement of successful rigid fixated implants is variable[21] and dependent on bone density.[35] An implant in D4 bone may move 60 to 80 µm. In comparison, a lateral incisor tooth in health moves 97 µm, which can be seen with the naked eye. The illusion of movement may be attributed to bone density, not to a lack of rigid fixation or fibrous encapsulation.

Torque-to-failure trials in human dental applications have been limited to only two studies. Tjellstrom[21] measured the removal torque of osseointegrated titanium implants placed in the mastoid bone and found a range from 26 to 60 N-cm. The mastoid bone is cortical externally and coarse trabecular internally. This corresponds to a D2 bone description. Sullivan et al.[19] recorded that removal torque values for three implants in a human volunteer were between 45 and 58 N-cm. However, the implants for Sullivan were surrounded by "extremely dense marble-like bone quality." Therefore the RTT values to failure of these implants cannot be used to simulate the RTT of implants in D3 or D4 bone, which is not surrounded by a marble-like structure.

Bone, as a structure, exhibits significant variations in strength depending on the types of forces acting on it. These forces may take the form of compression, tension, or shear. Research has shown that cortical bone is strongest in compression, 70% as strong in tension, and only 35% as strong in shear.[36,37] An RTT places primary shear forces on root form implants with a round

WWW.HIGHDENT.IR

cross-section design. The only region of compression for RTT would be the bone grown into the apical hole at the bottom of the healed site. Therefore the force type (shear) is the weakest to the RTT method of testing.

The RTT may also be affected by implant design. Implant bodies, such as hollow basket designs or those with flat sides to resist shear forces, may exhibit greater RTT values to failure. An RTT on a threaded implant uses the mechanical advantage of a screw to break the implant-bone adhesion. The screw would also convert the shear force to some tensile force. An RTT attempts to lift the implant from the crypt, using the screw design to assist this aim.

Occlusal loads aim at being primarily compressive in origin. Implant bodies are not designed for pure shear loads; therefore RTT applies forces that do not correspond to clinical loads. Failure of an implant during RTT does not necessarily mean that it could not have functioned as a successful implant under (primarily) compressive loads.

The stated need for RTT is to help reduce the number of early loading failures, with the assumed cause being a subclinical nonintegration at stage II surgery. A more likely cause of these failures is early implant loading beyond the bone strength present around the implant. Progressive bone loading studies have demonstrated the density (and related strength) of bone increases after loading.[38,39] Early implant failures may be reduced to less than 1% when progressive loading is performed on implants of adequate size, design, and number. Reverse torque testing has been represented as a possible means to predict early implant failure that most likely has a fibrous tissue interface. However, these failures are more likely from excessive stress at the bone-implant interface and excessive occlusal loading.

The criteria suggested for a clinical verification standard are that it be objective, be easy to administer, use available armamentaria, be as definitive as possible within the available knowledge base, and possess an adequate level of safety so that damage to the implant-bone interface does not occur. This is not found in RTT evaluation.

In the author's opinion, RTT presents too many subjective variables. Misjudgment can damage the bone-implant interface and prolong treatment and increase costs associated with the extra reparative treatment. The desire for objective standards for clinical verification of osteointegration is understandable. The most common methods of clinical verification used today, radiographs and manual mobility testing, have an admittedly subjective component. However, they do not place the implant at risk and have stood the test of time.

When the root form implant has a fibrous tissue interface, there is little resistance to rotation in either a clockwise or counterclockwise direction. Therefore if fibrous tissue surrounds the implant, and a permucosal extension is threaded into the implant body with a 10 N-cm force, the implant will visibly turn and the insertion and tightening of the healing cap can be used to evaluate the complete lack of integration with the bone. In addition, a 10 N-cm force on the permucosal healing cap places a screw implant under shear tension and compression on the bone. The clockwise turn on a screw will more likely resist these forces, even in very soft bone.

A vertical force places more compressive forces on the implant-bone interface. The mobility test with a lateral force applied to the side of the permucosal extension also places some compressive forces on the implant-bone interface. Therefore the implant is initially evaluated for rigid fixation with the torque test to thread the permucosal healing cap into position and then evaluated similar to a natural tooth mobility test, with a 5-lb axial and lateral load.

The Periotest (or resonance) is a nondestructive, objective, and useful test to determine implant micromovement. A study by van Steenberghe and Quirynen[40] suggests that the prognostic value of the Periotest remains unproven with regard to implants because too few failures have been studied and reported in the literature. However, several studies exist that describe the usefulness and sensitivity of this instrument.[41-45] Furthermore, a large-scale study of 1838 implants by Truhlar et al.[45] and 4-year evaluation by Misch[46] found the Periotest to be capable of assessing the status of the implant-bone interface. It is sensitive to slight differences in the implant dampening effect (mobility) and provides baseline Periotest values that can be helpful in evaluating improvement or degradation of the implant-bone complex.[47-49] However, a disadvantage is that many clinicians do not have access to these instruments.

IMPLANT POSITION

After the implant is uncovered and a PME is seated into the implant body, the prosthetic template is inserted and the implant position evaluated. An implant body in a position that compromises the prosthesis should not be maintained. The inappropriate implant may often be removed by reverse torquing with a hand ratchet. Luxation, as with a tooth, is not indicated because it places compression forces on the implant-bone interface and usually leads to the fracture of either the bone or the implant before its removal. A trephine drill may be required to remove an implant in denser bone qualities. A trephine drill can create a small space all around the implant to its base (Figure 32-25). A forcep then is used to rotate and shear the base of bone at the apex.

The implant is removed if its position places the final prosthesis at risk or if more than one half of the

WWW.HIGHDENT.IR

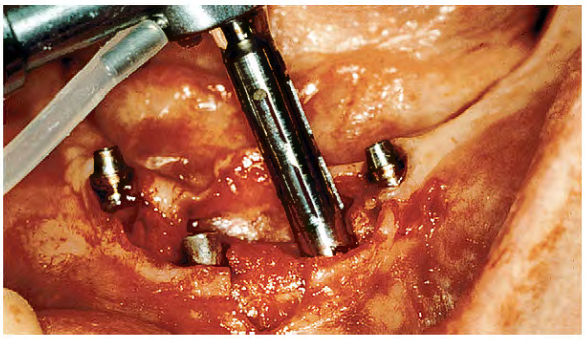


Figure 32-25 When the implant is in too poor a position to be restored or more than 50% of the bone is missing, a trephine bur may be used to remove the implant.

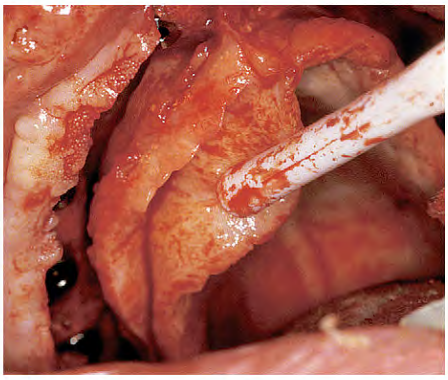


Figure 32-27 An incision is made on the periosteal side of the palatal flap, and the excessive connective tissue is excised to decrease the overall palatal tissue thickness.

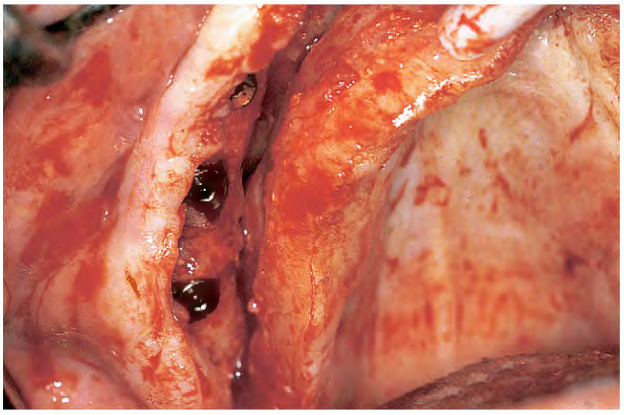


Figure 32-26 When the overlying tissue is greater than 4 mm thick, the soft tissue should be reduced in thickness, thus reducing the sulcus around an implant.

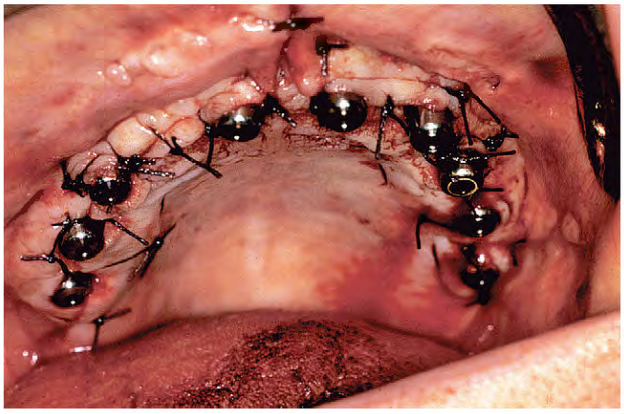


Figure 32-28 The tissue with a reduced thickness may then be approximated around permucosal extensions 3 to 5 mm in height.

implant has lost bone in a vertical dimension. Several options exist if implant removal is indicated: (1) the implant may be removed, and if enough implants remain, the prosthesis may still be fabricated; (2) the prosthesis may be converted from a fixed restoration to a removable RP-4 or RP-5 restoration to decrease forces to the superstructure in amount or duration; (3) an additional implant may be placed in an optional implant location at the same time as the implant removal; (4) the implant may be removed and a larger-diameter implant simultaneously inserted with a different angulation or deeper within the osteotomy; and (5) the implant may be removed, the site augmented, and an additional implant placed months later. The treatment of choice attempts to best satisfy the conditions for the patient and prosthesis without compromise.

TISSUE THICKNESS

The thickness of the overlaying crestal tissue is evaluated once it is reflected. Soft tissue thicker than 4 mm will result in a less than ideal pocket depth around the implant (Figure 32-26).[47,48] The tissue is relieved from the periosteal surface especially in the labial flap, until it is less than 3 mm thick (Figures 32-27 and 32-28). If abundant attached tissue is present in the palatal region of the maxillary implants, a gingivoplasty may be performed (Figure 32-29).

Permucosal Abutment Selection

The 4-mm permucosal healing cap should extend at least 1 mm above the tissue after suturing to help prevent tissue overgrowth during the next few weeks (Figure 32-30). An enlarged permucosal extension may be designed as part of the healing abutment (e.g., Maestro, BioHorizons) or attached to the healing abutment (e.g., Nobel Biocare). This larger contour helps maintain the apically positioned tissue in place, or it may support a periodontal dressing to maintain the tissue in place.

A suture groove, 3 to 5 mm above the platform connection, may be incorporated in the healing abut-

WWW.HIGHDENT.IR

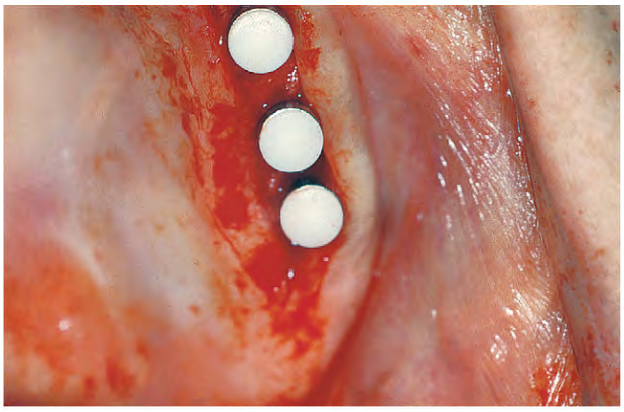


Figure 32-29 A gingivoplasty is often performed in abundant areas of keratinized, attached tissue, such as on the palate, to reduce sulcus depth around the implant.

Figure 32-30 The permucosal extension should extend at least 1 mm above the soft tissue after it is approximated around the implants.

Figure 32-31 A suture groove in the permucosal extension may be positioned 3 to 5 mm above the bone.

ment (e.g., Maestro, BioHorizons) (Figure 32-31). When the tissue requires apical repositioning or when it is 3 to 4 mm thick and may grow over the healing abutment, the suture groove may be used (Figure 32-32). A suture is placed next to the healing abutment. Tissue forceps lifts the suture from the incision line, and the suture is then rotated to form a loop. The loop is placed over the enlarged healing abutment and into the suture groove

Figure 32-32 The suture groove helps to apically reposition the tissue, so it will remain less than 3 to 5 mm thick, to reduce the sulcus depth.

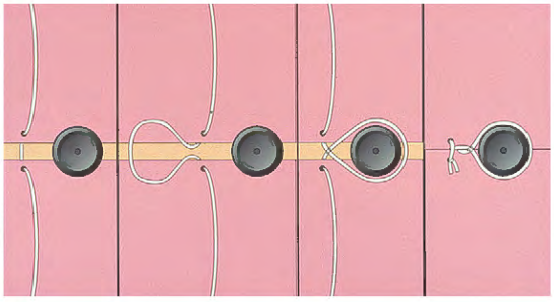


Figure 32-33 The suture groove in the permucosal extension may be used to loop the suture around the groove and tie the tissue at a more apical position.

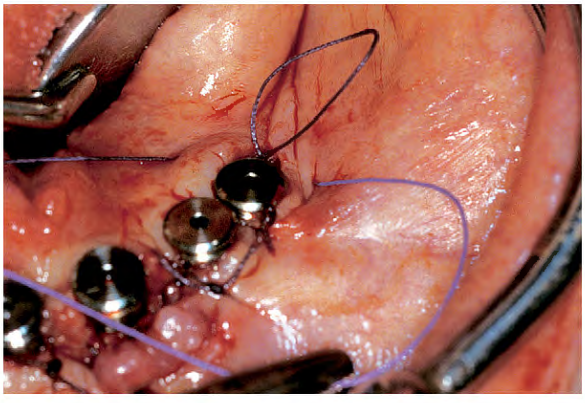


Figure 32-34 The most distal implant to the right has mobile tissue around it, which is difficult to reposition apically. The suture is placed on the distal aspect and looped over the suture groove to act as a soft tissue retainer.

or under the healing cap. The suture may then be tied, securing the tissue at the height of the suture groove (Figure 32-33). A similar technique is used on the other side of the healing abutment. These two sutures (one on each side) hold the tissue at the level of the suture groove and prevent it from lifting up and over the healing cap during soft tissue healing (Figures 32-34, 32-35).

A healing abutment wider than the implant crest module may be selected when the implant is not countersunk below the bone, with the following advantages:

WWW.HIGHDENT.IR

Figure 32-35 When the suture is tied around the permucosal extension, it apically positions the tissue around the implants.

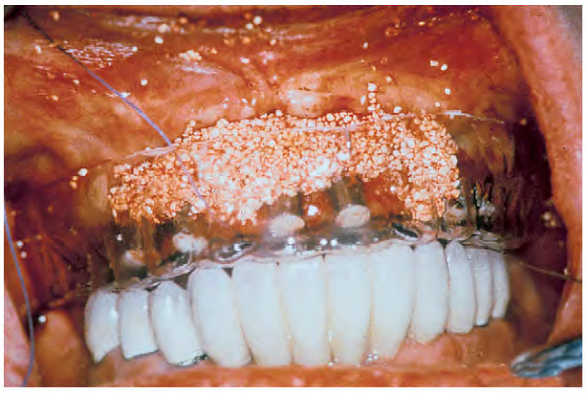


Figure 32-36 A prosthetic template of the labial flange of the denture indicates how much dense hydroxylapatite is required to support the lip above the fixed-implant prosthesis.

1. The perimucosal tissue heals with a larger diameter than the final abutment. As a result, when the final abutment is added after healing, the risk of entrapping soft tissue between the final abutment and the implant body is eliminated.
2. The wider space remains around the abutment for less than 30 minutes. Subgingival abutment preparations, impression, placement of retraction cord, and development of an emergence profile for the crown may all be facilitated when the space around the abutment is present.

The bacterial flora is similar around teeth and implants. Probing depth averages between 1.5 and 4 mm provide an environment that is conducive primarily to aerobic bacteria. As the sulcus depth increases beyond 5 mm, the incidence of anaerobic bacteria increases.[50] When 75% of the bacteria are anaerobes, an active process places the region at risk for disease. A dental probe next to a natural tooth is stopped by the connective tissue attachment zone. Unlike teeth, there is no connective tissue attachment next to an implant. In addition, the junctional epithelial attachment is less tenaciously bound to the implant compared with teeth. As a result, the same probing force used for a tooth will proceed almost to the crestal bone next to an implant. In addition, when plaque is allowed to accumulate next to an implant, the number of inflammatory cells infiltrating the connective tissue are found to be much larger and more extensive.[51] As a result, it has been suggested to maintain tissue thickness around the implant abutment less than 4 mm to decrease the risk of bacterial contributed bone loss.[52,53]

The sulcus depth next to an implant is directly related to the thickness of the tissue over the bone. The tissue thickness before implant placement is variable and may be greater than 6 mm, especially in the maxilla. During stage I implant placement surgery, excessive tissue thickness may be reduced. However, tissue thickness may be of benefit during initial healing to act as a cushion over the implant and reduce the risk of implant micromovement during healing. One of the goals of stage II uncovery surgery is to reduce the tissue thickness and therefore the final abutment sulcus depth to less than 4 mm. A 4- to 5-mm-tall PME is often selected at stage II uncovery to ensure that less than 4 mm of tissue exists. Otherwise the tissue will be higher than the healing cap. In addition, when taller healing caps are used, a soft tissue–borne transitional prosthesis must be sufficiently relieved over the abutments (to prevent excessive loads on the immature interface), and a greater moment of force is transmitted to the implant crest region under lateral loads.

Soft Tissue Support—Ridge Augmentation

When the maxillary lip or pontic position requires greater support or contour for esthetics, dense hydroxyapatite (HA) may be added to the labial bone surface for fixed prostheses. The prosthetic template is placed over the implant site and should represent the desired lip support region to determine the amount of labial HA graft required (Figure 32-36). The augmentation aims at providing an improved esthetic result and should not be attempted to increase the bone volume. Another alternative is the use of connective tissue or acellular tissue grafts (Alloderm, Lifecell, Branchburg, NJ), which may have the same goal. The advantage of HA for this intent is that limited long-term tissue shrinkage is observed. In addition, an increase in bone density is often observed in regions where the particulate HA attaches to the bone.

KERATINIZED TISSUE

Keratinized Tissue Concerns

The absence or presence of a zone of keratinized gingiva around a natural tooth or an oral implant is controversial. No direct evidence confirms or denies

WWW.HIGHDENT.IR

the need for nonmobile keratinized tissue next to natural teeth. Lang and Loe[54] advocate a minimum 2 mm of keratinized gingiva and 1 mm of attached gingiva to maintain gingival health. In longitudinal studies, however, Wennstrom[55] and Kennedy et al.[56] demonstrated that the lack of adequate keratinized and attached tissue does not compromise the long-term health of soft and hard tissue as long as patients maintain good oral hygiene. Moreover, Stetler and Bissada[57] concluded that if subgingival restorations were to be placed in areas of minimally keratinized gingiva and less than optimal plaque control, augmentation to widen the zone of keratinized tissue may be warranted. They also noted that in unrestored teeth, the difference in the inflammatory status of sites with or without a wide zone of keratinized tissue was not significant. The tooth with the least amount of keratinized tissue is often the mandibular first premolar. Yet this tooth is rarely the first tooth lost from periodontal disease. If all other periodontal indexes are normal, the amount or absence of keratinized gingiva has little to do with the expected longevity of the tooth.

The need for keratinized tissue around dental implants seems more controversial than around teeth.[58-64] Several reports demonstrate the long-term implant survival in the absence of keratinized tissue. Although reports are more cautious with mobile mucosa next to an implant, nonmobile tissue appears to be the primary criterion relative to tissue type.

Although keratinized tissue around a tooth may not be mandatory for long-term health, a number of benefits are present with keratinized mucosa. The color, contour, and texture of the soft tissue drape should be similar around implants and teeth. The interdental papillae should ideally fill the interproximal spaces. A high smile line often exposes the free gingival margin and interdental papillae zones. The keratinized tissue is more resistant to abrasion. Hygiene aids are more comfortable to use. The degree of gingival recession appears related to the absence of keratinized gingiva. Root sensitivity and esthetic concerns may be associated with gingival recession. From a restorative dental aspect, keratinized mucosa is more manageable during the retraction and impression-making process. Subgingival margin placement is more precise, as is long-term stability, in the presence of keratinized tissue.

The presence of keratinized tissue next to an oral implant may present even greater benefits than those with natural teeth (Figure 32-37). Some reports indicate the lack of keratinized tissue may contribute to implant failure. Kirsch and Ackermann[60] reported that the most important criterion for implant health in the posterior mandible was related to the absence or presence of keratinized gingiva. Mobile, nonkeratinized mucosa exhibits greater probing depths, which has been confirmed histologically. A study by Warrer et al.[59] in monkeys found that an absence of keratinized mucosa increases the susceptibility of peri-implant regions to plaque-induced destruction.

Keratinized gingiva has more hemidesmosomes; therefore the junctional epithelial attachment zone may be of benefit when in keratinized tissue. The orientation of collagen fibers in the connective tissue zone of an implant often appears perpendicular to the implant surface, whereas these fibers in mobile, nonkeratinized tissue run parallel to the surface of the implant. Schroeder et al.,[58] James and Schultz,[63] and Lisgarten et al.[64] have suggested that mobile mucosa may disrupt the implant-epithelial attachment zone and contribute to an increased risk of inflammation from plaque.

In addition to general advantages, keratinized tissue around implants may be beneficial in several other ways. In a two-stage protocol, the implant is less likely to become exposed during the healing process. The formation of interdental/implant papillae is completely unpredictable with mobile unkeratinized tissues. However, no clinical or histologic benefits are reported with unkeratinized nonmobile mucosa. When the unkeratinized tissue is mobile, several reports state that this is unsatisfactory.

The question relative to the need for keratinized tissue around implants should be modified to "which would you prefer?" No one has stated that the unkeratinized tissue is better than keratinized tissue for any reason. Therefore the controversy has abated. Some authors prefer keratinized mucosa more intensely than others. If one side of controversy demonstrates benefits and the other side states that keratinized tissue is not mandatory, both sides may be correct.

In specific clinical instances, attached keratinized gingiva is more often desirable. For example, an FP-1 (fixed prosthesis type 1) restoration in the esthetic zone requires keratinized mucosa to develop the soft tissue drape around the implant crowns. A second prime example is a mandibular overdenture, which benefits from a vestibule and zone of nonmobile tissue around the implant abutments.

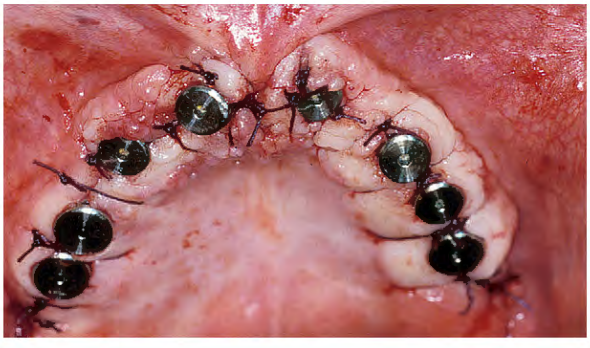


Figure 32-37 The presence of keratinized, attached tissue around implants provides even greater benefits than around natural teeth.

WWW.HIGHDENT.IR

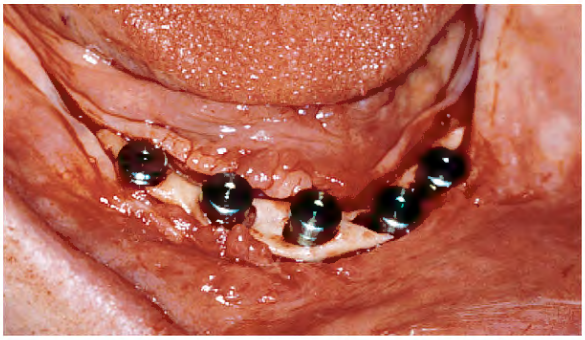


Figure 32-38 The anterior mandible was augmented with an iliac crest graft before implant placement. The crestal tissue presented little attached keratinized mucosa. An acellular tissue graft (AlloDerm) is placed around the implants.

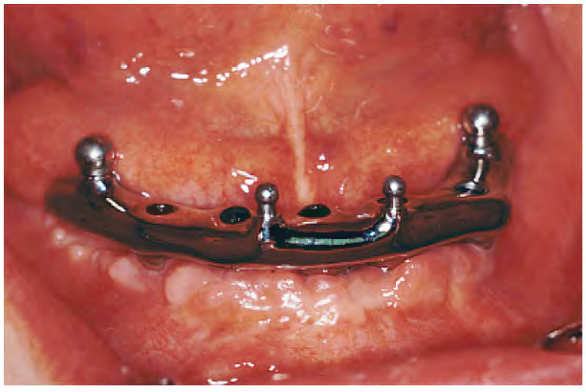


Figure 32-40 Long term, the tissue has remained attached around the implants.

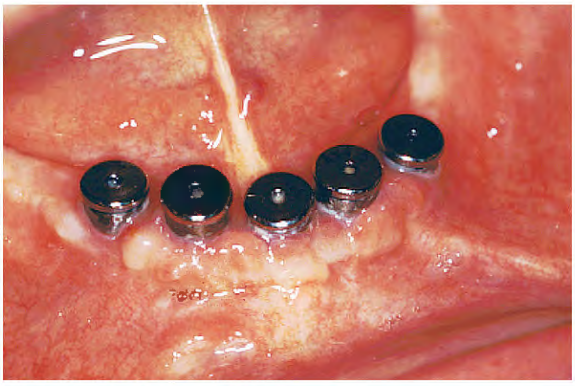


Figure 32-39 After several weeks, the tissue (although not keratinized) is not mobile around the implants.

Ono et al.[65] have proposed a classification of attached gingiva and surgical alternatives to improve soft tissue types in edentulous sites for implant placement. Meffert et al.[66] prefer to obtain keratinized tissue before implant placement.

Around natural teeth there are two main tissue types: attached, keratinized tissue and unattached, nonkeratinized tissue. These tissue types become more obscure around implants. When the tissue in a maxillary arch is thick before implant placement, the implant surgery often results with mobile, keratinized tissue that is several millimeters thick around the implant abutments. This type of tissue should be reduced in thickness to form a probing depth less than 4 mm.

When the implant is placed into a residual ridge that has lost overall width or had an iliac crest graft, the implant often has mobile, unattached nonkeratinized tissue in the facial aspect (Figure 32-38). This nonkeratinized tissue must become nonmobile, nonkeratinized tissue by placing either noncellular tissue graft (Alloderm) or hydroxyapatite below it (Figure 32-39). Although the tissue does not become keratinized with this technique, it does become nonmobile and provides a mass of tissue that may remain in health for the long term (Figure 32-40).

There are four types of oral mucosa that may be found around an implant: (1) keratinized, nonmobile tissue; (2) nonkeratinized, nonmobile tissue; (3) keratinized, mobile tissue; and (4) nonkeratinized, mobile tissue. In esthetic areas that expose the soft tissue, keratinized, nonmobile tissue is the best option. In nonesthetic regions, either keratinized, nonmobile, or nonkeratinized, nonmobile tissue is acceptable. Keratinized, mobile tissue may usually be made nonmobile by gingivoplasty techniques. Nonkeratinized, mobile tissue usually requires a soft tissue procedure to create a zone of keratinized tissue (i.e., free tissue graft) or a subdermal procedural to modify the mobile tissue base.

The width of keratinized tissue is evaluated before the initial incision and, if inadequate, is increased at the conclusion of the procedure. If less than 3 mm of keratinized gingiva is present and the crestal bone is cortical and in excellent condition around the implant bodies, the incision bisects the thin zone of attached tissue and the labial portion is approximated to the sole facial aspect of each implant. The lingual flap approximates the lingual aspect of each PME (Figure 32-41). Loose, interrupted figure-eight sutures are placed between the healing abutments. The section between the incision line is allowed to heal by secondary intention. In this manner, attached gingiva forms between the implants and the original attached tissue width is now divided between the facial and the lingual surfaces of the implants (Figure 32-42). The risk of this procedure involves the loss of crestal bone during soft tissue healing. The risk is reduced in the mandible, because the crestal bone is more cortical.

A second option to increase the zone of keratinized tissue at the stage II surgery is to use a split-thickness incision at the time of uncovery. After the implant body is evaluated (as previously addressed), a connective tissue or keratinized tissue graft harvested from the

WWW.HIGHDENT.IR

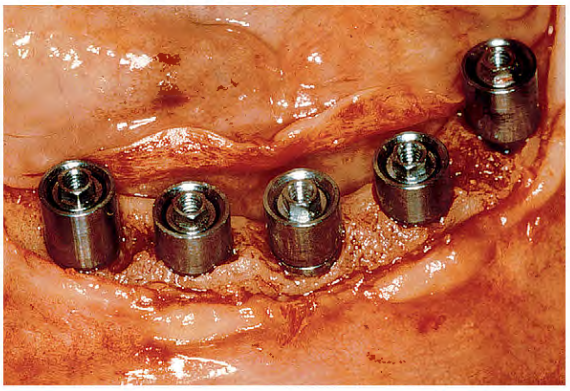


Figure 32-41 The quantity of keratinized tissue may be increased by allowing the margins of the tissue to granulate over the ridge.

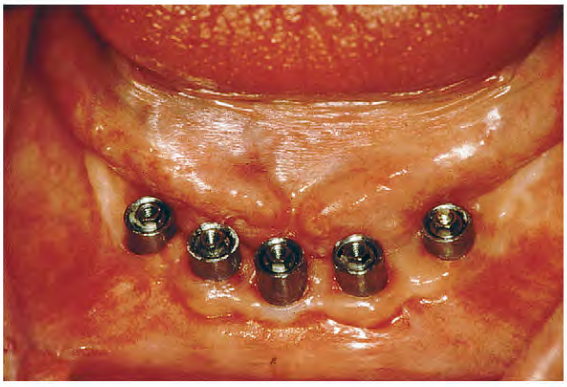


Figure 32-42 The tissue that heals by secondary intention between the margins of the original keratinized tissue creates attached, keratinized tissue around the implants.

palate may be placed and sutured around the stage II PMEs.

A third option to increase the zone of keratinized tissue is to harvest a 6- to 8-mm doughnut-shaped gingival collar from the palate. The open region of this collar is 4 mm in diameter and is inserted over the top of the PME and on top of the underlying bone or split-thickness incision.

A fourth option to increase a zone of mobile tissue is to place dense HA around the permucosal extension before approximating the tissues. This does not increase the zone of attached gingiva, but it makes the nonkeratinized tissue less mobile. However, this technique may also increase the gingival sulcus around the implant.

A fifth treatment option is to place an acellular tissue graft over the implants and directly onto the bone. The acellular tissue may be bound to the bone by tissue tacks. The overlying tissue is approximated around the implants. The tissue is then attached to acellular tissue by sutures or tissue tacks. The overlying tissue attaches to the acellular tissue, and the acellular tissue attaches to the bone. Although a keratinized tissue does not develop, the unkeratinized tissue is nonmobile. The nonmobile tissue has been observed to have benefits similar to nonmobile keratinized tissue.

TRANSITIONAL PROSTHESIS

A noneugenol periodontal dressing may be added over the top of the PMEs, but is usually not required. The complete denture or soft tissue–supported transitional prosthesis is aggressively relieved to prevent premature loading of the permucosal healing abutments. A tissue conditioner several millimeters thick is placed in the prosthesis over the implant healing abutment. The tissue conditioner is then relieved over the PMEs and in any region of desired secondary intention soft tissue healing. In nonesthetic regions of the partially edentulous patient, no transitional restoration is worn until after the sutures are removed.

The sutures are removed 2 weeks postoperatively. The soft tissue is not completely keratinized and healed at this time and may still be slightly inflamed. However, the patient may be referred to the restoring dentist for reconstruction. The final abutments will be selected by the restoring dentist after completion of the soft tissue maturation.

If the tissues appear healthy at the suture removal appointment, the first prosthetic appointment may be held at the same time. The stage II permucosal healing abutments are removed and replaced by the final abutment for screw retention or abutment for cement retention. An indirect impression transfer coping is placed into the abutment for screw retention. Vertical pressure is placed on the implants to ensure lack of tenderness. The surrounding gingiva may be recontoured by gingivoplasty with a diamond bur to shape an interdental papilla around the implant abutments for FP-1 prostheses or to reduce the depth of an implant sulcus.

A preliminary irreversible hydrocolloid or elastic impression is made of the implants. A preliminary impression made with additional silicone or polyether instead of irreversible hydrocolloid eliminates the need for the surgeon to have implant analogs or dental stone readily available. Instead, the impression is sent to the laboratory, where analogs and other materials are selected and used. The indirect impression transfer coping or abutment for cement retention is removed after the impression is made, and the stage II permucosal healing abutment is replaced.

The patient is scheduled to be seen again in 1 to 2 weeks, depending on the extent of soft tissue maturation at the suture removal. The indirect impression transfer coping or implant abutment analog (for screw or for cement) is placed into the preliminary impression, which is poured with dental stone. The purpose of this step is to permit the fabrication of the final impression tray and begin the fabrication of a transitional prosthesis if a fixed restoration is indicated.

WWW.HIGHDENT.IR

SUMMARY

Stage II surgery permits direct evaluation of the hard and soft tissues condition. The fabrication of the final prosthesis should begin with ideal abutment support. This includes an absence of crestal bone loss or defects, rigid fixation, no discomfort on loading, adequate zone of attached gingiva, pocket depths less than 5 mm, and acceptable implant body and abutment position for the intended prosthesis. Stage II surgery is an opportunity to evaluate these criteria and, if not present, restore them before prosthesis fabrication. On occasion, this requires additional time and surgeries. However, the alternative is a higher risk of future complication, which will require even more time, cost, and effort to correct or improve.

References

1. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
2. Kline R, Hoar JE, Beck GH et al: A prospective multicenter clinical investigation of a bone quality-based dental implant system, *Implant Dent* 11:332-334, 2002.
3. Zarb GA, Schmitt A: The longitudinal clinical effectiveness of osseointegrated dental implant: the Toronto study. Part 1: surgical results, *J Prosthet Dent* 63:451-457, 1990.
4. Naert I, Quirynen M: A six-year prosthodontic study of 509 consecutively inserted implants for the treatment of partial edentulism, *J Prosthet Dent* 67:236-245, 1992.
5. *Statistical results: implants lost between stage II surgery and prosthesis delivery,* Leawood, Kan, 1993, Root Laboratory.
6. Snauwaert K, Duyck J, van Steenberghe D et al: Time dependent failure rate and marginal bone loss of implant supported prostheses: a 15-year follow-up study, *Clin Oral Invest* 4:13-20, 2000.
7. Jividen G Jr, Misch CE: Reverse torque testing and early loading failures: help or hindrance? *J Oral Implantol* 26:82-90, 2000.
8. Brånemark PI: Osseointegration and its experimental background, *J Prosthet Dent* 50:399-410, 1983.
9. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 10:387-416, 1981.
10. Rosenquist B, Grenthe B: Immediate placement of implants into extraction sockets: implant survival, *Int J Oral Maxillofac Implants* 11:205-209, 1996.
11. Tal H: Spontaneous early exposure of submerged implants: classification and clinical observation, *J Periodontol* 70:213-219, 1999.
12. Tal H, Dayan D: Spontaneous early exposure of submerged implants: histopathology and histomorphometry of non-perforated mucosa covering submerged implants, *J Periodontol* 71:1224-1230, 2000.
13. Tal H, Dayan D: Spontaneous early exposure of submerged implants: histopathology of perforated mucosa covering submerged implants, *J Periodontol* 71:1231-1235, 2000.
14. Tal H, Artzi Z, Moses O et al: Spontaneous early exposure of submerged endosseous implants resulting in crestal bone loss: a clinical evaluation between stage I and stage II surgery, *Int J Oral Maxillofac Implants* 16:514-521, 2001.
15. Toijanic JA, Banakis ML, Willes LA et al: Soft tissue exposure of endosseous implants between stage I and stage II surgery as a potential indicator of early crestal bone loss, *Int J Oral Maxillofac Implants* 14:436-441, 1999.
16. Ericsson I, Randow K, Nilner K et al: Some clinical and radiographical features of submerged and non-submerged titanium implants: a 5-year follow-up study, *Clin Oral Impl Res* 8:422-426, 1997.
17. Barboza EP, Caula AL, Carvalho WR: Crestal bone loss around submerged and exposed unloaded dental implants: a radiographic and microbiological descriptive study, *Implant Dent* 11:162-169, 2002.
18. Rams TE, Link CC Jr: Microbiology of failing dental implants in humans: electron microscopic observations, *J Oral Implantol* 11:93-100, 1983.
19. Sullivan DY, Sherwood RL, Collins TA et al: The reverse torque test—a clinical report, *Int J Oral Maxillofac Implants* 11:179-185, 1996.
20. Sennerby L, Thomsen P, Ericson L: A morphometric and biomechanic comparison of titanium implants inserted in rabbit cortical and cancellous bone, *Int J Oral Maxillofac Implants* 7:62-68, 1992.
21. Tjellstrom A, Jacobsson M, Albrektsson T: Removal torque of osseointegrated craniofacial implant: a clinical study, *Int J Oral Maxillofac Implants* 3:287-289, 1988.
22. Johnson C, Albrektsson T: Integration of screw implants in the rabbit: a 1-year follow-up of removal torque of titanium implants, *Int J Oral Maxillofac Implants* 2:69-73, 1987.
23. Carlsson L, Rostlund T, Albrektsson B et al: Removal torques for polished and rough titanium implants, *Int J Oral Maxillofac Implants* 3:21-24, 1988.
24. Carr AB, Larson PE, Papazoglou E et al: Reverse torque failure of screw shaped implants in baboons. Baseline data for abutment torque application, *Int J Oral Maxillofac Implants* 10:167-174, 1995.
25. Gotfredsen K, Nimb L, Hgorting-Hansen R et al: Histomorphometric and removal torque analysis for TiO_2-blasted titanium implants: an experimental study on dogs, *Clin Oral Implant Res* 3:77-84, 1992.
26. Carr AB, Papazoglou E, Larsen PE: The relationship of Periotest values, biomaterial and torque to failure in adult baboons, *Int J Prosthodont* 8:15-20, 1995.
27. Steigenga J, Al-Snammari K, Misch C et al: Effects of implant thread geometry on percentage of osseointegration and resistance to reverse torque in the tibia of rabbits, *J Periodontol* 75:1233-1241, 2004.
28. Cordioli G, Majzoub Z, Piattelli A et al: Removal torque and histomorphometric investigation of 4 different titanium surfaces: an experimental study in the rabbit tibia, *Int J Oral Maxillofac Implants* 15:68-74, 2000.
29. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, *Postgrad Dent* 2:3-16, 1995.
30. Truhlar RS, Morris HF, Ochi S et al: Second stage failures related to bone quality in patients receiving endosseous dental implants. DICRG interim report #7, *Implant Dent* 3:252-255, 1994.
31. Albrektsson T, Jacobsson M: Bone-metal interface in osseointegration, *J Prosthet Dent* 57:587-607, 1987.
32. Brånemark PI: Introduction to osseointegration. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue-*

WWW.HIGHDENT.IR

integrated prostheses: osseointegration in clinical dentistry, Chicago, 1985, Quintessence.
33. Roberts WE, Helm FR, Marshall KJ et al: Rigid endosseous implants for orthodontic and orthopedic anchorage, *Angle Orthod* 59:247-256, 1989.
34. Sekine H, Komiyama Y, Hotta H et al: Mobility characteristics and tactile sensitivity of osseointegrated fixture supporting systems. In van Steenberghe D, editor: *Tissue integration in oral maxillofacial reconstruction,* Amsterdam, 1996, Elsevier.
35. Misch CE: Bone density, its effect on treatment planning, surgical approach, healing and progressive loading, *Int J Oral Implantol* 6:23-31, 1990.
36. Cowin SL: *Bone mechanics,* Boca Raton, Fla, 1989, CRC Press.
37. Carter DR, Schwab GH, Spengler DM: Tensile fracture of cancellous bone, *Acta Orthop Scand* 5:733, 1980.
38. Appleton RS, Nummikoski PV, Pigmo M et al: *Peri-implant bone changes in response to progressive osseous loading,* Orlando, Fla, 1997, IADR Abs.
39. Rotter BE, Blackwell R, Dalton G: Testing progressive loading of endosteal implants with the Periotest, a pilot study, *Implant Dent* 5:28-32, 1996.
40. van Steenberghe D, Quirynen M: Reproducibility and detection threshold of peri-implant diagnostics, *Adv Dent Res* 7:191-195, 1993.
41. Schulte W, Lukas D: The Periotest method, *Int Dent J* 42:433-440, 1990.
42. Teerlinck J, Quirynen M, Darius P et al: Periotest: an objective clinical diagnosis of bone apposition toward implants, *Int J Oral Maxillofac Implants* 6:55-61, 1991.
43. Olive J, Aparicio C: The Periotest method as a measure of osseointegrated implant stability, *Int J Oral Maxillofac Implants* 5:390-400, 1990.
44. Tricio J, Laohapand P, van Steenberghe D et al: Mechanical state assessment of the implant-bone continuum, a better understanding of the Periotest method, *Int J Oral Maxillofac Implants* 10:43-49, 1995.
45. Truhlar RS, Morris HF, Ochi S et al: Assessment of implant mobility at second stage surgery with the Periotest, DICRG interim report #3, *Implant Dent* 3:153-156, 1994.
46. Misch CE: Unpublished data, 1996.
47. Alsaadi G, Quirynen M, Michiels K et al: A biomechanical assessment of the relation between the oral implant stability at insertion and subjective bone quality assessment, *J Clin Periodontol* 34:359-366, 2007.
48. Noguerol B, Munoz R, Mesa F et al: Early implant failure. Prognostic capacity of Periotest: retrospective study of a large sample, *Clin Oral Implants Res* 17:459-464, 2006.
49. Lachmann S, Laval JY, Jager B et al: Resonance frequency analysis and damping capacity assessment. Part 2: peri-implant bone loss follow-up. An in vitro study with the Periotest and Osstell instruments, *Clin Oral Implants Res* 17:80-84, 2006.
50. Newman MG, Flemming TF: Periodontal considerations of implants and implants associated microbiota, *J Dent Educ* 52:737-744, 1988.
51. Berglundh T, Lindhe J, Marinello C et al: Soft tissue reaction to de novo plaque formation on implants and teeth. An experimental study in the dog, *Clin Oral Impl Res* 3:1-8, 1992.
52. Yukna RA: Periodontal considerations for dental implants. In Block MS, Kent JN, editors: *Endosseous implants for maxillofacial reconstruction,* Philadelphia, 1995, WB Saunders.
53. Misch CE: The maxillary anterior single tooth implant aesthetic-health compromise, *Int J Dent Symp* 3:409, 1995.
54. Lang NP, Loe H: The relationship between the width of keratinized gingiva and gingival health, *J Periodontol* 43: 623-627, 1972.
55. Wennstrom JL: Lack of association between width of attached gingiva and development of soft tissue recession: a 5-year longitudinal study, *J Clin Periodontol* 14:181-184, 1987.
56. Kennedy J, Bird W, Palcanis K et al: A longitudinal evaluation of varying widths of attached gingiva, *J Clin Periodontol* 12:667, 1985.
57. Stetler K, Bissada NF: Significance of the width of keratinized gingiva on the periodontal status of teeth with submarginal restoration, *J Periodontol* 58:696-700, 1987.
58. Schroeder A, Pohler O, Sutter F: Tissue reaction to a titanium hollow cylinder implant with titanium plasma sprayed surface, *Schweiz Monatsschr Zahnmed* 86:713-727, 1976.
59. Warrer K, Buser D, Lang NP et al: Plaque-induced peri-implantitis in the presence or absence of keratinized mucosa: an experimental study in monkeys, *Clin Oral Implants Res* 6:131-138, 1995.
60. Kirsch A, Ackermann KL: The IMZ osteointegrated implant system, *Dent Clin North Am* 33:733-791, 1989.
61. Adell R, Lekholm U, Rockler G et al: Marginal tissue reactions at osseointegrated titanium fixtures. I. A 3-year longitudinal prospective study, *Int J Oral Maxillofac Implants* 15:39-52, 1986.
62. Nevins M, Kenney E, van Steenberghe D et al, editors: Consensus report: implant therapy II. Proceedings of the 1996 World Workshop in Periodontics, *Ann Periodontol* 1:816-820, 1996.
63. James RA, Schultz RL: Hemidesmosomes and the adhesion of junctional epithelial cells to metal implants: a preliminary report, *J Oral Implantol* 4:294, 1974.
64. Listgarten M, Lang NP, Schroeder HE et al: Periodontal tissues and their counterparts around endosseous implants, *Clin Oral Implants Res* 2:81-90, 1991.
65. Ono Y, Nevins M, Cappetta M: The need for keratinized tissue for implants. In Nevins M, Mellonig JT, editors: *Implant therapy,* Chicago, 1998, Quintessence.
66. Meffert RM, Langer B, Fritz ME: Dental implants: a review, *J Periodontol* 63:859-870, 1992.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 33

Anterior Single-Tooth Replacement: Surgical Considerations

Carl E. Misch

Single-tooth replacement will most likely comprise a larger percentage of prosthetic dentistry in the future, compared with past generations. In 1960 the average American older than 55 years had just seven original teeth. Today, the baby boomers (those born between 1946 and 1964) can expect to have at least 24 teeth left when they reach 65 years of age.

Contrary to missing a posterior tooth, most patients have an emotional response regarding a maxillary anterior missing tooth. No question exists regarding the need to replace the tooth, and financial considerations are less important. When posterior teeth are extracted, little resistance to the preparation of adjacent teeth may be given to the dentist. However, when anterior, normal-looking teeth must be prepared to serve as fixed partial denture (FPD) abutments, the patient is more anxious and often looks for an alternative. In the patient's perspective, anterior FPD restorations are never as esthetic as natural teeth. This in part is because they are able to distinguish between good and poor esthetic results. More important, because patients are only able to notice the restorations that are not natural in appearance, they think anterior FPDs are not esthetic.

In younger patients with congenitally missing maxillary lateral incisors or with trauma to the maxillary central incisor (which resulted in its failure, often after endodontic therapy), the parents are eager to provide the best possible replacement option. They often perceive this option to be a single-tooth implant. As a consequence of these psychological factors, a common site for a single-tooth implant in a restorative practice is the maxillary central or lateral incisor.

Single-tooth implants are now one of the most common implant procedures performed in the United States. In the nonesthetic posterior region, the single-tooth implant is one of the simplest procedures in implant surgery and prosthetics. However, it should be noted the maxillary central incisor single-tooth replacement is often the most difficult procedure in all of implant dentistry.

The highly esthetic zone of the premaxilla often requires both hard (bone and teeth) and soft tissue restoration. The soft tissue drape is often the most difficult aspect of treatment. As a consequence, maxillary anterior single-tooth replacement is often a challenge, regardless of the experience and skill of the dentist or dentists.

ALTERNATE TREATMENT OPTIONS FOR ANTERIOR SINGLE-TOOTH REPLACEMENT

The alternatives to restore a single, maxillary anterior missing tooth include a traditional FPD; a cantilever FPD (for a missing lateral incisor); a removable partial denture (RPD); an acid-etched, resin-bonded prosthesis; or a single implant–supported restoration. These options were presented in Chapter 16. Maxillary single-tooth replacement is one of the most challenging restorations in dentistry. However, in light of all the advantages of single-implant longevity, bone maintenance, reduced abutment teeth complications, and increased survival of adjacent teeth, single-tooth implants have become the treatment of choice.

The single-tooth implant has the highest success rate compared with any other treatment option to replace missing teeth with an implant restoration (e.g., overdentures, short-span FPD, full-arch FPD, single-tooth implant).[1-11] In 2005, Misch et al.[7] reported on 276 anterior maxillary single implants used to restore missing teeth from agenesia. In 255 adolescent patients, the implants were monitored for a range of 2 to 16 years, with a 98.6% implant and crown survival rate. In the same year, Wennstrom et al.[8] reported on a 5-year prospective study with 45 single-tooth implants, with a 97.7% implant survival rate with minimal bone loss.

WWW.HIGHDENT.IR

In 2006, Zarone et al.[11] reported on lateral maxillary agenesis replacement with 34 implants, with a 97% survival rate at 39 months. A review of the literature by Goodacre et al.[6] found single-tooth implant studies had the highest survival rate of any prosthesis type and averaged 97%.

More recently, a trend toward single-stage and immediate-extraction implants has emerged, appearing especially attractive in the maxillary anterior region, where preferably the soft tissue drape is present before the tooth extraction and patients are more anxious to get a fixed replacement. Kemppainen et al.,[12] in a prospective study of 102 single-tooth implants in the anterior maxilla, reported a 99% success rate using one-stage and two-stage implants. Other studies have recommended one stage and immediate load with some success in specific situations.[13-15]

Figure 33-1 A single missing central incisor is often the most challenging implant restoration. The soft and hard tissues should be perfect to obtain an acceptable result.

Figure 33-2 The maxillary left central incisor implant crown in position. The soft tissue drape is established through both surgical and prosthetic methods.

CHALLENGING ESTHETICS

The esthetics of a maxillary anterior single crown on a natural tooth is often one of the most difficult challenges in restorative dentistry. It is even greater on an implant abutment (Figures 33-1 and 33-2). The implant is often 5 mm or less in diameter and round in cross section. A natural maxillary anterior crown cervix region is 4.5 to 7 mm in mesiodistal cross section and is never completely round. In fact, the natural central incisor and canine teeth are often larger in their faciopalatal dimension at the cement-enamel junction (CEJ) than in the mesiodistal dimension. Because the bone is lost first in the faciopalatal width, the greater width of implants in this dimension would require even greater augmentation than presently advocated. As a result, the cervical esthetics of a single-implant crown must accommodate a round-diameter implant and balance hygiene and esthetic parameters. Additional prosthetic steps and components with varied emergence profiles or customized tooth-colored abutments are often required to render the illusion of a crown on a natural abutment.

Bone Height

The available bone should be closely evaluated because it will greatly influence the soft tissue drape, implant size, implant position (angulation and depth), and ultimately the final esthetic outcome. Hard tissue topography is a prerequisite to an optimal, esthetic implant restoration. Available bone volume is necessary, and the position of the osseous crest is specific. The model midcrestal position of the edentulous site should be 2 mm below the facial CEJ of the adjacent teeth. On occasion, the bone crest may be above this position when the interproximal bone height is higher and/or a bone graft was performed at extraction. The interproximal bone should be scalloped 3 mm more incisal than the midcrestal position.

The position of the interproximal crest of bone is an important anatomical consideration, especially for the development of the interproximal soft tissue height.[16,17] Becker et al.[18] classified the range of interproximal bone height above the midfacial scallop from less than 2.1 mm (flat) to scalloped (2.8 mm) to pronounced scalloped (<4.1 mm). The flat anatomy should correspond to a square-shaped tooth, the scalloped to an ovoid-shaped tooth, and the pronounced scalloped to a triangular-shaped tooth (Figure 33-3). However, these relationships do not always exist. When a flat interdental-to-crest dimension is found on triangular teeth, the interproximal space will usually not be filled with soft tissue, because the dimension of the interproximal contact to the bone will be greater than 5 mm (Figure 33-4).[21]

Often the osseous crest may be more apical than ideal, in both the implant site and the adjacent tooth

WWW.HIGHDENT.IR

Figure 33-3 A triangular tooth form corresponds to the greatest width and height of interdental papillae and the most incisal interproximal contact position on the crown.

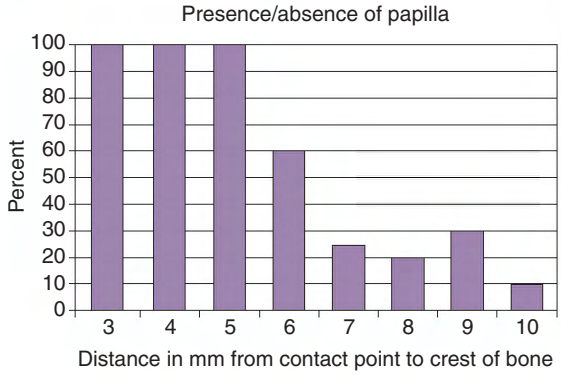


Figure 33-4 When natural teeth have an interproximal crown contact to interseptal bone distance of 5 mm or less, the interdental papilla almost always fills the interproximal space. When the distance is 6 mm, the interproximal space is not filled with soft tissue almost 40% of the time; at 7 mm the interproximal space is filled with an interproximal papilla 25% of the time.

roots. Under these conditions, ideal crown contour, soft tissue emergence, and interproximal tissue conditions are less likely (Figures 33-5 and 33-6). Bone and soft tissue changes after maxillary anterior tooth loss are rather rapid and of considerable consequence. As a result, many maxillary anterior edentulous sites require at least some bone and/or soft tissue modification before, in conjunction with, and/or at implant uncovery. Under perfect conditions, the implant body should not be inserted until the bone and soft tissue are within normal limits.

Mesiodistal Space

An adequate mesiodistal space is necessary for an esthetic outcome of an implant restoration and the interproximal soft tissue of the adjacent teeth. A traditional two-piece implant should be at least 1.5 mm from an adjacent tooth. When the implant is closer than this to an adjacent tooth, any bone loss related to

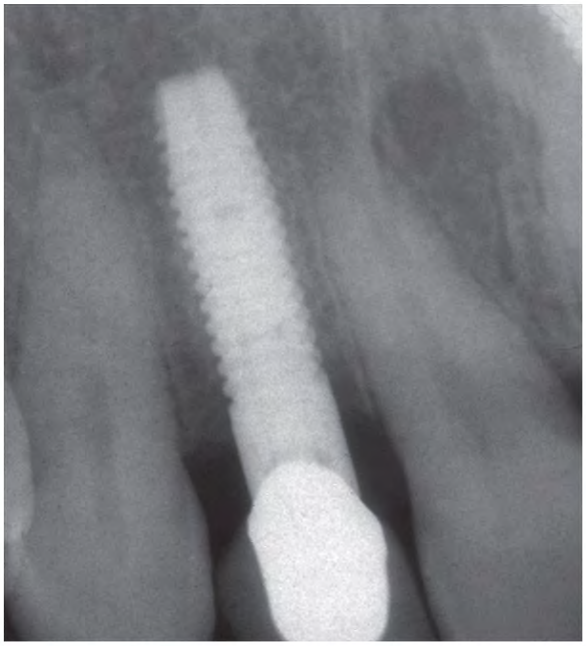


Figure 33-5 A periapical radiograph of an implant and crown placed in the maxillary right lateral incisor region. The interproximal bone adjacent to the canine is ideal; the interproximal bone on the central incisor is 4 mm below the cement-enamel junction (CEJ).

Figure 33-6 The lateral incisor implant crown from Figure 33-5. The interproximal distal papilla is acceptable as a result of the canine crestal bone level. The mesial papilla is depressed, and a "black hole" is evident. This is related to the crestal bone level on the central incisor. In addition, the triangular tooth form translates into a more incisal interproximal crown contact position, which further enhances the triangular defect. The distal bone loss on the central also increases the risk of root exposure (as shown).

the microgap, the biological width, and/or stress might cause the implant and adjacent tooth to lose bone. This may compromise interproximal esthetics and/or sulcular health of the implant and natural tooth[19-21] (Figure 33-7).

The smallest-diameter implant body offered by most commercial companies is 3.2 mm. However, the crest module of these two-piece implants is usually 3.5 mm or more. Therefore the mesiodistal edentulous space for a two-piece implant should be 6.5 mm or greater.

WWW.HIGHDENT.IR

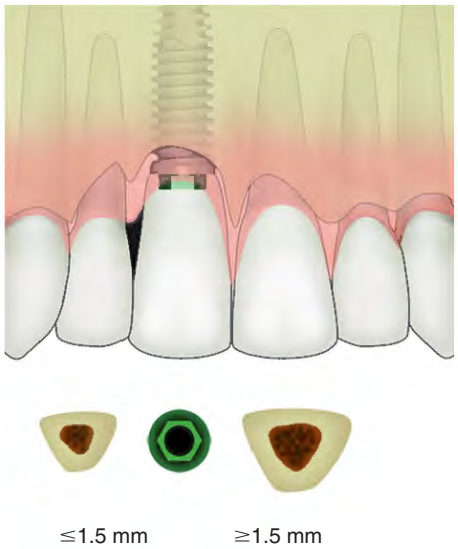


Figure 33-7 If bone loss occurs on an implant placed closer than 1.5 mm to a tooth (on the distal), then bone will also be lost on the tooth. As a result, the distance from the interproximal crown contact to the interproximal bone increases, and the risk of soft tissue shrinkage and loss of interdental papilla increases. When the implant is greater than 1.5 mm from the tooth (on the mesial); bone loss on the implants does not cause bone loss on the tooth root. The interproximal crown contact to interproximal bone relationship remains ideal, and the interdental papilla is maintained.

One-piece dental implants may be fabricated in 2.5- to 3.0-mm[22-24] diameters to accommodate a reduced mesiodistal dimension criterion. These implant designs do not have a microgap, and the vertical defect is narrower than most two-piece implant systems. As such, they may be placed as close as 1 mm from an adjacent tooth, and therefore can accommodate a 5-mm mesiodistal missing tooth space. However, because these implants have the abutment post attached to the implant body, they must be immediately restored the day of surgery. These implants are addressed in Chapter 35.

Faciopalatal Width

Most of the conditions that lead to single-tooth loss result in the loss of some or all of the facial bone in the region of the missing tooth. In addition, a 25% decrease in faciopalatal width occurs within the first year of tooth loss and rapidly evolves into a 30% to 40% decrease within 3 years. As a result, even an intact alveolus 6 to 8 mm wide is often inadequate in width after 1 year for a Division A root form implant in a central incisor position, and after 3 years it almost never presents adequate available bone for the proper-size implant. The bone width loss is primarily from the facial region, because the labial plate is very thin compared with the palatal plate, and facial undercuts are often found over the roots of the teeth (Figure 33-8). A bone graft is often necessary to restore the proper anatomy of the ridge and to avoid a compromised implant position more palatal and apical.

The amount of available bone width (faciopalatal) should be at least 2.0 mm greater than the implant diameter at implant insertion. Therefore a 3.5-mm implant requires at least 5.5 mm of bone width. Bone augmentation in width is very predictable. In many instances it is performed before implant placement; however, it may be performed at the time of implant insertion, especially when no dehiscence of the implant is visible. It should be emphasized the implant diameter measurement is at the crest module of the implant. Most 3.75-mm diameter implant bodies are 4.1 mm at the crest module. In these situations, the mesiodistal limitation is 7.1 mm and the faciolingual width limitation is 6.1 mm.

Implant Size

The first factor that influences the size of an implant is the mesiodistal dimension of the missing tooth. The average mesiodistal dimension of a central incisor is 8.6 mm for a man and 8.1 mm for a woman, a lateral

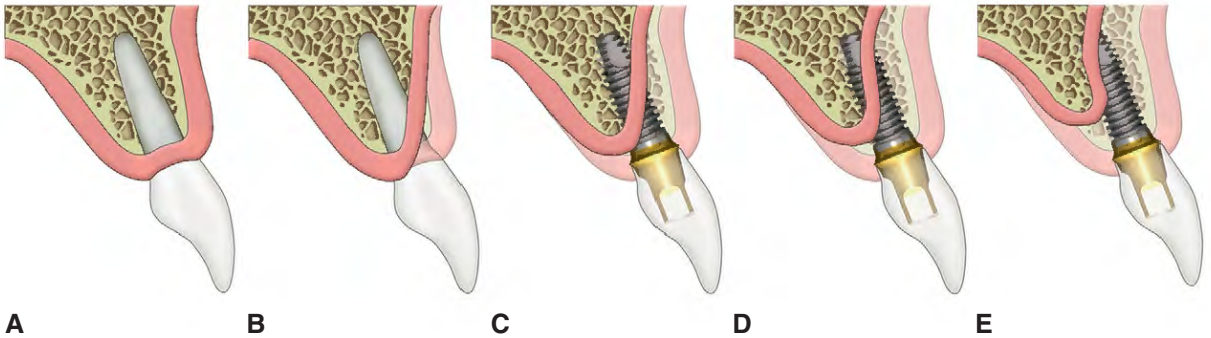


Figure 33-8 Before the loss of a maxillary anterior tooth, the bone around the roots most often is present **(A).** The cause of extraction often causes a loss of the thin labial plate of bone over the root **(B).** After extraction, the residual ridge most often is decreased in width (Division B). After 6 months to 1 year, the residual ridge continues to resorb and becomes Division B–w **(C).** Eventually, the residual ridge forms a C–w bone volume that is slightly deficient in height and less than 2.5 mm in width **(D).** This bone volume often extends almost to the floor of the nose **(E).**

WWW.HIGHDENT.IR

Table 33-1 Maxillary Teeth Dimensions

TYPE OF TOOTH	MESIODISTAL CROWN (mm)	MESIODISTAL CERVIX (mm)	FACIOLINGUAL CROWN (mm)	FACIOLINGUAL CERVIX (mm)	2 mm BELOW CEMENT-ENAMEL JUNCTION
Central incisor	8.6	6.4	7.1	6.4	5.5
Lateral Incisor	6.6	4.7	6.2	5.8	4.3
Cuspid	7.6	5.6	8.1	7.6	4.6

incisor is 6.6 mm for a man and 6.1 mm for a woman, and a canine is 7.6 mm for a man and 7.2 mm for a woman. Obviously, the implant body should not be as wide as the natural tooth or clinical crown. Otherwise, the emergence contour and interdental papillae region cannot be properly established.

The mesiodistal dimensions of the maxillary central incisor at the cervix (preferably 1 mm below the free gingival margin) averages 6.4 mm, the lateral incisor dimension is 4.7 mm, and canine natural teeth at the cervix are 5.6 mm (Table 33-1).[25,26] However, these dimensions are also too large for an implant.

The bone level on natural teeth is 2 mm below the CEJ; the natural tooth dimensions at this bone level are reduced to 5.5 mm for central incisors, 4.3 mm for lateral incisors, and 4.6 mm for canines. Therefore in theory, the latter dimensions most closely resemble the consummate implant diameter to mimic the emergence profile of a natural tooth. However, this dimension is usually too large to adequately restore the soft tissue drape of the missing anterior tooth.

The second factor that determines the mesiodistal implant diameter is the necessary distance from an adjacent tooth root.[20] Initial vertical bone loss around an implant during the first year of loading is variable and ranges from 0.5 to more than 3.0 mm.

The height of the interseptal (interimplant) bone in part determines the incidence of presence or absence of the interdental papillae between the teeth.[21] When the distance from the interseptal bone to interproximal contact is 5 mm or less, the papilla fills the space. When the distance is 6 mm, a partial absence of papilla is seen 45% of the time, and at 7 mm the risk of a compromise in the interproximal space is 75%. Therefore the intraseptal bone height is relative to the maintenance of the interdental papilla and should be preserved. As a consequence, the implant should be at least 1.5 mm from the adjacent teeth whenever possible, and the interseptal bone on the adjacent teeth should be within 5 mm of the desired interproximal crown contact position.

In summary, two mesiodistal parameters determine the preferable implant size. The suggested width of the single-tooth implant should correspond to the width of the missing natural tooth, 2 mm below the CEJ. The distance between the roots of the adjacent teeth should also be measured. The implant diameter + 3 mm (1.5 mm on each side) should be equal to or less than the distance between the adjacent roots, at the crest of the ridge (which is 2 mm below the interproximal CEJ).

The next dimension that determines the width of an anterior implant is the faciopalatal dimension of bone. The width of bone should allow at least 1.5 mm on the facial aspect of the implant so that if a vertical defect forms around the crest module, then that defect would not become horizontal and change the cervical contour of the facial gingival. Because of its initial reduced volume, facial bone tends to be labile, and its resorption is responsible for most of the compromised long-term esthetic results in the anterior maxilla.[27-29] The faciopalatal width dimension is not as critical on the palatal aspect of the implant, because it is dense cortical bone, more resistant to bone loss, and not within the esthetic zone. Facial bone grafting at the time of implant insertion is frequently needed, because the bone volume in width is often compromised (Figures 33-9 to 33-11).

The width of the implant should not only mimic the emergence of a natural tooth, but also help to preserve the bone and health of the adjacent teeth. The natural intraroot distance of the two central incisors distance is approximately 2 mm. However, the natural roots of the central to lateral and lateral to canine are usually

Figure 33-9 An implant placed in the central incisor position, with less than 1.5 mm of bone on the facial.

WWW.HIGHDENT.IR

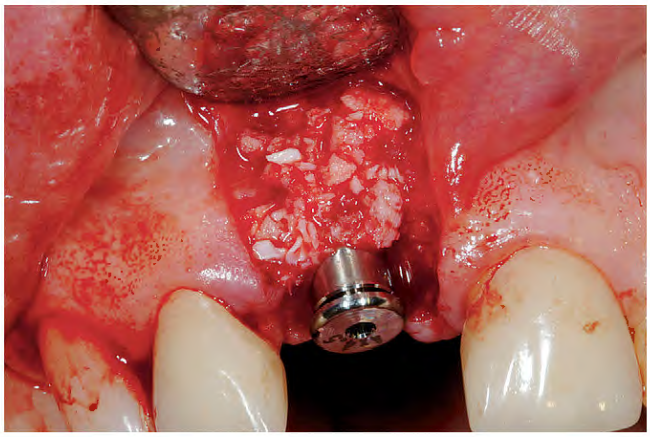


Figure 33-10 A layer of freeze-dried bone (MinerOss) is placed over the autograft on the facial bone over the implant.

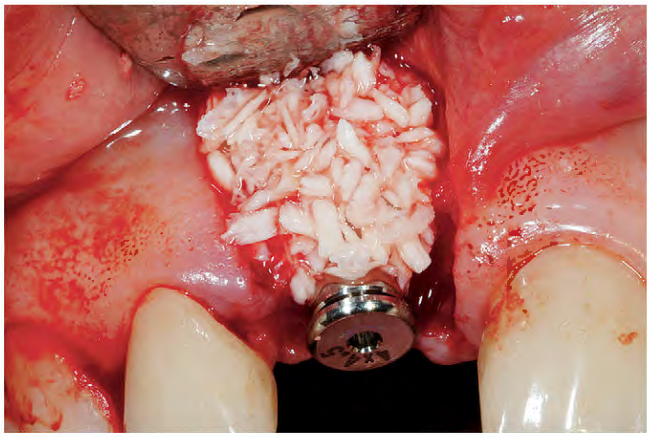
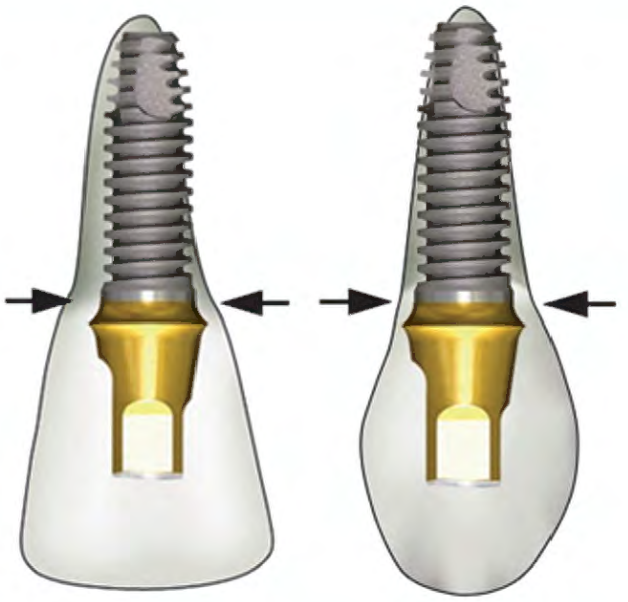


Figure 33-11 A layer of autograft, collected from the drills during osteotomy preparation, is placed on the facial bone over the implant.

less than 1.5 mm apart and often only 0.5 mm of space exists between them. As a consequence, the archetypal size of the single-tooth implant is usually smaller in diameter than the natural tooth root.

The typical diameters of the implant used to replace the average-size tooth often results in a 4.0- to 5.2-mm implant for a central incisor, a 3.0- to 3.5-mm implant for a lateral incisor, and a 3.7- to 4.2-mm implant for a canine. The difference in the emergence profile of a 4-mm-diameter implant and a 5-mm-diameter implant is negligible and often not clinically relevant for an anterior tooth, because 0.5-mm difference occurs on each side of the implant. Therefore when in doubt, the surgeon should use a smaller-diameter implant. As such, a 4-mm-diameter implant may often be used in the central-implant position for a single-tooth replacement. Likewise, a 3.0- to 3.5-mm implant is often used for a lateral incisor single-tooth restoration.



A B

Figure 33-12 The best implant position is under the incisal edge of the final crown, or slightly more palatal **(A).** The ideal mesiodistal implant position for a central incisor is 0.5 to 1.0 mm more distal than the midtooth position. This decreases the risk of encroachment on the incisive canal **(B).** The best mesiodistal position for a cuspid is centered in the cuspid position **(C).**

IMPLANT BODY POSITION

Mesiodistal Position

The maxillary anterior single-tooth implant should be located precisely in three planes. From a mesiodistal aspect, the implant most often is placed in the middle of the space, with equal amount of interproximal bone toward each adjacent tooth. On occasion, the central incisor implant is positioned slightly to the distal of the intratooth space (Figure 33-12). The incisive foramen is variable in size and position. When a central incisor implant is planned and the foramen between the existing central incisor root and implant site is larger than usual, the remaining bone may be inadequate. The foramen may also expand off to one side of the midline within the bony canal. When the central incisor implant is placed, the implant may encroach on the canal and result in a probing pocket depth of 10 mm or greater on the mesiopalatal surface of the implant. As a precaution, the surgeon should reflect the palatal tissue when placing a maxillary central incisor implant, probe the foramen, and if necessary, place the implant in a more distal position. This usually requires a smaller-diameter implant than usual to remain 1.5 mm or more from the lateral incisor. On occasion, the contents of the foramen must be removed and a bone graft inserted, to decrease the size of the incisive canal (Figure 33-13).

WWW.HIGHDENT.IR

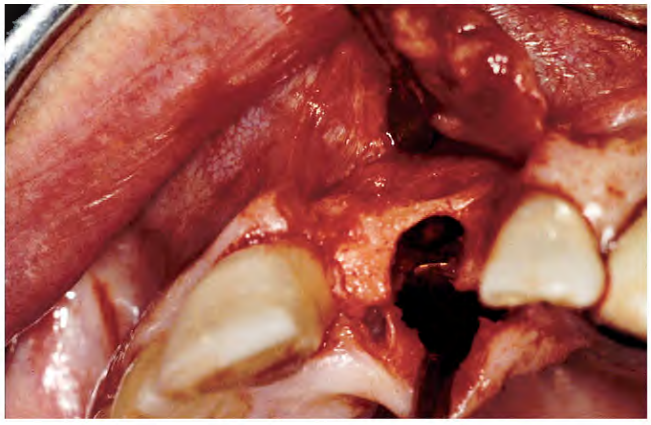


Figure 33-13 On occasion, the incisive canal extends into the site of the missing central incisor. As a result, the foramen should be enucleated and filled with a bone graft to restore the site and allow implant placement.

Faciopalatal Position

The midfaciopalatal position of the implant is in the middle to slightly palatal 0.5 mm of the edentulous ridge of adequate contour. This approach permits the use of the greatest-diameter implant. The crestal bone should be at least 1.5 mm wider on the facial aspect of the implant and 0.5 mm on the palatal aspect. Therefore for a 4-mm-diameter implant, a minimum 6-mm faciopalatal width of bone is required for the central or canine position, and 5.5 mm of bone width is required for a lateral incisor with a 3.5-mm implant. Bone spreading in conjunction with implant placement or bone grafting on the facial aspect of the edentulous site may be used when the ridge is less wide than is desirable. The thickness of bone on the facial aspect of a natural root is usually 0.5 mm thick. As a result, the implant is 1 mm or more palatal than the facial emergence of the adjacent crowns at the free gingival margin.

Implant Angulation

The implant center is positioned in the faciopalatal center of the edentulous ridge and the midmesiodistal position. The implant body angulation from this point is considered next. In the literature, three faciopalatal angulations of the implant body are suggested: (1) a facial angulation so that emergence of the final crown will be similar to adjacent teeth, (2) under the incisal edge of the final restoration, and (3) within the cingulum position of the implant crown (Figure 33-14).

Facial Implant Body Angulation

Researchers often theorize that a maxillary anterior implant body angulation be positioned at the facial emergence of the final crown. The facial implant position is predicated on the concept that the facial emergence of the implant crown at the cervical should be in the same position as a natural tooth. At first, this makes some sense. However, the crown of a natural tooth has two planes, and its incisal edge is palatal to the facial emergence of the natural tooth by 12 to 15 degrees (Figure 33-15). This is why anterior crown preparations are in two or three planes. The implant body is more palatal than a natural root, so 1.5 mm of bone exists facially. In addition, because the implant is narrower in diameter than the faciopalatal root dimension, when the implant body is oriented as a natural tooth and has a facial emergence, a straight abutment is not wide enough to permit the two or three plane reduction to bring the incisal edge of the preparation more palatal. As a result, the incisal edge of the preparation remains too facial. Therefore when the implant is angled to the facial emergence of a tooth, an angled abutment of 15 degrees must be used to bring the incisal edge more palatal.

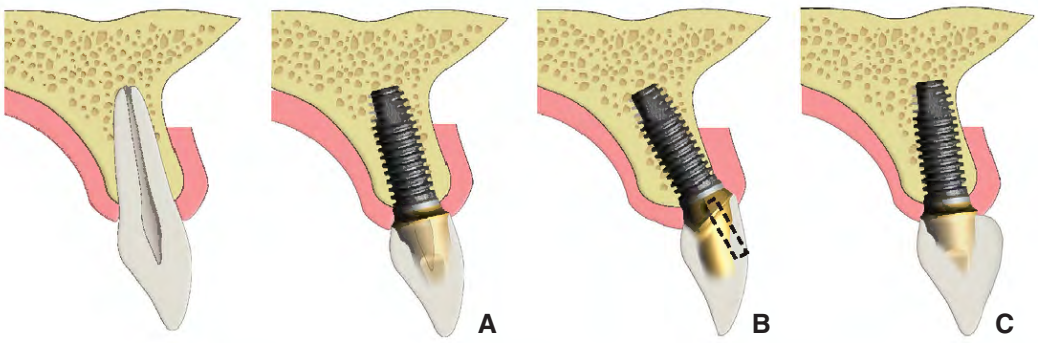


Figure 33-14 Three implant positions are found in the literature related to the final crown position. **A,** A position below the incisal edge is best used for a cemented crown in the esthetic zone. **B,** An implant is in the position of the natural root of the tooth. Although this makes sense, it places the implant too facial, and an angled abutment is usually necessary. **C,** An implant in the cingulum position that is used when a screw-retained crown is the treatment of choice. This position requires a facial ridge lap of porcelain when used for FP-1 prostheses in the esthetic zone.

WWW.HIGHDENT.IR

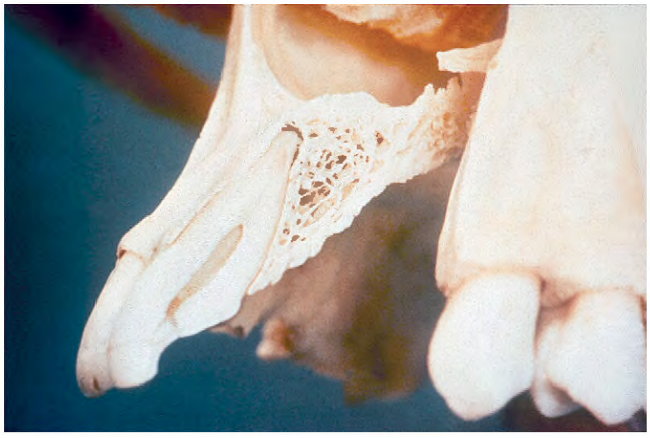


Figure 33-15 A natural tooth has very thin facial cortical bone over the root, and the incisal edge of the crown is 12 to 15 degrees palatal to the facial emergence profile. This is not an ideal position for an implant. The bone to the palatal region is better suited for an implant and allows the implant to be positioned under the incisal edge.

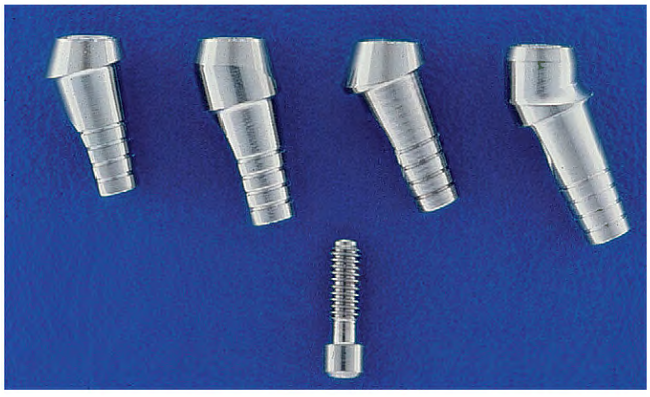


Figure 33-16 Most angled implant abutments, regardless of manufacturer, have an extra metal flange, opposite to the angled post. This increases the thickness of metal so that the component will not fracture. However, the extra metal flange is more facial than the implant body and results in increased risk of tissue shrinkage and/or metal seen through the thin facial soft tissue.

Most two-piece angled abutments have a design flaw that compromises facial cervical esthetics (Figure 33-16). The metal flange facial to the abutment screw is thinner than a straight abutment and may result in fracture (especially because angled loads are placed on the facial-positioned implant). The manufacturers thicken the profile of the abutment on the facial aspect to reduce the risk of fracture. However, this design flaw brings the cervical facial margin more facial and wider than the implant body, which is already as facial as the adjacent tooth. As a result, the implant crown margin is facially overcontoured. The restoring doctor then has to prepare the facial aspect of the abutment metal flange for esthetics, which weakens it and makes it prone to fracture. Because the doctor modified the abutment, the implant company is held harmless but, nonetheless, the patient and doctor are at risk.

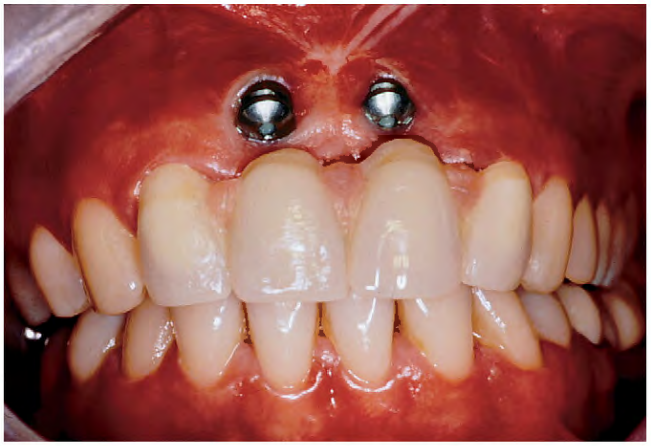


Figure 33-17 When implants are placed too facial, no prosthetic method is available to improve the poor esthetic condition.

When the surgeon attempts to align the implant body with the facial aspect of adjacent teeth, the implant may inadvertently be inserted too facial. No single method exists to restore proper esthetics when the implant abutment is located above the free gingival margin of the adjacent teeth. At best, the final crown appears too long and too facial. Soft tissue grafts and/or bone augmentation do not improve the condition once the implant is already incorrectly inserted (Figure 33-17).

The natural maxillary anterior teeth are loaded at a 12- to 15-degree angle, because of their natural angulation in comparison with the mandibular anterior teeth. This is one reason the maxillary anterior teeth are wider in diameter than mandibular anterior teeth (which are loaded in their long axis). The facial angulation of the implant body often corresponds to an implant body angulation, which leads to 15 degrees off axial loads and increases the force to the abutment screw-implant-bone complex by 25.9%, compared with a long axis load. These offset loads increase the risks of abutment screw loosening, crestal bone loss, and cervical soft tissue marginal shrinkage. As a result, implants angled too facially compromise the esthetics and increase the risk of complications (Figure 33-18).

Cingulum Implant Body Angulation

A second angulation suggested in the literature is more palatal, with an emergence under the cingulum of the crown. This may also be the result of an implant insertion in a width-deficient ridge (Division B), because the bone is lost primarily on the facial (see Figure 33-8, *C*). This position is often the goal when a screw-retained crown is used in restoration. The prosthesis fixation screw (to retain a maxillary anterior crown) cannot be located in the incisal or facial region of the crown for obvious reasons.

The cingulum implant position may cause a considerable health compromise. The implant body is round

WWW.HIGHDENT.IR

Figure 33-18 An implant that is too close to a tooth and/or too facial increases the risk of tissue shrinkage and poor cervical esthetics. The central implant crown and the natural lateral incisor have tissue recession as a result of the bone loss and improper implant position (too facial and too close to the tooth).

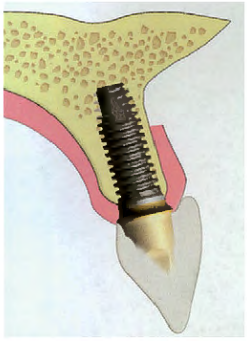


Figure 33-19 An implant placed in the cingulum position requires a ridge lap on the implant crown to restore the facial contour of the tooth.

and usually 4.0 to 5.5 mm in diameter. The labial cervical contour of the implant crown must be similar to the adjacent teeth for the ultimate esthetic effect. Because the long axis of the implant for a screw-retained crown must emerge in the cingulum position, this most often requires a facial projection of the crown or "buccal correction" facing away from the implant body. The facial ridge lap must extend 2 to 4 mm and is often similar in contour to the modified ridge lap pontic of three-unit fixed prosthesis (Figure 33-19).

The modified ridge lap crown has become a common solution to correct the esthetics of the restoration when the implant is placed in narrow bone or follows a palatal angulation position.[30,31] However, plaque control on the facial of the implant is almost impossible. Even if the toothbrush could reach the gingival sulcus, no hygiene device could be manipulated to a right angle to proceed into the facial gingival sulcus. As a result, although an acceptable esthetic restoration may be developed, especially with the additional cervical porcelain, the hygiene requirements and present implant dentistry standards render this approach unacceptable (Figure 33-20).

Some authors argue that an improved contour may be developed subgingivally with a palatal implant position. To create this contour, the implant body must be positioned more apical than desired. This position may prevent food from accumulating on the cervical "table" of the crown. However, the subgingival ridge lap does not permit access to the facial sulcus of the implant body for the elimination of plaque, as well as to evaluate the bleeding index or facial bone loss. Therefore the maintenance requirements for the implant facial sulcular region do not permit the surgeon to consider this modality as a valid primary option.

Greater interarch clearance is often needed with an implant palatal position, because the permucosal post exits the tissue in a more palatal position. Inadequate interarch space may especially hinder the restoration of Angle's Class II, division 2 patients, with the implant in this position. The bony ridge should be augmented if too narrow for the model implant diameter and position, or an alternate treatment option should be selected. The anterior single-tooth implant should use a cement-retained crown, so the cingulum screw position is not necessary.

IDEAL IMPLANT ANGULATION

The third implant angulation in the literature describes the most desirable implant angulation. A straight line is determined by connecting two points. The clinician determines the line for the best angulation by the point of the incisal edge position of the implant crown and the midfaciopalatal position on the crest of the bone. The center of the implant is located directly under the incisal edge of the crown so that a straight abutment for cement retention emerges directly below the incisal edge (Figure 33-21). Because the crown profile is in two planes, with the incisal edge more palatal than the cervical portion, the incisal edge position is perfect for implant placement and also accommodates some of the facial bone loss that often occurs before implant placement. The facial emergence of the crown mimics the adjacent teeth, proceeding from the implant body under the tissue. The angle of force to the implant is also improved, which decreases the crestal stresses to the bone and abutment screws (Figure 33-22). When in doubt, the implant surgeon should err toward the palatal aspect of the incisal edge position, not to the facial aspect, because it is easier to correct a slight palatal position in the final crown contour compared with the implant body angled too facial.

WWW.HIGHDENT.IR

Figure 33-20 **A,** The maxillary left central incisor *(right side)* with a ridge lap crown presents acceptable esthetics. **B,** However, probing on the facial aspect measures the distance to the implant but cannot evaluate facial bone loss, because it cannot be directed apically to the pocket depth. **C,** Periodically, the left central incisor implant becomes inflamed, and the bleeding index increases as a result of the inability to clean the implant sulcus on the facial.

The implant abutment selected for a maxillary anterior single-tooth implant is almost always for a cemented restoration. Single anterior crowns do not require readily retrievable restorations. In addition, a greater range of corrective options exists with a cement-retained crown for implants not well placed. The location of the cervical margin of a cemented crown can be anywhere on the abutment post or even on the body of the implant, provided it is 1 mm or more above the bone.

The implant body angulation under the incisal edge may also be used for screw-retained restorations. In these cases an angled abutment for screw retention is placed, and the coping screw for the crown may be located within the cingulum. This method does not require a facial ridge lap of the final crown, which decreases the risk of compromised hygiene.

When ideal bone volume is present, a surgical template that indicates the incisal edge and facial contour of the final prosthesis may be used. The incisal edge of the template may be notched for the drills, because the best placement of the drill is directly through the incisal edge. However, most often the surgeon does not require a template, because the adjacent teeth provide a guide for a single-tooth implant. In addition, the integrity of facial cortical plate is more readily assessed during the surgery when a template is not used.

Implant Position: Depth

Too Deep (> 4 mm)

Some authors have suggested that the implant be countersunk below the crestal bone more than 4 mm below the facial CEJ of the adjacent teeth to develop a crown emergence profile similar to a natural tooth, to prevent soft tissue recession, and to support the adjacent tissue of the adjacent natural teeth.[30,31] In concept this provides an emergence transition of about 5 mm on the facial aspect to achieve the width of the natural tooth (the ideal free gingival margin on the facial is 1 mm above the CEJ). Because the root of a natural tooth is approximately 4 mm in diameter at this point, very esthetic restorations may be fabricated with this technique. The bulk of subgingival porcelain provides

WWW.HIGHDENT.IR

A B

Figure 33-21 **A,** The perfect position for an anterior single-tooth implant is under the incisal edge to the approximate midcrest position. A central incisor should have the position slightly distal and slightly toward the palate. **B,** The implant crown has a 1.0- to 1.5-mm subgingival margin and begins a facial, mesial, and distal contour at this point to exit the tissue similarly to the contour of the adjacent tooth.

Figure 33-22 The implant crown on the left is positioned under the incisal edge. The crown on the right is for an implant in the cingulum position. The reader should note the facial emergence of the crown on the left compared with the ridge lap crown on the right.

good color and contour for the crown. However, several concerns arise regarding the long-term sulcular health around the implant (Figure 33-23, *A*).

The first year of function often corresponds to a mean bone loss range of 0.5 to 3.0 mm, dependent in part on implant design. Malevez et al.[32] noted more pronounced bone loss for conical implants that had a long, smooth, tapered crest module. The bone is lost at least 0.5 mm below the abutment to implant body connection and extends to any smooth or machined surface beyond the crest module (depending on the implant design). This may lead to facial probing depths of 7 to 8 mm or greater. Grunder[33] evaluated single-tooth implants in function for 1 year and noted the bone levels were 2 mm apical to the implant-abutment connection and sulcular probing depths were 9.0 to 10.5 mm using a Brånemark implant design. As a result, daily care devices cannot maintain the sulcus health, and anaerobic bacteria are more likely to develop. The interproximal regions of the implant crown, which correspond to the incidence or absence of interdental papillae, usually exhibit even greater probing depths. As a result, gingival shrinkage of the tissue is more likely to occur when the implant is placed more than 4 mm below the facial position of the adjacent CEJ.

The attachment mechanism of the soft tissue above the bone is less tenacious compared with a tooth, and the defense mechanism of the peri-implant tissues may be weaker than that of teeth.[34] The clinician, to err on the side of safety for the best sulcular health conditions, should limit sulcular depths adjacent to implants to less than 5 mm.[35] This may be even more relevant for single-tooth implants because of the devastating consequences of gingival shrinkage for long-term esthetics. In addition, the interproximal regions of the single-tooth implant crown are shared with the adjacent teeth, and anaerobic bacteria that form in the region next to the implant eventually may affect the adjacent natural tooth because a horizontal defect may form (especially when the implant is closer than 1.5 mm to the tooth).

When the implant is countersunk below the crestal cortical bone, the trabecular bone around the crest module is weaker against occlusal loads. In addition, when the implant is placed below the crestal bone, the resultant initial crown height is increased, as are moment forces. A further increased risk of soft tissue shrinkage occurs long term, with additional bone loss at the crest module. The result is longer clinical crowns, which also decrease gradually in width (as the narrowing dimensions approach the implant body), with resultant black triangular spacings in lieu of interdental papillae and compromised long-term esthetics.

Too Shallow (< 2 mm)

When the implant body is positioned less than 2 mm below the facial free gingival margin of the crown, the cervical esthetics of the restoration are at an increased risk (Figures 33-23, 33-24). The porcelain of the crown may not be subgingival enough to mask the titanium color of the abutment below the margin. If bone loss

WWW.HIGHDENT.IR

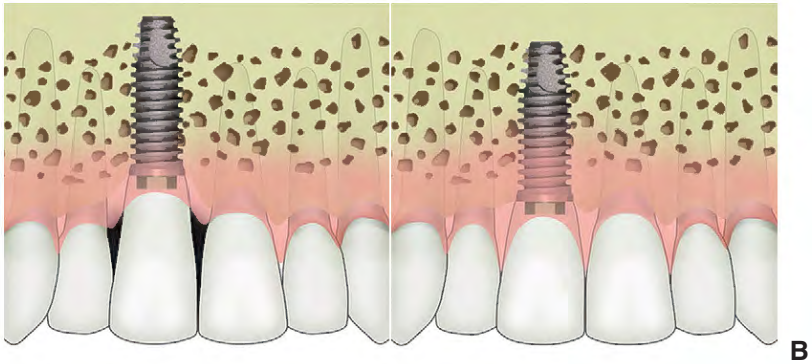


Figure 33-23 The literature usually reports two positions for the depth of the implant. The facial bone of a healthy natural tooth is about 2 mm below the cement-enamel junction (CEJ), and the soft tissue from the free gingival margin to the bone is 3 mm. An implant inserted 4 mm or more below the facial CEJ often is countersunk below the crestal bone 2 mm or more **(A).** This gives "running room" for the porcelain on the crown to create a natural-looking emergence profile. However, bone loss is likely to occur beyond the implant abutment connection and often proceeds to the first thread beyond the crest module. As a result, an increase in probing depth and growth of anaerobic bacteria are more likely. **B,** An implant placed 2 mm below the adjacent facial CEJ provides 3 mm of soft tissue drape to develop with a natural appearance and is best. An implant placed too shallowly causes severe emergence profile angles and compromises esthetics.

occurs, then the titanium implant abutment and/or body may also cast a dark shadow to the gingival tissues. If apical shrinkage of tissue occurs, then the dark titanium abutment and implant body may become directly visible. Periodontal surgical procedures to position soft tissue over the titanium roots are unpredictable.

On occasion, the crestal bone height is coronal to the perfect height. The two most common conditions that result in this finding are (1) when the adjacent teeth are closer than 6 mm (in agenesis of a lateral incisor) and (2) when a block bone graft regenerated width and height of bone. Ideally, the interproximal bone is 3 mm above the midcrestal bone. When the teeth are closer than 6 mm (i.e., a lateral incisor in the maxilla), the interproximal bone height of each adjacent tooth to the missing space is able to stimulate and maintain bone at the interproximal level. When a single-tooth implant replaces this missing tooth, an osteoplasty should be performed so that the midcrestal region is 3 mm apical to the free gingival margin of the future crown. The same conditions may occur when bone augmentation gains height to the interproximal height of bone.

To solve the problem of an implant body placed too shallow, the restoring dentist may need to prepare the implant crest module and place the margin of the crown directly on the implant body (see Figure 33-24).

Ideal Depth (3 mm)

The best platform level for a two-stage implant is similar to the most desirable bone level before the loss of a natural tooth, which is 2 mm below the adjacent tooth CEJ. This positions the platform of the implant 3 mm below the facial free gingival margin of the implant crown (see Figure 33-23). In addition, it provides 3 mm of soft tissue for the emergence of the implant

Figure 33-24 An implant platform 2 mm or less below the adjacent cement-enamel junction is too shallow and results in crown contour or increases the risk of inadequate interdental papilla.

crown on the midfacial region and more as the soft tissue measurements proceed toward the interproximal (Figure 33-25). This depth also increases the thickness of the soft tissues over the titanium implant body, which masks the darker color above the bone. It should be noted that the free gingival margin of a lateral incisor is often 1 mm more incisal than the adjacent central and canine natural tooth.

SOFT TISSUE INCISION

Once the midmesiodistal position, midfaciopalatal position, angulation under the incisal edge, and implant depth in relation to the crest and final crown have been

WWW.HIGHDENT.IR

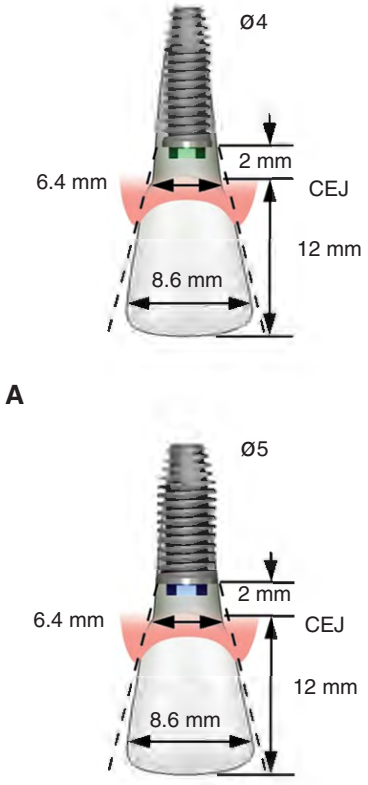


Figure 33-25 The ideal depth of the implant platform is 2 mm below the natural tooth cement-enamel junction (CEJ) or 3 mm below the free gingival margin. This provides adequate emergence profile for the implant crown and creates an environment for soft tissue health around the implant.

Box 33-1 Soft Tissue Contouring and Emergence Profile

Preprosthetic Surgery

Soft tissue graft before bone augmentation
Soft tissue graft in conjunction with bone graft before implant surgery

Surgery Stage I

Soft tissue augmentation
Nonresorbable hydroxyapatite (HA) graft

Surgery Stage II

Connective tissue graft (subepithelial)
Soft tissue plastic surgery
Gingivoplasty (coarse diamond)

Prosthetics

Wide healing abutment
Temporary contouring through provisional restoration
Anatomical abutment, tooth-colored abutment
Pink porcelain on abutment

determined, the osteotomy preparation may be started.

The ideal tissue drape is often the most difficult aspect of maxillary anterior single-tooth replacement within the esthetic zone. Several different approaches are used to enhance the soft tissue appearance. The approaches may be surgical (addition or subtraction) or prosthetic: (1) a soft tissue graft before bone augmentation, (2) a soft tissue augmentation in conjunction with a bone graft before implant insertion, (3) soft tissue augmentation in conjunction with implant insertion, (4) soft tissue manipulation at the implant uncovery procedure, (5) a prosthetic modification of interproximal contact position, (6) creeping attachment around the implant crown, or (7) a prosthetic replacement of the soft tissue with pink-colored porcelain (Box 33-1).

Surgical additive techniques such as pouch procedures, interpositional grafts, sliding flaps, and connective tissue grafts (autogenous or acellular dermal matrix) have all been proposed. A soft tissue graft is performed as a separate procedure, before any other surgery when the patient has a high lip dynamic and the soft tissue color and/or volume is grossly deficient. Most often, a connective tissue graft to improve the soft tissue drape is indicated.

A bone graft and soft tissue augmentation is indicated when the bone on the adjacent teeth is within normal limits (2 mm below the CEJ) but deficient in width and midcrestal volume. When the interproximal bone is not within normal limits, orthodontic extrusion is considered, followed by a crown and possibly endodontic therapy. The goal of a soft tissue augmentation for either of the two previous procedures is to obtain soft tissue on the crest of the ridge at the height of the interproximal papilla height.

The interproximal soft tissue in the implant site may be classified into three categories: (1) the papillae have an acceptable height in the edentulous site, (2) the papillae have less than acceptable height, or (3) one papilla is acceptable and the other papilla is depressed and requires elevation. When the interproximal papilla has an acceptable height, "papilla-saving" incisions are made adjacent to each neighboring tooth (Figure 33-26). The vertical incisions are made on the facial aspect of the edentulous site and begin 1 mm below the macrogingival junction, within the keratinized tissue. Extending the vertical incisions beyond the macrogingival junction increases the risk of scar formation at the incision site. The full-thickness incision then approaches the crest of the edentulous site, leaving 1.0 to 1.5 mm of the interproximal papilla adjacent to each tooth. The vertical incisions are not wider at the base than the crestal width of tissue. This permits the facial flap to be advanced over the implant or short and adjacent to a permucosal extension (PME) at the conclusion of the procedure, with no voids at the incision line and primary closure (Figure 33-27).

WWW.HIGHDENT.IR

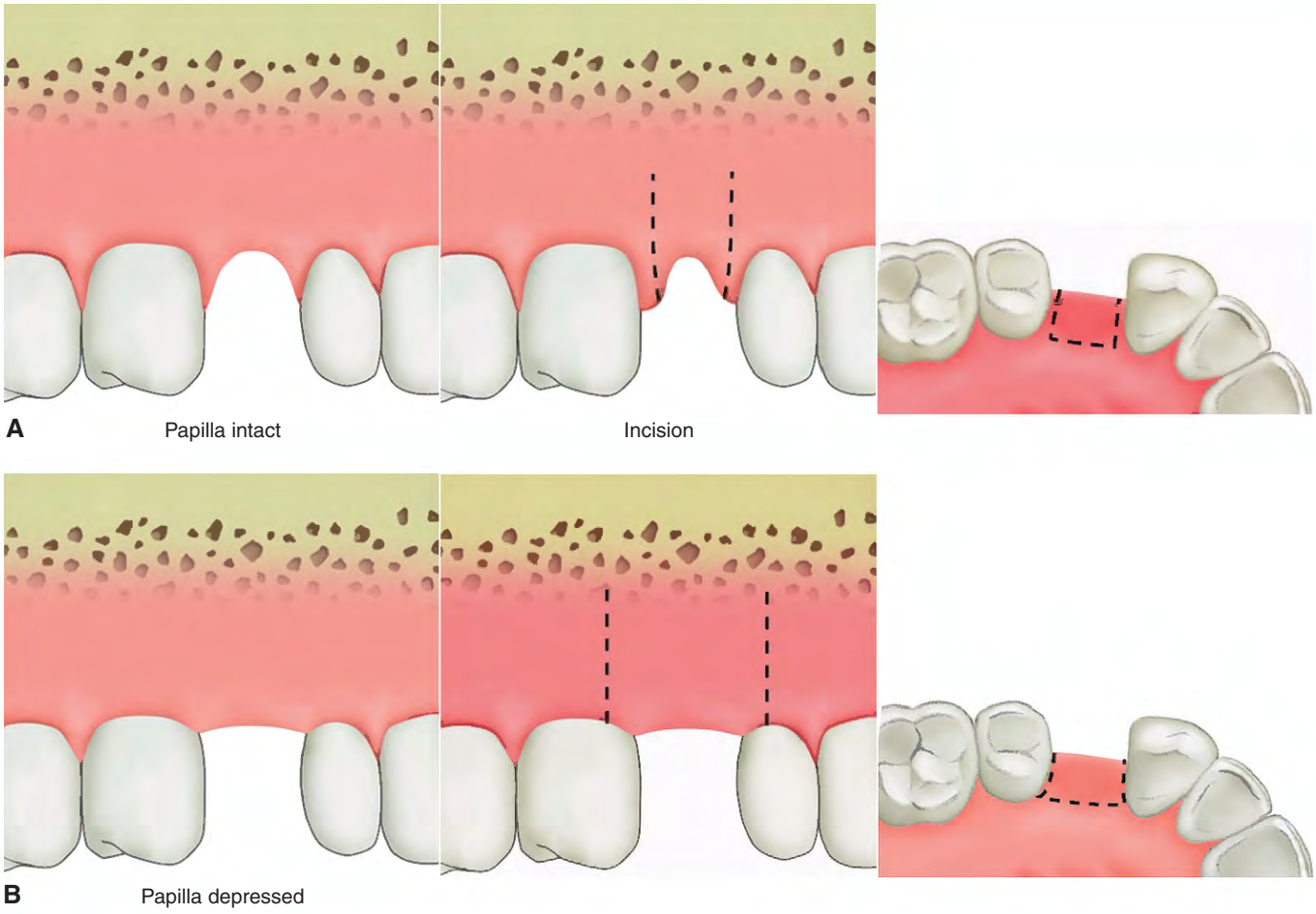


Figure 33-26 When the interdental papillae are in an acceptable position **(A),** papilla-saving incisions are made to minimize soft tissue reflection. The incisions are vertical to allow primary closure. When the papillae are depressed, the vertical release incisions include the papilla in the edentulous site. In situations with a more depressed soft tissue, facial soft tissue and papillae over each adjacent tooth are also reflected. The crestal incision is on the palatal incline on the ridge **(B).**

When the papillae are depressed in the edentulous site, vertical-release incisions are made along the root angle of each adjacent tooth, beginning 1 mm below the macrogingival junction, as well as in the sulcus of each adjacent tooth. Therefore the interproximal papilla region becomes part of the facial soft tissue flap (see Figure 33-26, *B*).

When the papillae are mixed in acceptance, a vertical-release incision is made that does not include the adequate papilla. The second vertical incision includes the depressed papilla (Figure 33-28). In either case, the crestal incision is extended to the palatal aspect on the crest of the edentulous site.

The crestal incision is made on the palatal incline of the edentulous site to provide greater thickness of keratinized tissue on the facial aspect of the flap. This also allows more interproximal tissue to be elevated to enhance the papilla height.

The soft tissue is reflected, and the crestal bone width of the ridge is evaluated. When a central incisor site is reflected, the palatal flap is reflected to the incisive foramen for identification and evaluation. On occasion, its position may require the soft tissue to be enucleated and a graft positioned in its site.

A 2-mm-diameter pilot drill is positioned in the midmesiodistal and faciopalatal aspect of the ridge and proceeds 9 mm within the bone under copious cooled sterile saline (Figure 33-29, *A*). A 9-mm-long force direction indicator is positioned into the site for evaluation (Figure 33-29, *B*). The stop on the direction indicator is often 4 mm in diameter and may be used to directly assess the amount of bone on the facial, palatal, mesial, and distal of the final implant position. A radiograph is taken, and the implant body position in relation to the adjacent roots and opposing landmark (i.e., floor of nose) is assessed. If corrections are required, then a side-cutting drill (i.e., Lindemann drill) may be used.

When the osteotomy may be extended to 12 to 15 mm, starting 3 mm below the free gingival margin of the future implant crown, the 2-mm-diameter drill is reinserted to the final depth position. Most often, the drill prepares the bone 1 to 2 mm deeper than the length of the implants (when the opposing landmark permits) (see Figure 33-29, *C*). This allows the implant

WWW.HIGHDENT.IR

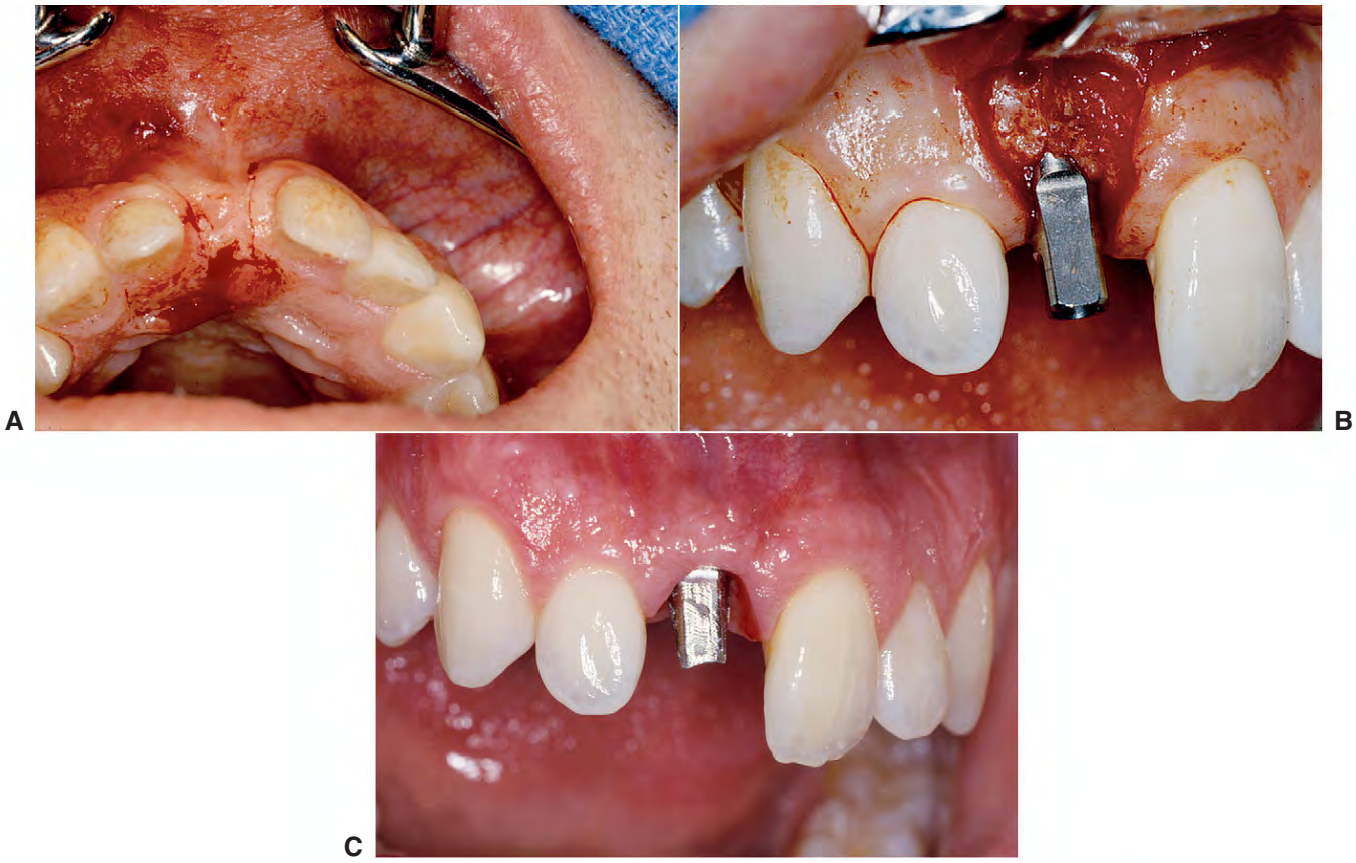


Figure 33-27 **A,** When the papillae are in an acceptable position, papilla-saving incisions are placed and the interdental papillae are not reflected for the implant surgery. **B,** The facial soft tissue is reflected to the mucogingival junction, and the central incisor implant is positioned slightly to the distal, 3 mm below the facial free gingiva margin. **C,** After initial healing and soft tissue contouring with a temporary crown, the soft tissue drape and implant components are ready for the final restoration.

to be rotated to a perfect position at the end of the procedure, without "bottoming out" at the end of the osteotomy, which may prevent the desired crestal position of the implant.

A 2.5-mm end-cutting twist drill, rotating 2500 rpm under copious amount of cooled sterile saline is then used to beyond the final depth. The drill is removed from the hand piece and reinserted into the osteotomy (see Figure 33-29, *D*). The top of the 2.5-mm drill should be directly under the incisal edge of the future implant crown. On occasion, it is slightly facial. This is because the more dense palatal bone has pushed the end-cutting drill facially to the less dense trabecular bone, rather than permitting its uniform preparation. When this occurs, the side-cutting drill is introduced into the osteotomy, and the palatal bone is removed by "shaving" up and down in the palatal aspect of the preparation.

A 3.0- and then a 3.4-mm-diameter end-cutting drill then prepare the site for a 4.0-mm diameter implant (the final drill of each implant system may be a slightly different dimension) (see Figure 33-29, *E* and *F*). Again, each drill is removed from the hand piece, inserted into the bone site, and evaluated as to position under the incisal edge. When facial, the side-cutting drill corrects its position.

A crestal bone drill is most often not used in the maxillary anterior region, because the bone is usually D2 or D3 and little to no cortical bone is found on the crest. This allows the implant to compress and slightly expand the bone in the crestal region of the implant site.

Once the final osteotomy diameter and depth is prepared, a bone tap is used in a low-speed, high-torque hand piece (i.e., 30 rpm, 70 N-cm) to form the threads within the bone for the implant (see Figure 33-29, *G*). This is an important step for all threaded implants. Because dense cortical bone is on the palatal aspect of the implant osteotomy, a self-tapping, threaded implant will tend to be pushed to the softer bone on the facial, and the implant will end up more facial (with less facial bone over the implant) than expected. In fact, even the bone tap may be pushed facially. Therefore once the tap is inserted to the final depth, it is removed from the hand piece and the angulation is evaluated relative to the incisal edge position of the future crown. When it is more facial than desired, a side-cutting drill is used to remove some of the palatal bone.

WWW.HIGHDENT.IR

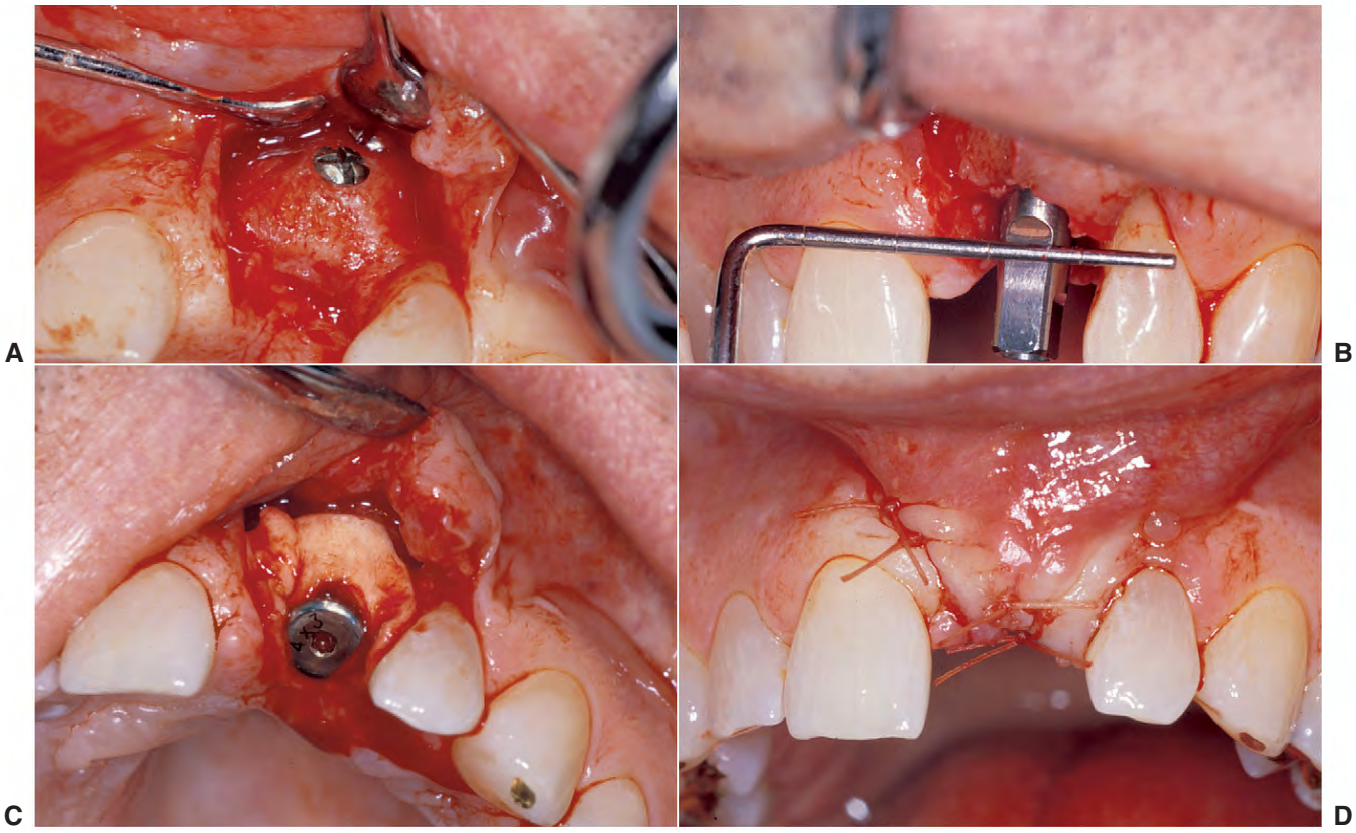


Figure 33-28 **A,** A papilla-saving incision is made to the mesial aspect. The incision to the distal included and reflected the papilla (which was depressed). **B,** The implant is positioned 3 mm below the free gingival margin of the implant crown. **C,** A 3-mm-high permucosal extension (PME) is placed into the implant body, and an acellular tissue graft (AlloDerm) is positioned around the PME to help augment the distal papilla and crest of the ridge. **D,** The tissue is elevated over the PME and membrane to increase the height of the crest and distal papilla.

The threaded implant is inserted with a hand piece at 30 rpm (see Figure 33-29, *H*). When the implant (or bone tap) is threaded by a hand wrench or with the surgeon's fingers, it is easily pushed more facial, without the awareness of the surgeon. When a hand piece is used, counterforces on the hand piece handle and the fingers of the other hand on the hand piece head may permit the implant to be inserted without compromise to angulation or position.

The implant is rotated in final position, with a flattening of the antirotational component to the facial. This position allows a greater thickness of metal on the abutment to the facial so that the restoring dentist may slightly prepare the facial aspect of the abutment when necessary, to allow a greater thickness for porcelain.

The implant mount may be removed, and the surgeon decides whether a low-profile cover screw or PME healing cap is used within the implant. When the hard and soft tissue contours are ideal and a papilla-saving incision was made, a permucosal abutment may be used. When a bone graft was placed and/or the crestal tissue elevated to increase the height of the papilla, a low-profile cover screw is more often used.

Once the cover screw is inserted, the surgeon decides whether a bone graft on the facial bone is indicated. When the facial bone over the implant is less than 1.5 mm thick, a particulate graft and a resorbable membrane may be used to augment the site (See Chapter 37). Most often the membrane is an acellular dermal matrix such as AlloDerm, because it will improve the bone graft site and also increase the tissue thickness.

SOFT TISSUE CLOSURE

As previously discussed, several different steps exist at which the soft tissue contour may be addressed. The ideal tissue height during initial implant healing is level with the height of the desired interdental papillae. A connective tissue graft or acellular dermal matrix (AlloDerm) material may be placed at the implant placement surgery under the facial and interproximal tissue to improve and/or thicken the contour of the soft tissue drape. The soft tissue is approximated and sutured with a resorbable material (4-0 or 5-0) around the PME or over the cover screw, depending on whether

WWW.HIGHDENT.IR

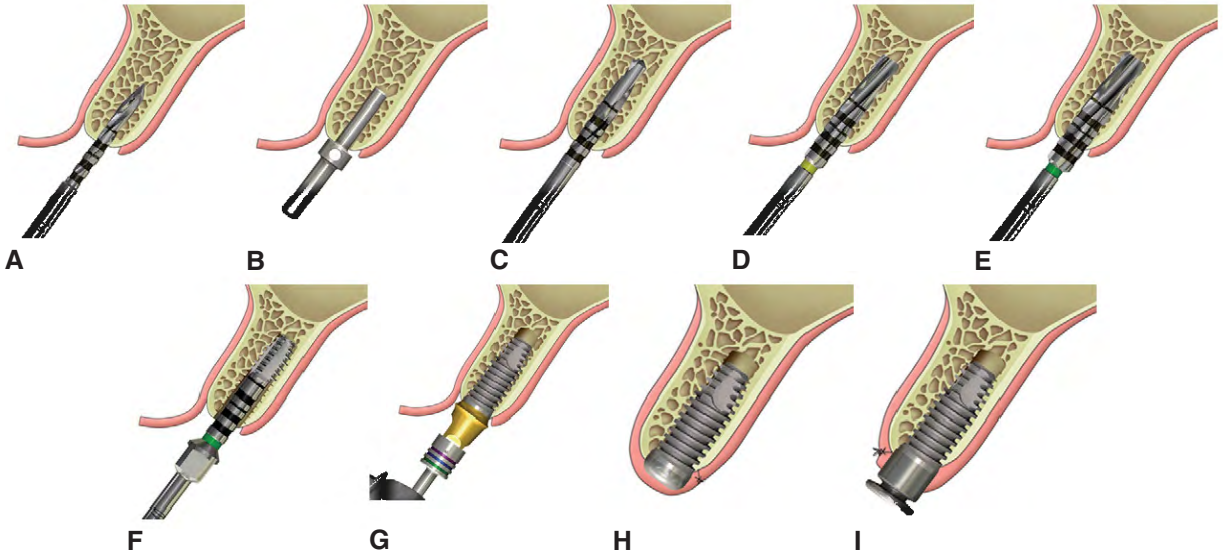


Figure 33-29 A 2-mm-diameter pilot drill is positioned in the midmesiodistal and faciopalatal aspect of the ridge and proceeds 9 mm within the bone under copious cooled sterile saline. **A,** A 9-mm-long force direction indicator is positioned into the site for evaluation. **B,** The 2-mm-diameter drill is reinserted to the final depth position. Most often, the drill prepares the bone 1 to 2 mm deeper than the length of the implants (when the opposing landmark permits). **C,** A 2.5-mm end-cutting twist drill, rotating at 2500 rpm under copious amount of cooled sterile saline, is then used to beyond the final depth. The drill is removed from the hand piece and reinserted into the osteotomy to evaluate the implant site angulation. **D,** A 3.0- and then a 3.4-mm-diameter end-cutting drill then prepare the site for a 4.0-mm-diameter implant (the final drill of each implant system may be a slightly different dimension). **E,** Once the final osteotomy diameter and depth is prepared, a bone tap is used in a low-speed, high-torque hand piece (i.e., 30 rpm 70 N-cm) to form the threads within the bone for the implant. **F,** The threaded implant is inserted with a hand piece at 30 rpm. **G,** The soft tissue is approximated and sutured with a resorbable material (4-0 or 5-0) around the permucosal extension or over the cover screw, depending on whether the tissue is at the right position or is being augmented **(H** and **I).**

the tissue is at the right position or is being augmented (see Figure 33-29, *H* and *I*). The increased tissue thickness from augmentation facilitates the sculpting of interdental papillae at stage II surgery, improves ridge contour, and prevents the grayish hue of the titanium implant body from showing through the labial mucosa in the event of crestal bone loss in the future.

The transitional prosthesis often needs to be relieved in the cervical region of the implant site during the healing period to accommodate the increased soft tissue height over the implant and to avoid pressure on the area over the implant body.

Transitional Prosthesis

It is strongly suggested that a resin-bonded fixed restoration be fabricated to provide improved speech and function, especially when crestal bone regeneration is performed. The soft tissue–borne transitional restoration enhances crestal bone loss during the graft healing, may cause bone loss around the implant during stage I healing (or even implant failure from the early loading), and may depress the interdental papillae of the adjacent teeth. As a result, a resin-bonded fixed prosthesis is fabricated for the extended healing, and a removable device may be used short term for cosmetic emergencies should the device become debonded. When a resin-bonded restoration is used, the adjacent teeth are not prepared and the device is bonded to the tooth regions below the centric occlusal contacts of the teeth.

The transitional restoration for the single-tooth implant has the benefits of being off the soft tissue drape, the developing bone augmented site, and the healing implant-bone interface. Other options exist besides the resin-bonded device that permit these goals. An Essix appliance is an acrylic shell, similar to a bleaching tray, that has a denture tooth attached to replace the missing tooth. This device is the easiest for tooth replacement after surgical procedures. When an adjacent tooth requires a crown in the overall treatment plan, the adjacent tooth may be prepared and a cantilevered transitional FPD with a pontic over the surgical site may be used. When the patient requires orthodontics, a denture tooth and an attached bracket may be added to the orthodontic wire. A cast clasp RPD with indirect rest seats to prevent rotation movements on the surgical site is also an excellent option.

IMMEDIATE IMPLANT INSERTION AFTER EXTRACTION

According to Kois,[36] five diagnostic keys exist for predictable single-tooth peri-implant esthetics when

WWW.HIGHDENT.IR

Table 33-2 Implant Placement in Extraction Site

IMMEDIATE	DELAYED
Advantages	
↓ Surgery	↑ Surgical control, position, angulation
↓ Time	↑ Hard tissue quality
↑ Maintenance of soft tissue	↑ Success, less risk of infection
Intraosseous bone graft	
Disadvantages	
↑ Facial bone loss after loading or during healing	Extended time for transitional
Implant too facial and/or angled abutment	Onlay graft may be needed at surgery

an immediate extraction and implant insertion is contemplated: (1) the tooth position relative to the free gingival margin, (2) the form of the periodontium, (3) the biotype of the periodontium, (4) the tooth shape, and (5) the position of the osseous crest before extraction. Therefore the soft tissue and bone comprise three of the five factors for predictable esthetics.

The goal of the anterior implant restoration is to simulate the appearance of a natural tooth. Adequate bone volume must be present for correct hard and soft tissue contours. Implant placement soon after initial alveolar bone healing or bone grafting is usually advantageous. As such, immediate implant placement in an extraction site has become a very popular topic[37-57] (Table 33-2).

Disadvantages

When placing an implant in an immediate-extraction site, the surgeon should consider the socket dimension and the defect between the labial plate of bone and the implant. The faciopalatal dimension of an anterior tooth is often greater than its mesiodistal dimension. When an anterior tooth requires extraction, during the extraction process the thin facial cortex often becomes compromised or lost. As a result, it is most always several millimeters apical to the palatal cortical plate, and frequently bone grafting and/or membrane placement in conjunction with the implant insertion are needed. Immediate implant placement in the anterior region using a round implant often requires that the osteotomy and implant insertion engage the lingual wall of the alveolus and penetrate halfway to two thirds of the way down the extraction site into the remaining lingual apical bone for rigid fixation. This surgical approach is more challenging than preparing the osteotomy in a homogenous bone density.

The best implant size is often 4 to 5 mm in diameter for a central incisor, and the extraction socket is often greater than 6 mm (especially in the faciopalatal dimension), so a surgical defect as large as 2 mm remains around the implant. More or less broad, oval- or kidney-shaped spaces have been described to run coronoapically along the entire surface of the socket next to the implant.[41] The natural resorption of the facial plate may not be halted by implant insertion, and bone-implant contact is reduced when the facial plate resorbs.[41a] When the facial bone is missing, the bone regenerated over the facial aspect of the implant with guided bone regeneration (GBR) is often immature woven bone, which is more prone to resorption because of occlusal overload. Typically, techniques for immediate implant placement after extraction include countersinking the implant 2 mm or more below the facial plate (which is already more apical than the palatal plate) and placing calcium phosphate ($CaPO_4$), resorbable HA, and/or autologous bone to fill the labial defect, with or without the addition of connective tissue grafts and/or membrane. Prolific literature proposing varied classifications and protocols has been published within the last 10 years.

The implant will obtain rigid fixation with nearly all of these techniques. However, the goal of the anterior single-tooth implant is not limited solely to rigid fixation. The inability to achieve proper esthetic and health parameters constitutes a compromised result and increased risk of esthetic or implant failure.[47] When the implant is countersunk below the facial bone, the implant platform may be as much as 4 mm apical to the CEJ of the adjacent teeth, which increases the anatomical crown height and the pocket depth, especially after crestal bone loss during the first year. In addition, synthetic grafts placed around the titanium implant grow less dense quality bone, which is also limited in implant contact. The capacity of this less dense bone promoted by barrier membranes around implants to withstand loading seems to be limited, and animal studies indicate as much as 85% may be lost after loading.[58] An explanation may be that no blood vessels arise from the implant; to the contrary, it reduces the number of bony walls of the defect and limits blood supply to the facial bone graft. As a result, bone is less likely to form, and when it forms it is less dense and more at risk of resorption once the implant is loaded.

Primary closure of the soft tissue provides a more predictable environment when grafting is performed on the facial aspect of the implant and may be difficult with the immediate extraction technique. Although not advocated, the labial tissue is often reflected to approximate the tissue over the socket defect. This technique further compromises the blood supply to the labial cortical bone and also decreases the amount of facial attached gingiva, because the facial tissues are placed over the extraction socket. As a consequence, a free tissue, subepithelial, or connective tissue graft may be indicated after stage I healing to restore the facial attached keratinized tissue.

WWW.HIGHDENT.IR

The labial bone usually remodels to 0.5 mm below the abutment to implant connection (which was, in most cases, already countersunk below the facial bone and several millimeters below the palatal bone). Bone loss may continue to the region of the first thread (as a result of the implant crest module design), then stabilize in a region of greater bone density. As a consequence, reports often illustrate soft tissue pocket depths greater than 7 to 8 mm at the midfacial tooth position. The presence of anaerobic microorganisms in soft tissue pockets of 5 mm or more has been documented. With good hygiene the soft tissue often recedes, with a resulting lengthened clinical crown and "black triangles" in the interproximal areas caused by the absence of properly developed interdental papillae, which compromises long-term esthetics and/or contributes to soft tissue complications.

An increased risk of postoperative infection exists around the implant with an immediate insertion, by the bacteria associated with the cause of tooth loss when judicious case selection has not been exercised. The presence of exudate lowers the pH, which causes a solution-mediated resorption of the grafted bone and contaminates the implant body with a bacterial smear layer, which in turn reduces bone contact.

An improved bone interface may be obtained if the large-diameter extraction site is grafted before implant placement. If the labial plate is compromised, then additional intraorally harvested bone and/or GBR are indicated. The delayed implant insertion method appears to enhance capillary propagation and trabecular formation before implant placement, facilitating the formation of an implant-bone interface.[59] Before grafting, the soft tissue granulates over the extraction site, thus creating an increased zone of attached gingiva. The result of the augmentation is evaluated before implant placement, rather than dealing with compromises after implant integration. In this way, the implant may be placed in an ideal position in relation to the crestal bone, and adjacent teeth and within the exact contours of the final restoration.

Advantages

The benefits of immediate implant insertion after tooth extraction are related to an improved preservation of the soft tissue drape and the bone architecture, compared with their collapse after tooth extraction. As a result, bone augmentation and soft tissue grafts may be avoided. The procedure has been described as a preservation technique aiming at maintaining the harmonious gingival architecture. The procedure also reduces the number of surgeries, which may decrease the cost to the patient. Whenever this technique is considered, the technique of orthodontic extrusion before extraction and implant insertion offers many benefits.

Teeth with a tapered crown form have a higher risk of soft tissue compromise after extraction. The tapered crown also has more interproximal bone between the teeth and more facial bone over the tapered root. As such, under perfect conditions, the tapered tooth form may be more advantageous for extraction and immediate implant insertion. Therefore under the best conditions, which include lack of pathology, thick gingival tissues, ideal bone contours, ideal soft tissue contours, and tapered tooth forms, an immediate implant insertion after tooth extraction is considered.

A square tooth form has less gingival shrinkage after extraction and exhibits less scalloping of the interproximal and facial bone with adjacent tooth roots. There is also less bone between the roots and larger spaces between the extraction site and the implant. As a result, an immediate implant insertion after extraction offers less benefit for the soft tissue and greater risk for the implant-bone interface. Therefore root extrusion before extraction, or extraction with a delayed implant insertion, is more often considered.

Technique

When ideal conditions for an extraction and immediate implant insertion are present, the following scenario may occur. First of all, the patient is informed that the procedure may be aborted and only a bone graft may occur. The surgeon does not want to compromise a 30-year prosthesis simply to avoid waiting an additional 3 to 6 months.

The tooth is removed with periotomes, a class I lever forceps (Physics Forceps, Detroit, Mich.), and/or other atraumatic procedure. The surrounding walls of bone are evaluated, and confirmation of an intact, noninfected boney socket is made (Figure 33-30).

The best position for the implant is under the incisal edge, in the midfaciopalatal and mesiodistal center. This does not coincide with the root apex position; therefore a pilot drill penetrates the palatal incline of the root, approximately two thirds of the way down from the crest, slightly more palatal than desired, to the depth of 12 mm (Figure 33-31). The drill is then repositioned under the incisal edge of the future crown. A radiograph with a force direction indicator (or the drill removed from the hand piece) in place confirms its mesiodistal position relative to the adjacent roots and the opposing landmark.

Once the correct position is confirmed, the 2-mm twist drill prepares the osteotomy to the opposing landmark, up to 18 mm (Figure 33-32, *A* and *B*). The drills overprepare the depth of the osteotomy so that the apical bone may be harvested from the drill. The side-cutting drills are used as previously indicated, to keep the osteotomy under the incisal edge. The final length implant is usually 12 to 15 mm long. The incremental drills prepare to this final length and diameter (Figure 33-32, *C*). The bone tap is used to this final depth, as previously presented (Figure 33-33).

The bone harvested from the cutting drills is then positioned within the osteotomy, primarily on the inner facial wall, where the largest defect is present (Figure 33-34). The implant is threaded into position

WWW.HIGHDENT.IR

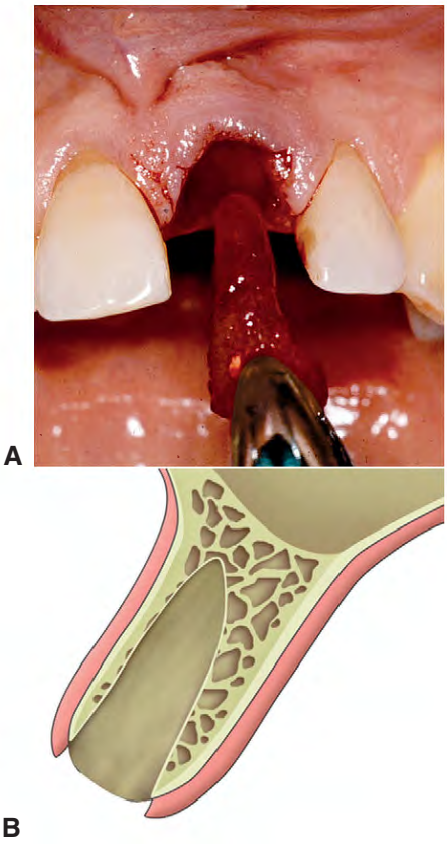


Figure 33-30 The patient's left maxillary incisor is atraumatically extracted **(A).** The surrounding walls are evaluated and confirmed to be intact and free of infection **(B).**

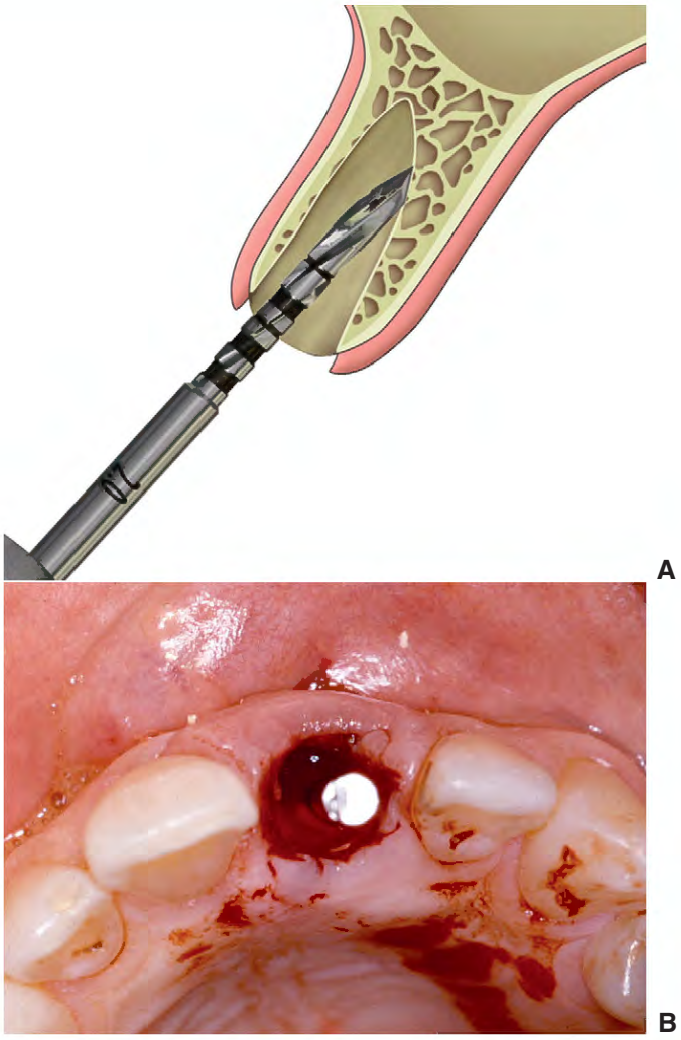


Figure 33-31 **A,** A 2-mm pilot drill first prepares the initial osteotomy two thirds of the way down from the crest and through the thick palatal cribiform plate. **B,** The drill is then rotated to prepare the site under the incisal edge of the future implant crown.

using a slow-speed, high-torque hand piece and no irrigation. Some of the bone graft will be pushed to the apex of the osteotomy (Figure 33-35). However, because it is deeper than required, it does not impede the implant seating in its final position. Therefore the implant is in the correct position, and the socket defects are filled with autologous particulate bone.

A PME has a higher profile than the cover screw. A 4.5-mm PME placed into the implant helps to support the tissue above the crest of bone (Figure 33-36). Once the PME is inserted into the implant, the transitional prosthesis is delivered. The primary motivator for immediate implant insertion after extraction is to maintain the soft tissue drape. Therefore the PME and/or the transitional device should fill the emergence contour, without voids or compression to the soft tissue (Figure 33-37).

STAGE II SURGERY AND SOFT TISSUE EMERGENCE CONTOURS

The clinician should use radiographs to closely evaluate the crestal mesial and distal bone-implant interface relative to an absence of crestal bone loss before the abutment post is added to the implant body. Probing is necessary to evaluate the facial and palatal conditions. If bone loss is suspected, then the tissue should be reflected for direct evaluation. Correction of a cervical horizontal defect includes local autogenous grafts covered with a barrier membrane and reapproximation of the soft tissue. For a vertical defect less than 2 mm, autogenous bone may be added and the uncovery of the implant may proceed, because bone growth is more probable in the presence of the lateral walls of bone.

Before the stage II uncovery procedure, the soft tissue drape may be modified: (1) before a bone graft procedure, (2) in conjunction with the bone graft in the implant site, and (3) at the time of implant insertion. The fourth time in this sequence that the tissue may be addressed is at the stage II uncovery procedure.

When the implant-bone interface is acceptable, the exposure of the implant body should be accomplished with the soft tissue final architecture in mind. To achieve

WWW.HIGHDENT.IR

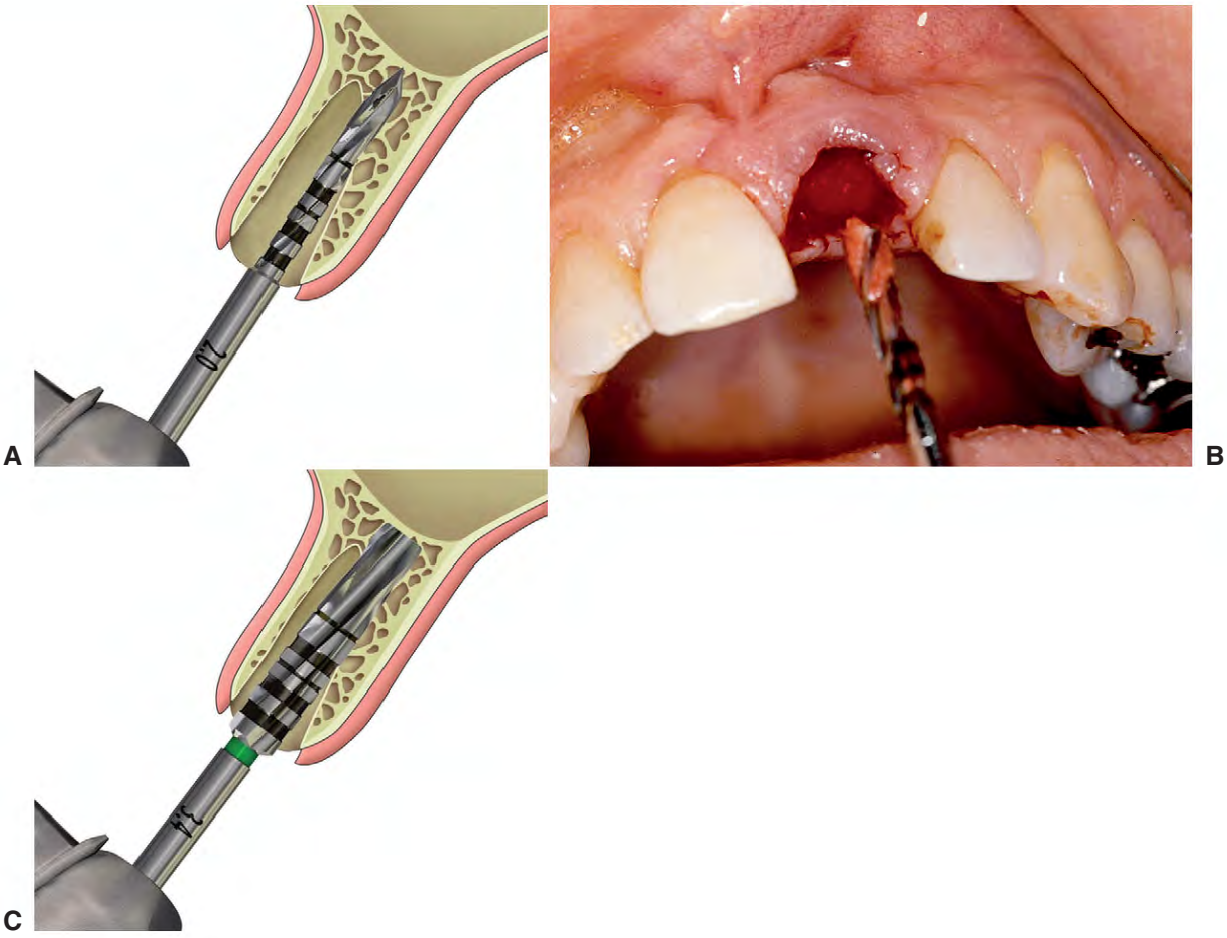


Figure 33-32 The implant drills prepare the site to the floor of the nares **(A)** and harvest bone beyond the tooth apex **(B).** The osteotomy is kept in an angulation aligned with the incisal edge of the adjacent teeth **(C).**

Figure 33-33 A bone tap is used to final depth using a hand piece.

the proper soft tissue architecture, several options are available, depending on the soft tissue appearance before stage II uncovery (Box 33-2). These soft tissue procedures may be classified as *subtractive, additive,* or *a combination of each.*

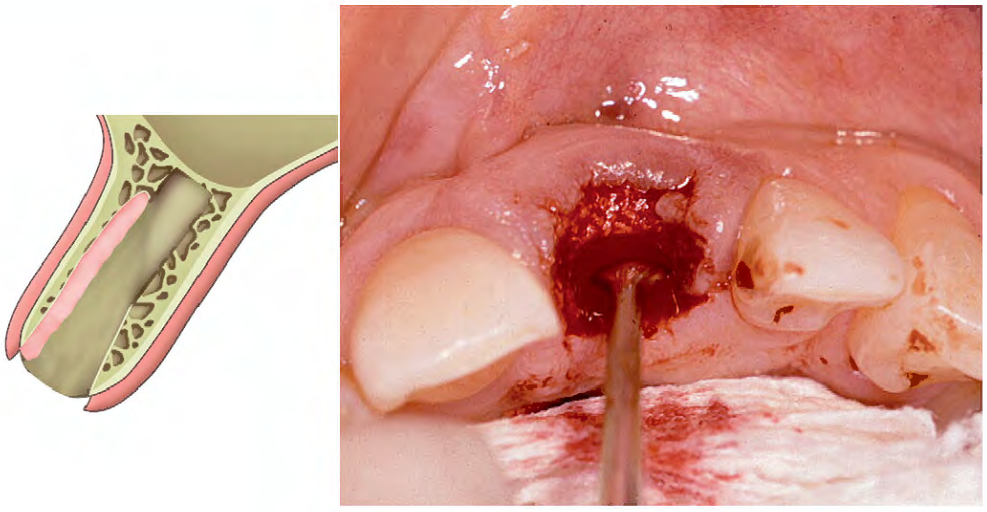
Subtractive Technique

When the soft tissue along the edentulous crest is at the level of the desired interdental papillae and is of sufficient quality and volume, a subtraction technique (e.g., gingivoplasty with a coarse diamond) sculpts the crestal gingival tissues to reproduce the cervical emergence contour of the crown, complete with interdental papillae and proper labial gingival contour (Figure 33-38). The contour of the midfacial position of the tissue is 1 mm more incisal than the contour of the adjacent teeth to allow for the gingival shrinkage commonly observed during the first year of implant loading. The interdental papilla zones are also made slightly larger than the final desired form to accommodate possible shrinkage.

Addition Technique

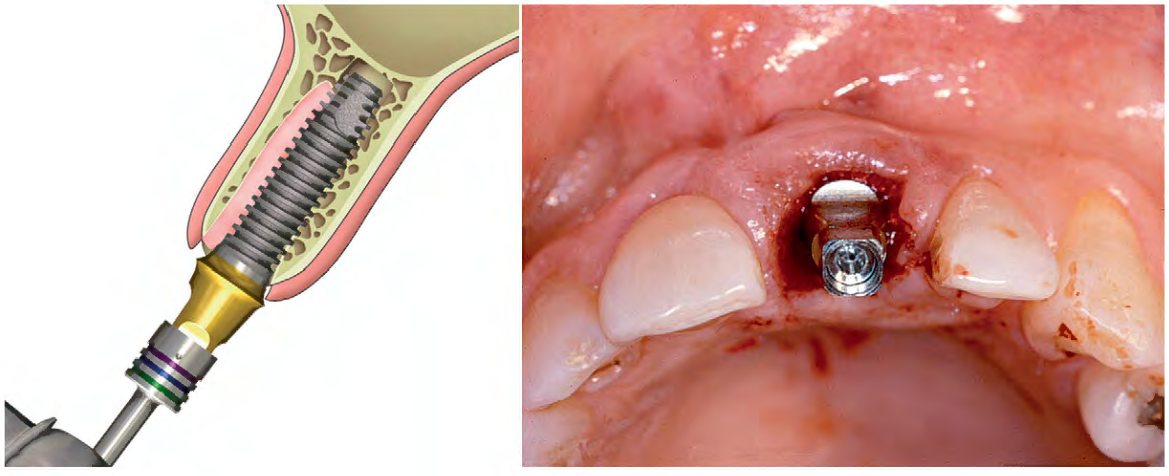
If the gingival contour at stage II uncovery is insufficient for the proper architecture for the interdental papillae, then an additive surgery is performed to gain tissue thickness. Several addition techniques have been proposed.[60-65] For example, an incision is made on the palatal aspect of the ridge, from the palatal angle of each adjacent tooth; a

WWW.HIGHDENT.IR



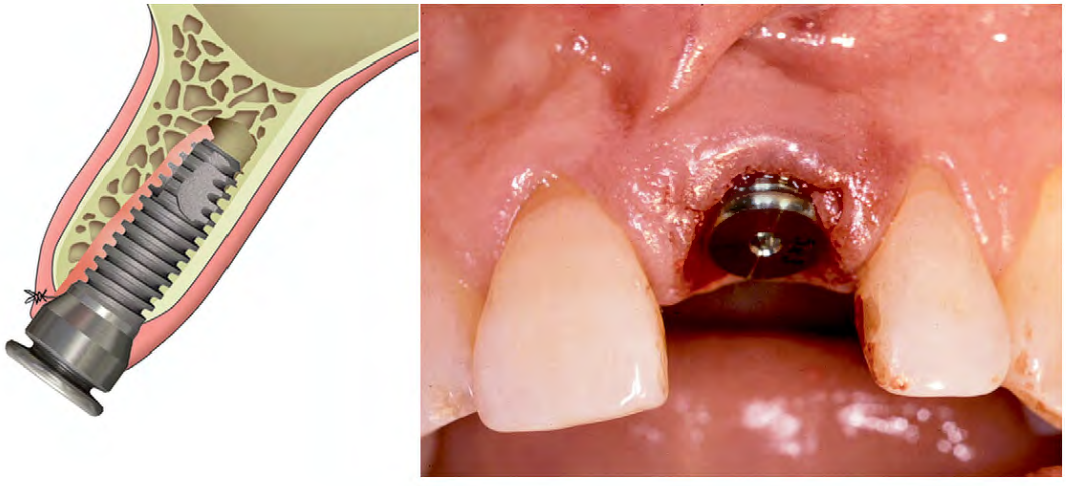
A B

Figure 33-34 The autograft from the osteotomy drills is positioned on the facial aspect of the alveolus, especially near the crestal region.



A B

Figure 33-35 A 15-mm implant (when possible) is threaded into position, under the incisal edge and 3 mm below the facial free gingival margin.



A B

Figure 33-36 A permucosal extension is placed into the implant and fills the gingival void above the crestal bone.

WWW.HIGHDENT.IR

Figure 33-37 After a 4-month healing period, the implant is ready to be restored.

release incision can also be made in the midline to the height of the facial gingival contour desired. The tissue is elevated from the crest of the ridge, and the first-stage cover screw is identified. Once removed, a low-profile healing cap of 3 mm is inserted. A connective tissue graft or AlloDerm is placed around the low-profile healing cap. The crestal tissues are then draped over the healing cap and sutured to the palatal tissue. The tissue heals by secondary intention in the palatal area, and excess tissue forms on the facial and interproximal regions. After 4 to 6 weeks, a stage II uncovery and gingivoplasty (both subtraction techniques) is performed to the contour of the desired crown emergence profile.

An alternative stage II uncovery procedure developed by the author is called the *split-finger technique*.[65] An incision is made in the gingival sulci of the adjacent teeth. The incision is started at the distolingual line angle of the adjacent teeth and forms a loop at the facial emergence location of the implant crown. This creates two "fingers" at least 2 mm in width adjacent to each natural tooth. Once elevated, these two facial fingers will become the facial aspects of the interdental papillae. A central palatally supported finger is also created. The tissues are then elevated, and the implant cover screw is exposed and replaced with an abutment. The palatal finger may then be split into two segments (i.e., the split-finger approach). Each segment is rotated to the interproximal region to support the elevated facial fingers. A 4-0 or 5-0 modified mattress suture positions the papillae in the proper location, next to a transitional crown (Figures 33-39).

When additional height is required, the split-finger technique may be used in conjunction with a connective tissue graft or AlloDerm to further augment the tissue height. This procedure may also be performed at insertion surgery, when a one-stage approach is desired.

Once the soft tissues are subtracted or added to obtain the desired emergence contour, the surgeon has primarily two options to maintain this region. The first option is that a PME abutment may be inserted. Its size and shape should be smaller than the cervical contour of the final crown and extend through the tissue 1 to 2 mm. The emergence of the PME abutment will develop the initial soft tissue form. A wide-profile PME abutment should not be used, because it may cause gingival shrinkage and limit the restoring dentist's ability to shape it into its ideal form. A transitional prosthesis is recontoured to fit over the healing cap for the next few weeks, until the patient is seen for the first prosthetic appointment. The soft tissue heals to the contour of the healing abutment or permucosal device. The patient is referred to the restoring dentist for fabrication of the final crown after 4 to 6 weeks of tissue maturation. This

A B

Figure 33-38 **A,** When the soft tissue at uncovery is level with the height of the papilla, a subtraction technique is used to sculpt the crest of the ridge (usually with a round diamond) and form a scalloped emergence profile around the abutment. **B,** The patient's implant crown replaces the maxillary right lateral incisor and maintains the papilla height long term.

WWW.HIGHDENT.IR

Box 33-2 Flow Pattern for Maxillary Anterior Single-Tooth Replacement

I. Treatment plan
  A. Single-tooth implant FPD
    1. Mobile abutments
    2. Inadequate bone height, including adjacent teeth
    3. Inadequate mesiodistal space
    4. Fear of multiple surgeries
    5. Cost
  B. Adjacent remaining teeth
II. Transitional device
  A. Removable
    1. Essix
    2. Flipper
    3. Clasp, cast RPD with rests
  B. Resin bonded
  C. Orthodontic wire and denture teeth
  D. Transitional cantilevered prosthesis from adjacent tooth requiring crown
III. Implant site development
  A. Orthodontics
    1. Space (mesiodistal)
    2. Tooth extrusion
    3. Soft tissue enhancement
  B. Soft tissue augmentation
  C. Bone augmentation
IV. Soft tissue incisions, wire, and reflection
  A. Papillae saving
  B. Ridge elevation
V. Implant size
  A. Implant 1.5 mm from each adjacent tooth
  B. Implant 1.5 mm from facial plate and 1 mm from palatal plate
  C. Usual size 4.3 mm for centrals, 3.0 to 3.7 mm for laterals, 4.2 mm for canines
VI. Implant body position
  A. Midmesiodistal—if central incisor, then incisive foramen size and position evaluated
  B. Midfaciopalatal
  C. Angulation—one half in front, two thirds behind incisal edge of implant crown to midfaciopalatal
  D. Depth—position should be 3 mm below facial free gingival margin of future implant crown
VII. Soft tissue and implant healing approach
  A. Primary closure
  B. One stage—PME added
  C. Soft tissue crest elevated—PME added and covered with soft tissue
  D. Nonfunctional immediate restoration
    1. Abutment added
    2. Cemented crown out of occlusion
VIII. Stage II uncovery and soft tissue
  A. Subtraction technique (canine soft tissue drape)
  B. Addition technique
    1. Split-finger approach
    2. Crest elevated and PME added as "tent pole" for soft tissue
IX. Abutment selection
  A. Manufacturer straight abutment
  B. Custom abutment
    1. Laboratory prepared
    2. Porcelain on abutment
X. Final crown

FPD, Fixed partial denture; *RPD,* removable partial denture; *PME,* permucosal extension.

WWW.HIGHDENT.IR

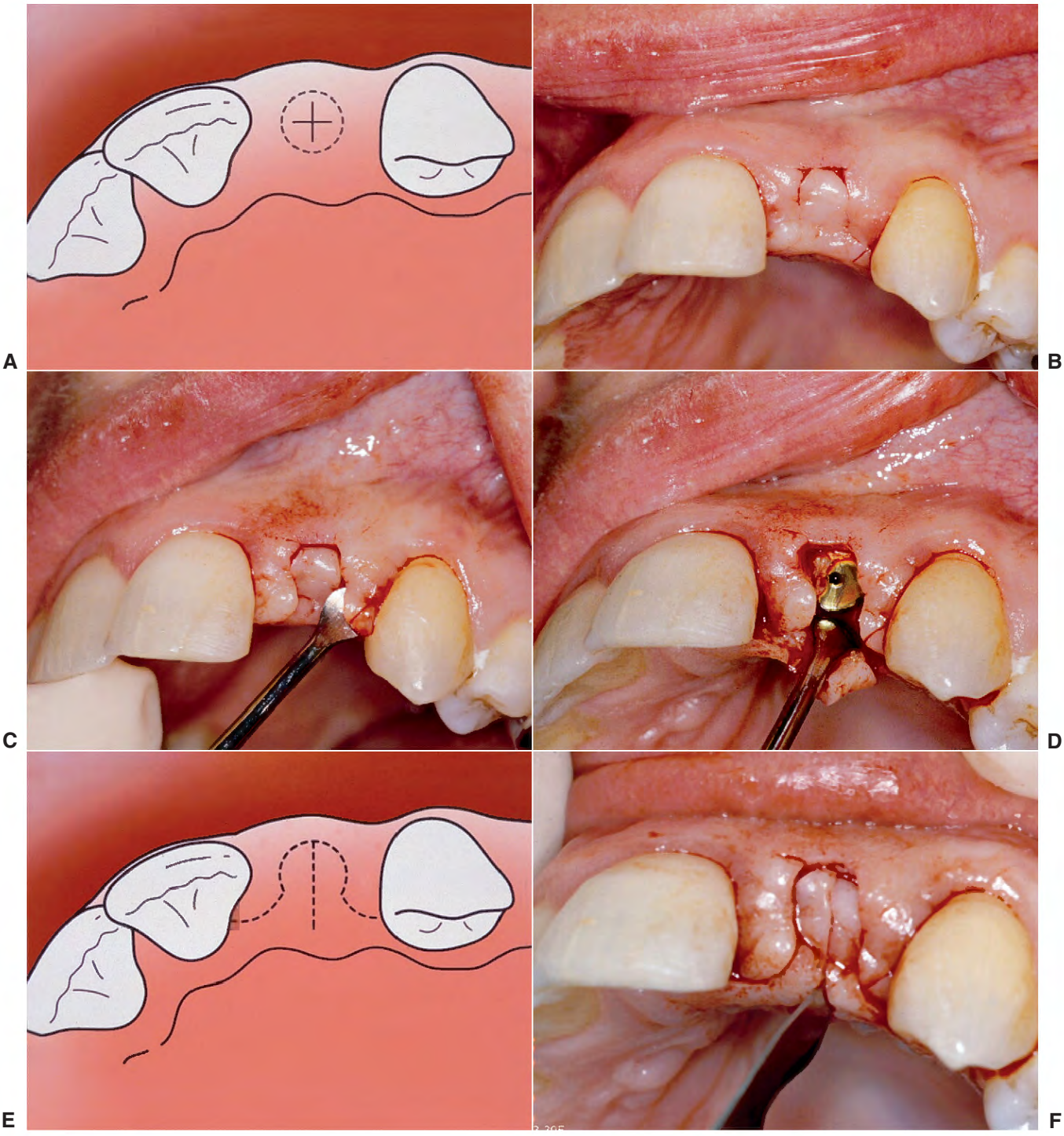


Figure 33-39 **A,** When the desired papilla height is almost perfect, a split-finger technique may be used. **B,** An incision is made in the sulcus of the adjacent teeth and continues from the palatal aspect of each tooth for 1.5 to 2.0 mm and loops to the facial, parallel to the adjacent interproximal teeth. The incisions then connect on the facial aspect at the desired midfacial emergence of the crown. **C,** The facial fingers are elevated facially. **D,** The palatal finger is reflected palatally. **E,** The palatal finger is split into two sections (mesial and distal). **F,** This creates four interfacing fingers (two on the facial and one toward the palate).

Continued

is the simplest option when using the team approach to implant dentistry.

In addition to the implant insertion, the surgeon may elect to make an impression to aid the restoring dentist in the fabrication of the abutment and crown. An additional silicone impression may be made at the implant insertion appointment or at the uncovery appointment, with an indirect impression transfer placed into the implant body. The indirect impression transfer is removed from the implant, connected to

WWW.HIGHDENT.IR

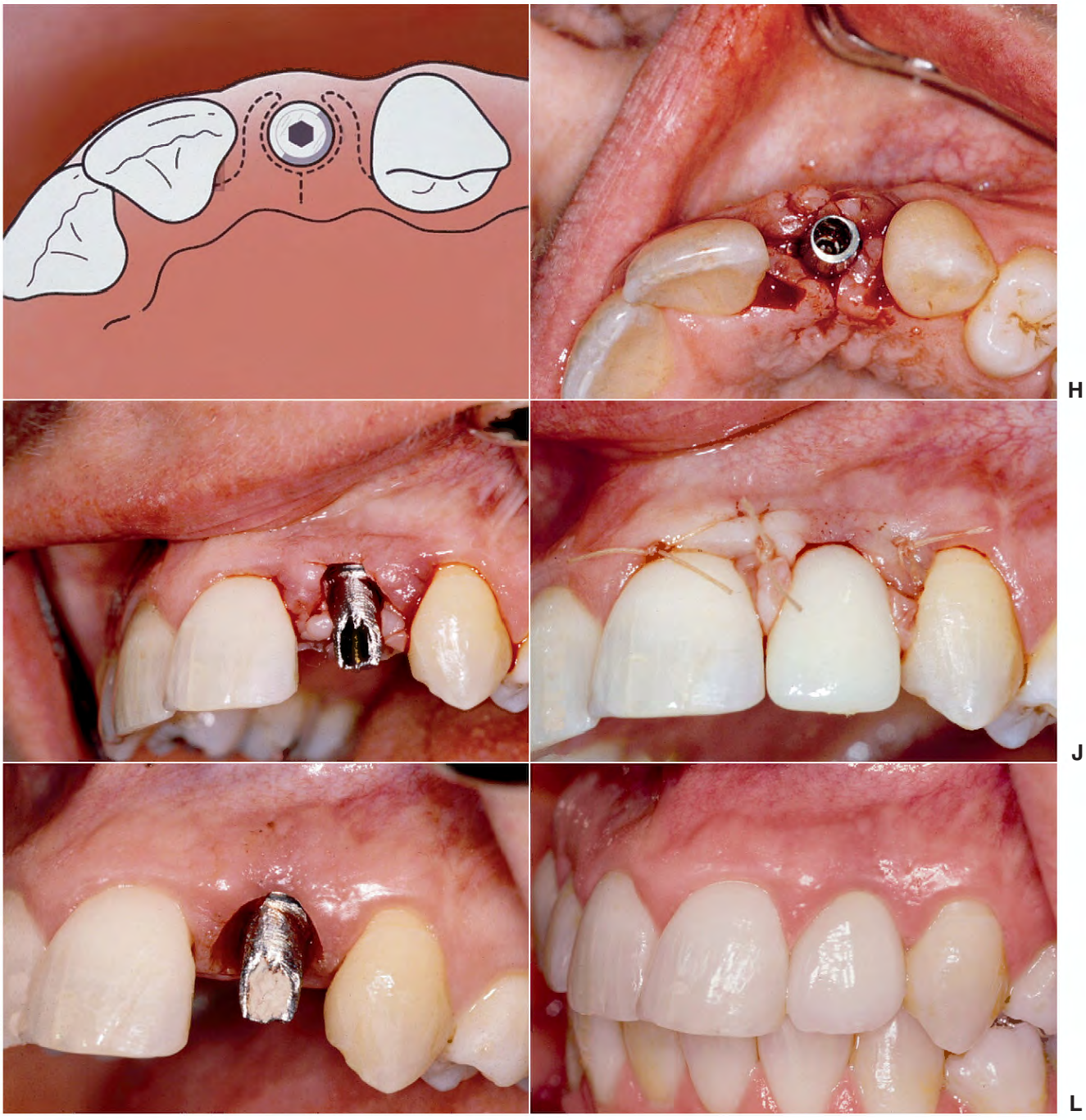


Figure 33-39, cont'd **G,** The implant abutment (or permucosal extension) is added, and each split palatal finger is positioned under the facial corresponding finger. **H,** The split palatal fingers are rotated and support the appropriate facial finger. **I,** The implant abutment and elevated facial fingers are in position. **J,** The temporary crown is cemented, and the fingers are sutured into position. **K,** After soft tissue maturation, the final crown is fabricated. **L,** The final crown in position with properly developed interdental papillae.

the implant body analog, and reinserted into the impression. A PME is inserted into the implant, and the patient is discharged.

A master cast is made in the laboratory from the impression. The laboratory may fabricate a soft tissue model and/or fabricate or select the abutment and fabricate a transitional or possibly final prosthesis. The soft tissue model may be modified in the laboratory to represent the contour of the desired final soft tissue and restoration. An abutment is placed and modified in the implant body analog, and a provisional restoration is fabricated to the soft tissue contour created on the model.

The patient returns to the restoring dentist after 14 days or more. The laboratory-created final abutment and temporary restoration are inserted, and the soft tissues are allowed to achieve full maturation around the best contours of the transitional restoration before

WWW.HIGHDENT.IR

Figure 33-40 The most common esthetic complication is the inadequate soft tissue drape around an implant crown. The patient's left central incisor exhibits black interproximal spaces.

making the final restoration. The advantage of this procedure is that the laboratory may select and prepare the final abutment, or it may fabricate a custom post and fabricate the transitional prosthesis with an ideal emergence profile to encourage maturation of the soft tissue in its final position.

When the surgeon is also the restoring dentist or is able to make a fixed transitional crown; the initial restoration appointment may be combined with the stage II uncovery procedure. A prefabricated, machined two-piece abutment is placed into the implant body at the conclusion of the soft tissue recontouring. The antirotational hex is engaged, and the abutment screw is hand tightened to approximately 5 to 10 N-cm (or moderate hand force). A periapical radiograph is taken of the crest module and abutment connection region to ensure proper component fit. Once confirmed, the abutment screw is then secured at 20 to 30 N-cm, while a hemostat holds the abutment with a countertorque resistance to reduce the shear forces to the crestal bone. The post is prepared in situ similarly to an anterior tooth preparation, with juxtagingival or supragingival margins. A transitional single crown is then fabricated completely out of occlusion. The soft tissue is then permitted to heal, using the transitional crown to help form the soft tissue drape.

COMPLICATIONS

The primary esthetic complications of maxillary anterior single-tooth implants include interdental papillae deficiency and gingival shrinkage after crown delivery.

Interdental Papilla Deficiency

The interproximal CEJ of a natural tooth exhibits a reverse scallop toward the incisal edge. The same pattern is followed by the alveolar interproximal bone, which is more coronal in the interproximal regions than in the facial or lingual plates. As a consequence, the probing depth in the papilla region of a natural tooth is quite similar to the facial or palatal probing depths. Interproximal bone around an implant does not follow such a contour. As a result, the interdental papillae, which look natural and rise to fill the interproximal regions between healthy adjacent teeth, exhibit greater probing depths than the other surfaces of the implant crown. In fact, because the interproximal bone height also may be lost next to the adjacent teeth, the dental and implant papilla also correspond to a greater proximal probing depth next to the natural tooth. A greater sulcus depth increases the risk of shrinkage after gingivoplasty, or later, even with good daily care. As a result, even years later the tissue may shrink and result in a poor interproximal esthetic situation (Figure 33-40).

As previously addressed, four surgical time sequences exist to address the interproximal tissue height: (1) before a bone graft with a connective tissue graft; (2) in conjunction with a bone graft, often using an acellular tissue graft (i.e., AlloDerm); (3) at implant insertion, with an elevation of the tissue over a PME; and (4) at implant uncovery (i.e., split-finger technique). There are several other methods to improve the soft tissue drape. These approaches used to modify the soft tissue are prosthetic-related methods.

The most common prosthodontic solution to alleviate soft tissue limitations is helpful when soft tissue surgery has not recreated an ideal interproximal papilla height. The interproximal region may be treated similarly to the pontic interproximal region of a three-unit FPD (Figure 33-41). Rarely are interdental papillae present next to the pontics of a fixed prosthesis. Instead, rather than raising the tissue to the interproximal contact of the crown, the interproximal contact is extended toward the tissue, and the cervical region of the pontic is slightly overcontoured. A similar approach may be applied to the single-tooth implant. The interproximal contacts of the adjacent teeth are recontoured, especially on the palatal line angle, to become oblong and extend toward the tissue. The contact areas of the single-tooth crown are extended, especially on the palatal line angle, toward the gingiva. The cervical region of the single-tooth implant is slightly overcontoured in width, similarly to the pontic of a fixed prosthesis. This concept does slightly compromise the interproximal esthetics. The papilla is not as high next to the implant crowns as it is between the natural teeth, and the cervical width of the crown is 0.5 mm wider.

However, the sulcus depth is reduced on the tooth and implant crown, and the daily hygiene conditions are improved. In addition, long-term shrinkage of the tissue is less likely to occur. This option should be the method of choice whenever possible and especially when the high lip position during smiling does not display the gingival regions around the teeth.

WWW.HIGHDENT.IR

Figure 33-41 **A,** A prosthetic solution for inadequate papilla height is to lower the interproximal contact of the crown by reshaping the adjacent teeth. **B,** The canine implant crown fills the space and eliminates the interdental spaces caused by the lack of papilla height.

SUMMARY

The replacement of a single tooth in the premaxilla is challenging because of the highly specific soft and hard tissue criteria, in addition to all other esthetic, phonetic, functional, and occlusal requirements. Anterior tooth loss usually compromises ideal bone volume and position for proper implant placement. Implant diameter, compared with that of natural teeth, results in challenging cervical esthetics. Unique surgical and prosthetic concepts are implemented for proper results. In spite of all the technical difficulties that the restoring dentist may face, the anterior single-tooth implant is the modality of choice to replace a missing anterior maxillary tooth.

References

1. Priest GF: Failure rates of restorations for single tooth replacements, *Int J Prosthodont* 9:38-45, 1996.
2. Watson MT: Implant dentistry, a 10 year retrospective report, *Dent Prod Rep* 25-32, December 1996.
3. Scurria MS, Bader JD, Shugars DA: Meta-analysis of fixed partial denture survival: prostheses and abutments, *J Prosthet Dent* 79:459-464, 1998.
4. Aquilino SA, Shugars DA, Bader JD et al: Ten year survival rates of teeth adjacent to treated and untreated posterior bounded edentulous spaces, *J Prosthet Dent* 85:455-460, 2001.
5. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
6. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
7. Misch CE, D'Alessio R, Misch-Dietsh F: Maxillary partial anodontia and implant dentistry—maxillary anterior partial anodontia in 255 adolescent patients: a 15-year retrospective study of 276 implant site replacement, *Oral Health* 95:45-57, 2005.
8. Wennstrom JL, Ekestubbe A, Grondahl K et al: Implant-supported single-tooth restorations: a 5-year prospective study, *J Clin Periodontol* 32:567-574, 2005.
9. Creugers NH, Kreuler PA, Snoek RJ et al: A systematic review of single tooth restorations supported by implants, *J Dent* 28:209-217, 2000.
10. Lindhe T, Gunne J, Tillberg A et al: A meta-analysis of implants in partial edentulism, *Clin Oral Implants Res* 9:80-90, 1998.
11. Zarone F, Sorrentino R, Vaccaro F et al: Prosthetic treatment of maxillary lateral incisor agenesis with osseointegrated implants: a 24-39 month prospective clinical study, *Clin Oral Implants Res* 17:94-101, 2006.
12. Kemppainen P, Eskola S, Ylipaavalniemi P: A comparative prospective clinical study of two single tooth implants: a preliminary report of 102 implants, *J Prosthet Dent* 77:382-387, 1997.
13. Kan JY, Rungcharassaeng K: Immediate implant placement and provisionalization of maxillary anterior single implants: a surgical and prosthodontic rationale, *Pract Periodont Aesthet Dent* 12:817-824, 2000.
14. Choquet V, Hermans M, Adriaenssens P et al: Clinical and radiographic evaluation of the papilla level adjacent to single tooth dental implants: a retrospective study in the maxillary anterior region, *J Periodontol* 72:1364-1371, 2001.
15. Groisman M, Frossard WM, Ferreira H et al: Single tooth implants in the maxillary incisor region with immediate provisionalization: 2-year prospective study, *Pract Proced Aesth Dent* 15:115-122, 2003.
16. Tarnow D, Elian N, Fletcher P et al: Vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants, *J Periodontol* 74:1785-1788, 2003.
17. Kan JY, Rungcharassaeng K, Umezu K et al: Dimensions of peri-implant mucosa: an evaluation of maxillary anterior single implants in humans, *J Periodontol* 74:557-562, 2003.
18. Becker W, Ochsenbein C, Tibbetts L et al: Alveolar bone anatomic profiles as measured from dry skulls: clinical ramifications, *J Clin Periodontol* 24:727-731, 1997.
19. Tarnow DP, Eskow RM: Preservation of implant esthetics, soft tissue and restorative considerations, *J Esthet Dent* 8:12-19, 1996.
20. Tarnow DP, Cho SC, Wallace SS: The effect of inter-implant distance on the height of inter-implant bone crest, *J Periodontol* 71:546-549, 2000.
21. Tarnow DP, Magner AW, Fletcher P: The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal papilla, *J Periodontol* 63:995-996, 1992.

WWW.HIGHDENT.IR

22. Misch CE, Wang HL: The procedures, limitations and indications for small diameter implants and a case report, *Oral Health* 94:16-26, 2004.
23. Misch CE: Treatment options for congenitally missing lateral incisor—a case report, *Dent Today* 23:92-95, 2004.
24. Vigolo P, Givani A, Majzoub Z et al: Clinical evaluation of small-diameter implants in single-tooth and multiple-implant restorations: a 7 year retrospective study, *Int J Oral Maxillofac Implants* 19:703-709, 2004.
25. Sterett JD, Olivier T, Robindon F et al: Width and length ratios of normal clinical crowns of maxillary anterior dentition in man, *J Clin Periodontol* 26:153-157, 1999.
26. Wheeler RC: *A textbook of dental anatomy and physiology*, ed 4, Philadelphia, 1965, Lea & Febiger.
27. Jemt T, Lekholm U: Single implants and buccal bone grafts in the anterior maxilla: measurements of buccal crestal contours in a 6-year prospective clinical study, *Clin Implant Dent Relat Res* 7:127-135, 2005.
28. Cardaropoli G, Lekholm U, Wennstrom JL: Tissue alterations at implant-supported single-tooth replacements: a 1-year prospective clinical study, *Clin Oral Implants Res* 17:165-171, 2006.
29. Jemt T, Ahlberg G, Henriksson K et al: Implant changes of anterior clinical crown height in patients provided with single-implant restorations after more than 15 years of follow-up, *Int J Prosthodont* 19:455-461, 2006.
30. Perel S, Sullivan P, editors: *Esthetics and osseointegration*, Chicago, 1994, Quintessence.
31. Saadoun AP, Sullivan DY, Krischek M et al: Single tooth implant management for success, *Pract Perio Rest Dent* 6:73-82, 1994.
32. Malevez C, Hermans M, Daelemans P: Marginal bone levels at Brånemark system implants used for single tooth restoration. The influence of implant design and anatomical region, *Clin Oral Implants Res* 7:162-169, 1996.
33. Grunder U: Stability of the mucosal topography around single-tooth implants and adjacent teeth: 1-year results, *Int J Periodontics Restorative Dent* 20:11-17, 2000.
34. Berglundh T, Lindhe J, Ericsson I et al: The soft tissue barrier at implants and teeth, *Clin Oral Implants Res* 2: 81-90, 1991.
35. Yukna RA: Periodontal considerations for dental implants. In Block MS, Kent JN, editors: *Endosseous implants for maxillofacial reconstruction*, Philadelphia, 1995, WB Saunders.
36. Kois JC: Predictable single-tooth peri-implant esthetics: five diagnostic keys, *Compend Contin Educ Dent* 25:895-896, 898, 2004.
37. Gotfredsen K: A 5-year study of single tooth replacements supported by the Astra Tech implant: a pilot study, *Clin Implant Dent Relat Res* 6:1-8, 2004.
38. Gomez-Roman G, Kruppenbacher M, Weber H et al: Immediate postextraction implant placement with root-analog stepped implants: surgical procedure and statistical outcome after 6 years, *Int J Oral Maxillofac Implants* 16:503-513, 2001.
39. Barzilay I: Immediate implants, their current status, *Int J Prosthodont* 6:169, 1993.
40. Rosenquist B, Grenthe B: Immediate placement of implants into extraction sockets: implant survival, *Int J Oral Maxillofac Implants* 11:205-209, 1996.
41. Schwartz-Arad D, Chaushu G: The ways and wherefores of immediate placement of implants into fresh extraction sites: a literature review, *J Periodontol* 68:915-923, 1997.
41a. Aravjo MG, Sukekava F, Wennström JL et al: Tissue modeling following implacement in fresh extraction sockets, *Clin Oral Implants Res* 17:615-624, 2006.
42. Kan JYK, Rungcharassaeng K: Site development for anterior single implant esthetics, *Compend Contin Educ Dent* 22:221-232, 2001.
43. Garber DA, Belser VC: Restoration driven implant placement with restoration-generated site development, *Compend Cont Educ Dent* 16:796-804, 1995.
44. Garber DA: The esthetic dental implant-letting the restoration be the guide, *J Am Dent Assoc* 126:319-325, 1995.
45. Saadoun AP, Landsberg CT: Treatment classifications and sequencing for post extraction implant therapy: a review, *Pract Perio Rest Dent* 9:933-941, 1997.
46. Ferrara A, G'Alli C, Mauro G et al: Immediate provisional restoration of postextraction implants for maxillary single tooth replacements, *Int J Periodontics Restorative Dent* 26:371-377, 2006.
47. Schropp L, Isisor F, Kostopoulos L et al: Interproximal papilla levels following early versus delayed placement of single-tooth implants: a controlled clinical trial, *Int J Oral Maxillofac Implants* 20:753-761, 2005.
48. Schropp L, Wenzel A, Kostopoulos L et al: Bone healing and soft tissue contour changes following single tooth extraction: a clinical and radiographic 12-month prospective study, *Int J Periodontics Restorative Dent* 23:313-323, 2003.
49. Tehemar S, Hanes P, Sharawy M: Enhancement of osseointegration of implants placed into extraction sockets of healthy and periodontally diseased teeth by using graft material, an ePTFE membrane or a combination, *Clin Implant Dent Relat Res* 5:193-211, 2003.
50. Botticelli D, Berglundh T, Lindhe J: Hard-tissue alterations following immediate implant placement in extraction sites, *J Clin Periodontol* 31:820-828, 2004.
51. Garber DA, Salama H, Salama MA: Two-stage versus one-stage—is there really a controversy? *J Periodontol* 72: 417-421, 2001.
52. Funato A, Salama MA, Ishikawa T et al: Timing, positioning, and sequential staging in esthetic implant therapy: a four-dimensional perspective, *Int J Perio Rest Dent* 27:313-323, 2007.
53. Schwartz-Arad D, Chaushu G: Placement of implants into fresh extraction sites: 4 to 7 years retrospective evaluation of 95 implants, *J Periodontol* 68:1110-1116, 1997.
54. Schwartz-Arad D, Chaushu G: Immediate implant placement: a procedure without incisions, *J Periodontol* 69:743-750, 1998.
55. Lang NP, Tonetti MS, Suvan JE et al: Immediate implant placement with transmucosal healing in areas of esthetic priority: a multicenter randomized controlled clinical trial. I. surgical outcomes, *Clin Oral Implant Res* 18:188-196, 2007.
56. Esposito MA, Koukoulopoulou A, Coulthard P et al: Interventions for replacing missing teeth: dental implants in fresh extraction sockets (immediate, immediate-delayed and delayed implants), *Cochrane Database Syst Rev* (4):CD005968, 2006.
57. Schwartz-Arad D, Laviv A, Levin L: Survival of immediately provisionalized dental implants placed immediately into fresh extraction sockets, *J Periodontol* 78:219-223, 2007.
58. Becker W, Dahlin C, Becker BE et al: The use of e-PTFE barrier membranes for bone promotion around titanium implants placed into extraction sockets, a prospective

WWW.HIGHDENT.IR

multicenter study, *Int J Oral Maxillofac Implants* 9:31-40, 1994.
59. Ogiso M, Tabata T, Lee RR et al: Delay method of implantation enhances implant bone binding, a comparison with the conventional method, *Int J Oral Maxillofac Implants* 10:415-420, 1995.
60. Salama H, Salama M, Garber DA et al: Techniques for developing optimal peri-implant papillae within the esthetic zone, part I: guided soft tissue augmentation the three-stage approach, *J Esthet Dent* 7:3-9, 1995.
61. Salama H, Salama M, Garber D et al: The interproximal height of bone: a guidepost to predictable aesthetic strategies and soft tissue contours in anterior tooth replacement, *Pract Periodontics Aesthet Dent* 10:1131-1141, 1998.
62. Palacci P: Peri-implant soft tissue management: papilla regeneration technique. In Palacci P, Ericsson I, Engstrand P et al, editors: *Optimal implant positioning and soft tissue management for the Brånemark system*, Chicago, 1995, Quintessence.
63. Becker W, Becker B: Flap designs for minimization of recession adjacent to maxillary anterior sites, a clinical study, *Int J Oral Maxillofac Implants* 11:46-54, 1996.
64. Salama H, Salama M, Garber D et al: Developing optimal peri-implant papillae within the esthetic zone: guided soft tissue augmentation, *J Esthet Dent* 7:125-129, 1995.
65. Misch CE, Al-Shammari KF, Wang HI: Creation of interimplant papillae through a split-finger technique, *Implant Dent* 13:20-27, 2004.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 34

Premaxilla Surgery: Implant Insertion, Bone Spreading, Nasal Floor Elevation, and Incisive Foramen Implants

Carl E. Misch

INTRODUCTION

Seventeen percent of the U.S. population (30 million adults) has no maxillary natural teeth. A large segment of the population is also missing multiple maxillary anterior teeth, often from trauma or failed fixed partial dentures (FPDs). Therefore the premaxilla is a region often in need of implant abutments for either fixed or overdenture prostheses. In a 20-year review of the literature by Goodacre et al., restorations associated with the edentulous maxillae have the highest early implant failure rate (19% for overdentures and 10% for fixed prostheses). In comparison, mandibular overdentures or fixed restorations have some of the highest implant survival rates (3% failure rate).[1]

Several factors affect the condition of the edentulous maxilla and may result in a decrease in implant survival or an increase in surgical complications. After tooth loss, the facial cortical plate rapidly resorbs during initial bone remodeling, and the anterior ridge loses 25% of its width within the first year, as well as 40% to 50% within the next 3 to 5 years, mostly at the expense of the labial contour. As a result, the residual available bone migrates to a more palatal position.[2-7] The farther forward the maxillary anterior teeth are positioned from the more palatal bone position implants, the greater the moment force leverage on the bone-implant interface, abutment screws, and implants. Coupled with an angled force in both centric and excursions, more stress is transmitted to premaxillary implants than those in anterior mandibles. This often mandates more implants and larger-diameter implants with bone augmentation by bone-spreading or bone graft procedures before or in conjunction with implant placement.

In most patients with available bone, the bone is less dense in the anterior maxilla than in the anterior mandible. The maxilla presents thin porous bone on the labial aspect, very thin porous cortical bone on the floor of the nasal and sinus region, and a more dense cortical bone on the palatal aspect.[8,9] The trabecular bone in the premaxilla is usually fine and less dense than the anterior region of the mandible. This usually results in more overload implant failures or crestal bone loss. However, this type of bone lends itself to bone spreading or a compaction technique to insert implants.

In the anterior maxilla, the moderately resorbed bone categories of Division B to C–w often remain narrow almost to the floor of the nose; therefore osteoplasty to gain width is inappropriate. Once the tissues are reflected, the unusual anatomy may confuse the surgeon, and the resultant angulation of implant insertion complicates the restoration of the prosthesis (Figure 34-1). On occasion, osteoplasty to gain bone width results in a Division C–h or D bone volume. The incisive foramen may be used as an implant site in a C–w or C–h bone volume. Nasal elevation and implant insertion may also modify the opposing landmark in C–h bone volume. Therefore unique force factors and implant insertion procedures are more often observed in the anterior maxilla compared with the anterior mandible.

PREMAXILLA EDENTULOUS TREATMENT PLANS

Key Implant Positions

If a patient desires a fixed restoration, implants are required whenever any of the following three combinations of adjacent teeth are missing: (1) the first premolar, canine, and lateral incisor; (2) the canine,

WWW.HIGHDENT.IR

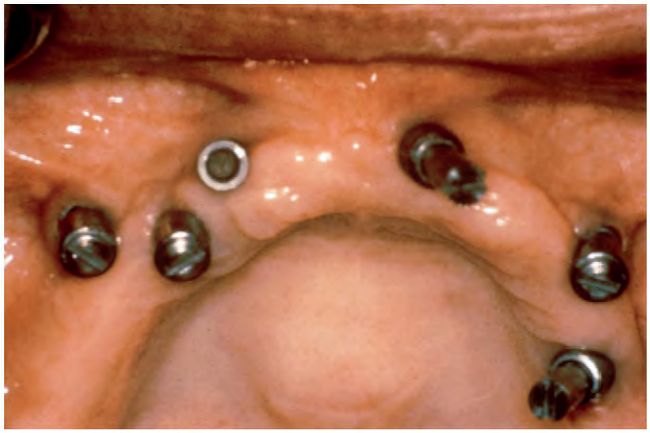


Figure 34-1 The maxillary bone often remains narrow to the floor of the nose. As a consequence, osteoplasty to gain width is often inappropriate. The surgical placement of implants in bone of minimal width is more challenging and may result in poor angulation and difficulty in restoring the implants.

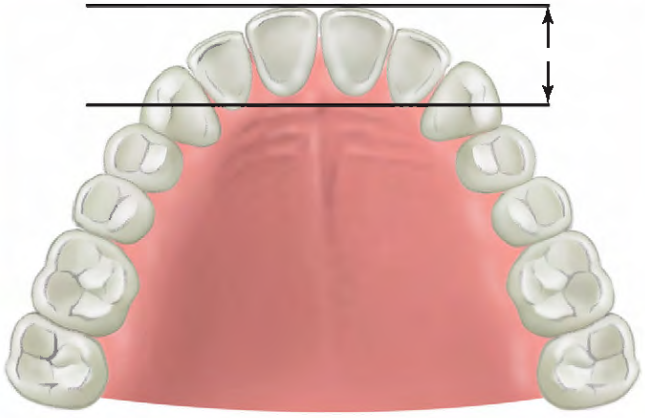


Figure 34-2 Two parallel lines perpendicular to the midline are drawn in the premaxilla to determine the dentate arch form. The anterior line is through the facial aspect of the central incisors. The posterior line is drawn through the tips of the canine cusps. A distance between these two lines of less than 8 mm is a square dentate arch form. An ovoid dentate arch form is 8 to 12 mm. A tapered dentate arch is more than 12 mm.

lateral incisor, and central incisor; and (3) the canine, first premolar, and second premolar. When any of these conditions exist, a fixed restoration is contraindicated because of the length of the span (three pontics), the amount of force (forces greater in the canine region compared with the anterior region), and the direction of the force (angled forces to the canine region).[10]

Under the condition of a missing canine and two adjacent teeth, at least two implants are indicated to support an independent fixed restoration (usually in the terminal positions of the span to eliminate cantilever forces). Therefore the canine implant position is one of the more important key positions in an edentulous premaxilla.

When one canine region cannot be used to place an implant in the edentulous premaxilla, at least one implant on each side of the missing canine is required to compensate for this vital position (a first premolar and lateral incisor implant). An incisor and canine position implant in the contralateral section can be splinted to these other two implants to act as abutments for the fixed or overdenture restoration in the edentulous premaxilla.

PREMAXILLA ARCH FORM AND IMPLANT NUMBER

The distance between two horizontal lines, drawn from the two canine tips and the facial of the central incisors, determines the dental arch form in the anterior maxilla. (Figure 34-2). A distance less than 8 mm corresponds to a square dental arch form. Lateral and central incisors are not cantilevered very much facially from the canine position, and therefore mandibular excursions and occlusal forces exert less stress on the canine implants.

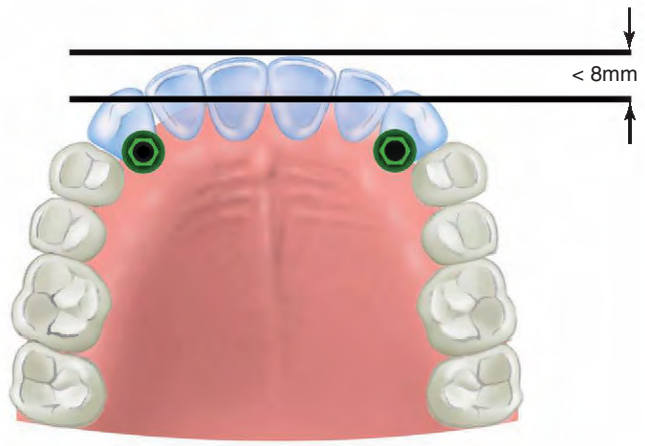


Figure 34-3 In a square arch form, implants in the canine region splinted to posterior implants may be sufficient with low patient force factors because the anterior cantilever is less than 8 mm.

Implants in the canine positions to replace the six anterior teeth may suffice when the force factors are low and if they are splinted to additional posterior implants (Figure 34-3).

An ovoid dentate arch form corresponds to a distance from the canine to central incisor horizontal lines between 8 and 12 mm. At least three implants should be inserted into the premaxilla, one in each canine and preferably one in a central incisor position (Figure 34-4). The central incisor position increases the anteroposterior (A-P) distance from the canine to central and provides improved biomechanical support to the prosthesis. In long-term edentulous maxillae, this most likely will require bone augmentation before implant insertion (Figure 34-5). When patient force factors are low to moderate, the anterior implant may be positioned in a lateral incisor site.

WWW.HIGHDENT.IR

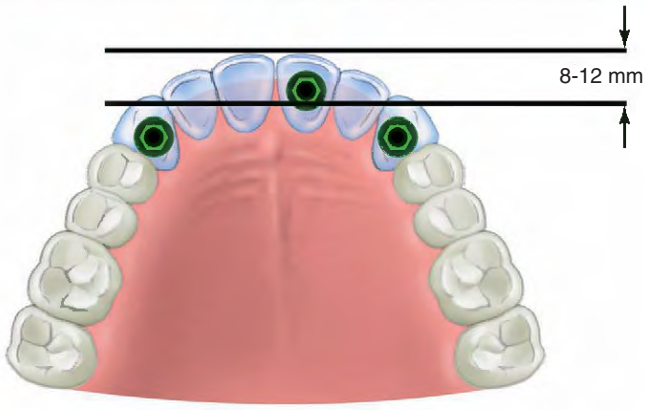


Figure 34-4 An ovoid dentate arch form has implants in the canine and an additional implant in the premaxilla. When posterior teeth are missing, additional posterior implants splinted to the anterior component are necessary.

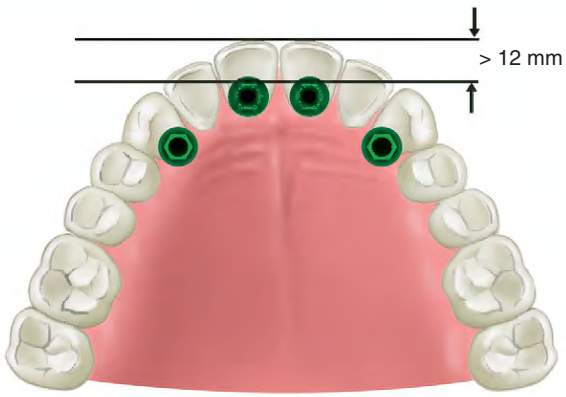


Figure 34-6 A tapered dentate arch form is subject to greater anterior moment forces, because the teeth are cantilevered farthest from the canines. As a result, two canine and two additional implants are usually indicated in the premaxilla.

Figure 34-5 **A,** An FP-3 prosthesis for an ovoid arch form. **B,** An intraoral view of maxillary and mandibular FP-3 prostheses. **C,** A panoramic radiograph of the FP-3 prostheses. The maxilla was restored with an iliac crest graft and bilateral sinus grafts. Three implants are found in the premaxilla (canines and central incisor) splinted to posterior implants. **D,** The high smile line lip dynamics of the maxillary FP-3 prosthesis.

When the distance between the canine and central incisor horizontal lines is greater than 12 mm, the dentate arch form is tapering and the restoration of a tapered dental arch form places the greatest forces on anterior implants, especially during mandibular excursions (Figure 34-6). The anterior teeth create a significant facial cantilever from the canine position. As such, four implants should be considered to replace the six anterior teeth. When more than six anterior teeth are missing, additional posterior implants should also be splinted to the anterior segment (Figure 34-7).

As a consequence of bone resorption, the residual ridge form becomes more squared. The edentulous ridge arch form may be different from the dentate arch form (which is determined by the final teeth position in the premaxilla and not the arch shape of the residual

WWW.HIGHDENT.IR

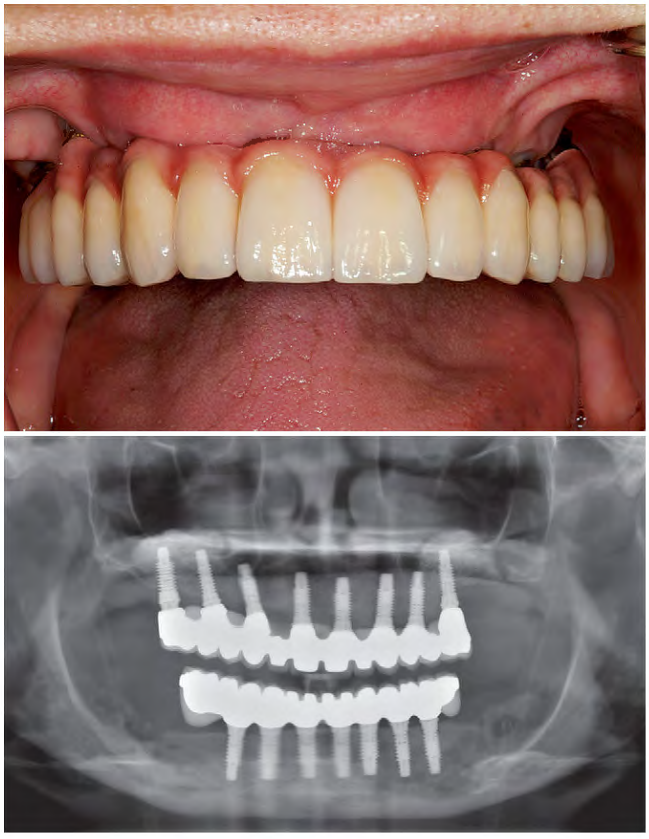


Figure 34-7 **A,** An intraoral view of the maxillary FP-3 prosthesis. The edentulous maxilla with an ovoid to tapered dentate arch form should have three to four implants in the premaxilla and additional posterior implants to support a full-arch prosthesis. **B,** A panoramic radiograph illustrating a cantilevered restoration in the mandible and a fixed maxillary restoration with four anterior implants and four posterior implants splinted together without a cantilever.

ridge). The number and position of implants are related to the arch form of the final dentition (restoration), not the existing edentulous arch form. The worst-case scenario is a patient requiring restoration of a dental tapered arch form with a square residual ridge form. When force factors are greater than usual in any arch form, four implants in the premaxilla should be splinted together and share any lateral forces during excursions. In this patient condition, four implants in the premaxilla are required to compensate for the cantilevered anterior tooth position. These implants should be connected to additional posterior implants, preferably to include implants as far distal as the second molar sites, to increase the A-P position of the implants (Figure 34-8).

The edentulous square-to-ovoid residual bone arch form dimension often does not accommodate interimplant spacing for more than four anterior implants. The largest interimplant distance is typically found in a tapered arch form. As the radius of the circle becomes smaller (from labial resorption, arch shape, and/or patient size), the premaxilla interimplant distance is reduced. As a result, no more than four implants are

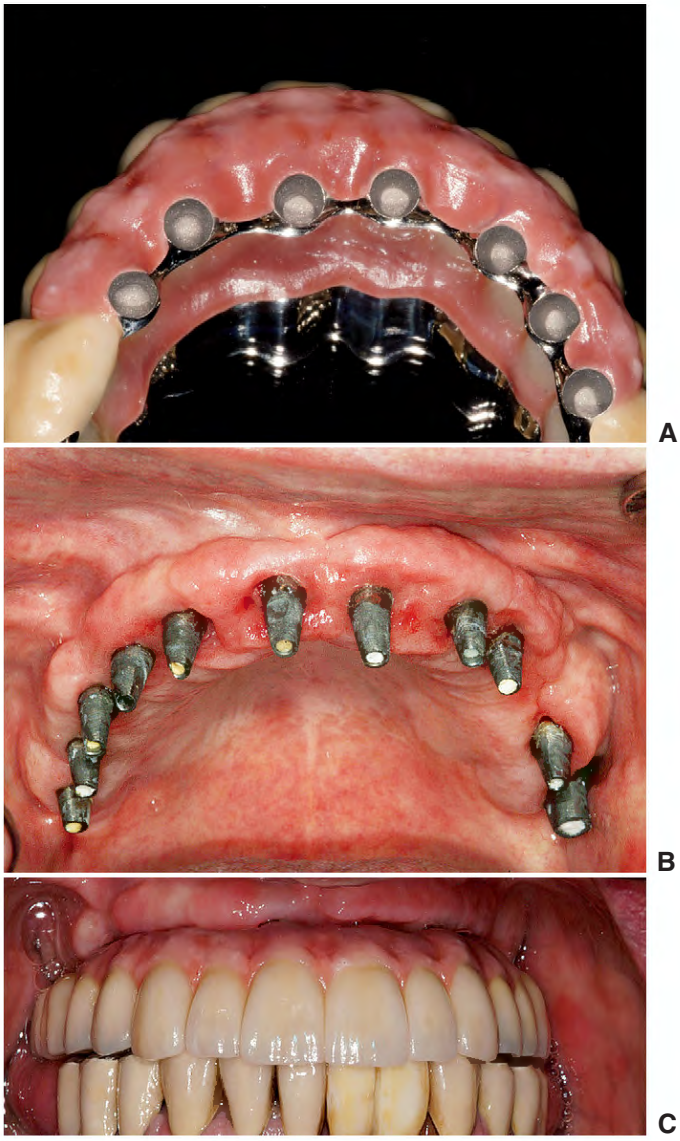


Figure 34-8 **A,** A square residual ridge arch may need to be restored with a tapered dentate arch form. As a result, the prosthesis is cantilevered from the more palatally positioned implants. **B,** An intraoral view of a tapered dentate arch form and a square residual ridge arch form. The four premaxillary implants should be splinted to posterior implants to compensate for the facial cantilever. **C,** Intraoral view of the prosthesis. A greater number of implants is indicated to compensate for the biomechanical force factors.

typically used to replace the anterior six teeth, regardless of the force factors.

Restorations with posterior pontics that are cantilevered should not be placed on maxillary anterior implants. The forces are two to five times greater in the posterior regions compared with the anterior region of the mouth. A cantilever magnifies these higher bite forces. As a result, the anterior implant abutments with a cantilever may receive a tenfold greater load compared with implants without a cantilever. If posterior teeth also are being replaced in the prosthesis, then additional implants are required. Seven to 10 implants often are inserted to restore a completely edentulous maxilla with

WWW.HIGHDENT.IR

Figure 34-9 When the bone loss occurs in the premaxilla, the smaller radius of the arch mandates only four implants to be positioned from canine to canine.

Figure 34-10 A FP-3 prosthesis does not require an exact tooth position for the implants, because the gingival porcelain separates the implants from the teeth. The implants in the canine positions are slightly more distal than desirable, and the patient's left lateral incisor implant is too mesial. Although the implant positions are not ideal in this intraoral view of the previous FP-3 prosthesis, no esthetic compromise exists.

a fixed prosthesis, especially when opposing natural dentition or a fixed restoration.

It should be noted that most full-arch maxillary prostheses are FP-3 fixed restorations or RP-4 overdentures. In either scenario, mesiodistal implant position does not have to strictly correlate with tooth position. The faciopalatal position may also be more flexible, because the emergence profile of the prosthesis is usually outside of the esthetic zone and because the gingival aspect of the restoration separates the clinical crown from the implant site. As such, the implant sites are determined more for biomechanics, interimplant spacing, or available bone, rather than a strict tooth position (as in an FP-1 prosthesis) (Figures 34-9 and 34-10).

When the natural canines are present and the missing teeth are the four maxillary incisors, the implant number is not as dependent on the dental arch form. Therefore the most common scenario is to place three Division B root forms (3.5 mm wide) in the lateral incisors and one central incisor region. When four anterior teeth are missing in a square dentate arch form, it may be easier to develop acceptable esthetics around central incisor pontics, compared with two implants in the central incisor regions. Therefore when biomechanics are favorable, the treatment plan may include only two implants in the lateral incisor sites. This two-implant treatment option is often designed for a square dental arch form in an older female, with little to no parafunction to fabricate an FP-1 prosthesis and a high lip line, when the natural canines are present.

MULTIPLE ANTERIOR IMPLANT DIAMETER

Several conditions should be considered for the proper implant diameter: tooth size, distance from an adjacent tooth, interimplant distance, facial bone dimension, and loading forces. A primary factor for the implant size when multiple anterior teeth are missing is the distance between the adjacent teeth or implants. The horizontal dimension of a wedge-shaped bone defect around an implant at the crest of the ridge from the biological width, implant design, or occlusal overload ranges from 0.5 to 1.4 mm.[11,12] Therefore when implants are placed adjacent to each other, a minimum interimplant distance of 3 mm is suggested to accommodate for eventual crestal bone loss and maintain interseptal bone levels.[11] This is of importance for an FP-1 restoration because the interseptal bone height in part determines the incidence of presence or absence of a lack of interdental papillae between the teeth or implants, with the most ideal results having a distance of 5 mm or less from the interproximal bone to the interproximal crown contact.[13,14]

Implants with 3 mm or greater interimplant space permit the development of papilla between the implants. When two adjacent teeth are missing in the premaxilla, the natural teeth adjacent to the edentulous span provide the interdental papilla support for proper esthetics, and an FP-1 restoration is often fabricated (Figure 34-11). However, the interdental papilla between the two implants is more difficult to restore.

The usual range of interimplant papillary height is 2 to 4 mm, even when the implants are 3 mm or more apart.[15] Therefore when an FP-1 restoration is desired, the prosthesis design (square-tooth forms) and implant positions may need to be altered accordingly to improve the esthetic result (Figure 34-12). Implants less than 3 mm apart often have a depressed interimplant papilla. As a result the crown contour is modified to a square form to lower the interproximal contact to the soft tissue. Although this technique reduces the black triangular space over the papilla, it is not an ideal result (Figure 34-13).

When four anterior adjacent teeth are missing, the soft tissue drape is usually inadequate to restore the height of the interimplant papillae; therefore the anatomy rarely permits an FP-1 prosthesis design. FP-2 or FP-3 prostheses are most always fabricated, dependent

WWW.HIGHDENT.IR

Figure 34-11 **A,** The two implants are farther than 3 mm apart. The interimplant papilla is easier to develop with the additional space. **B,** The two adjacent implant crowns have distal papilla developed from the adjacent natural teeth. The interimplant papilla is slightly reduced in height; however the crown shape lowers the interproximal contact, and the interproximal region is acceptable.

Figure 34-12 When two implants are adjacent to each other and an FP-1 prosthesis is desired, the implants should be 3 mm or farther apart to help develop the interproximal soft tissue papilla regions. The implants are too loose; therefore the papilla is often reduced in height between the implants, so the crowns should be more square in shape to eliminate the space above the lack of soft tissue height.

on the high lip dynamics of smiling within the esthetic zones (Figure 34-14).

The difference in the emergence profile from an anterior crown between a 4-mm-diameter implant and a 5-mm-diameter implant is negligible and is often not clinically relevant. However, it is more difficult to control the creation of an interdental papilla for FP-1 prosthesis on larger-implant diameters. Therefore when in doubt, a smaller-size diameter implant should be selected in the esthetic zone; thus a 3.5- to 4-mm-diameter implant often is used in the central implant position for an FP-1 prosthesis. Likewise, a 3.0- to 3.5-mm-diameter implant often is used for a lateral incisor FP-1 restoration.

When multiple adjacent teeth are missing in the premaxilla, rarely is an FP-1 prosthesis and ideal soft tissue contour a realistic goal. Instead, most often FP-2 restorations are fabricated for low smile line patients and FP-3 restorations for ideal to high smile line patients. The exact mesiodistal tooth position is not required for an FP-2 or FP-3 restoration or a removable overdenture. It is more important to have adequate width of bone on all the sides of the implants. Therefore the interproximal spaces of bone may be used without complication in either the anterior or posterior regions of the mouth. As a result, the mesiodistal position of the implants is often less specific in the edentulous premaxilla.

MAXILLARY OVERDENTURE TREATMENT PLANS

The edentulous maxilla has the lowest implant survival for removable implant restorations.[1,16,17] All reports concur that maxillary bone tends to be of poorer quality and volume and presents several biomechanical disadvantages. To compensate for the poor local conditions, a greater number of implants should be treatment planned, along with a greater A-P distance. With these concerns in mind, the minimum implant number for a completely edentulous maxillary overdenture prosthesis is four implants splinted in the premaxilla. The most usual sites are one central (or lateral) incisor position, bilateral canine positions, and at least one in the contralateral central or premolar site. Additional implants are often indicated when force factors are greater or for a RP-4 prosthesis overdenture.

WWW.HIGHDENT.IR

Figure 34-13 **A,** When four adjacent incisors are missing, the soft tissue drape is often inadequate in the interimplant region. **B,** When four anterior teeth are missing, the papillae are often depressed, and square-crown forms reduce the height requirement of the interimplant papillae.

Figure 34-14 FP-3 prostheses are often the more adequate prosthetic option to restore the interproximal papilla.

Figure 34-15 The flange on a removable prosthesis may be removed to evaluated the volume of graft required for lip support for a fixed prosthesis.

PREMAXILLA SURGERY

Maxillary Lip Support

The maxillary lip support above a fixed prosthesis should be evaluated before surgery. The maxillary lip should be more facial than the mandibular lip at repose, yet a depressed philtrum should be maintained, especially in a younger patient. As a general rule, when a vertical line is drawn along the patient profile, the maxillary lip should be 1 to 2 mm in front of the lower lip, and the chin should be 2 mm behind the lower lip position when the patient has the jaw in a vertical rest position. If the patient wears a labial flange on the removable prosthesis that provides acceptable maxillary lip support, or one exists at the prosthetic try-in, and a fixed prosthesis is desired, then a soft tissue template may be fabricated from the removable prosthetic try-in to indicate the soft tissue support required (Figures 34-15 and 34-16).

When the maxillary lip requires greater horizontal support, yet adequate bone is present to place implants, an acellular tissue graft (AlloDerm) and/or or dense hydroxyapatite (HA) graft may be used on the facial aspect of the bone before closure of the soft tissue over the implant (Figure 34-17). A bone graft may be used to support the maxillary lip above the prosthesis. This is often indicated when insufficient bone is present for the proper implant size, position, and/or number (Figure 34-18). The added labial tissue thickness will support the maxillary lip above the cervical regions of the teeth, increase the crestal tissue thickness, and improve ridge contour. In addition, the soft tissue augmentation to support the maxillary lip may also create sufficient volume of tissue to allow the sculpture of an interdental papilla around the implant crowns at stage II uncovery.

An RP-5 or RP-4 prosthesis does not require bone, HA, and/or AlloDerm to support the maxillary lip. Instead the flange of the overdenture may be designed similarly to a traditional denture to support the lip (Figure 34-19).

Division A Premaxilla

The maxillary anterior root form surgery with Division A bone is similar to that described in the maxillary anterior single-tooth implant (see Chapter 33). For a successful placement of standard-diameter endosteal

WWW.HIGHDENT.IR

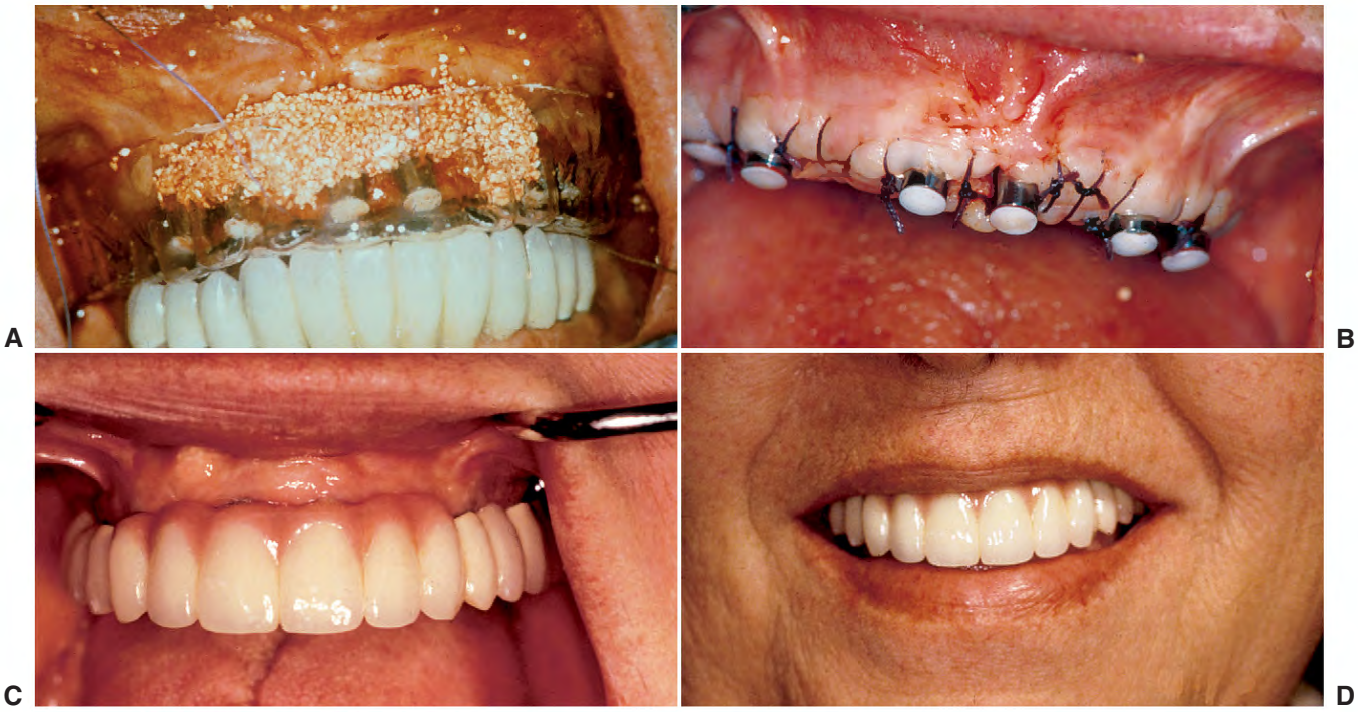


Figure 34-16 **A,** A soft tissue template evaluates the amount of dense hydroxyapatite (HA) required on the facial bone above the implants to support the maxillary lip similar to the removable restoration. **B,** The soft tissue is approximated over the HA graft. **C** and **D,** An FP-3 prosthesis supports the lip below the facial soft tissue from the HA graft and the prosthesis.

implants in the Division A anterior maxillary region, the recipient bone bed should be greater than 6 mm wide, greater than 12 mm high, and have a crown/residual bone height ratio less than 1.

When ideal anterior bone is present and the treatment plan is for an FP-1 prosthesis, a surgical template should identify the incisal edges and the mesiodistal tooth position of the final prosthesis. This template should be designed to rest on adjacent teeth (when present) or on the reflected ridge with a computed tomography (CT) bone model or index of the lower teeth to aid in correct positioning for the edentulous maxilla (Figure 34-20, *A* to *G*). The soft tissue crestal incision may be made on the lingual aspect of the edentulous ridge, providing additional keratinized tissue to the facial aspect (Figure 34-20, *H*). A vertical-release incision most often is made distal to the last tooth in each quadrant and includes the interdental papilla (Figure 34-20, *I*). When the mesial papilla is ideal, a "papilla-saving" incision may be medial to the papilla, similar to a single-tooth site.

When the premaxilla requires augmentation to give additional support to the maxillary lip, the facial flap is reflected to the inferior piriform rim. An adequate maxillary anterior ridge does not require reflection of the facial flap beyond the mucogingival junction. Once the soft tissues are reflected, the canine sites are first addressed. A surgical template helps guide their position and angulation. A large intraoral photographic mirror may be used to evaluate the parallelism of the two canine implants. It is not unusual for the facial trajectory of bone to complicate parallel preparation of the canine osteotomies. When multiple anterior teeth are missing, one to two implants between the canines may be inserted. The central incisor implant site is prepared next and should be placed slightly distal to the center of the final crown's mesiodistal length (Figure 34-20, *J* to *M*).

The incisive foramen often expands laterally within the palatal bone, and the central incisor implant osteotomy may inadvertently encroach on this structure and, as a result, form fibrous tissue at the interface in the mesiopalatal region. A more distal implant placement in the central incisor region prevents this complication. Because most restorations in an edentulous premaxilla are FP-2 or FP-3, the most favorable sites for bone width are selected, even when they are in the interproximal region of central and lateral sites.

When the labial soft tissue is not reflected to the inferior piriform rim of the nose, the facial tissues are palpated with the fingers of the hand not holding the hand piece during the actual bone preparation to feel whether the rotation of the drill can be detected underneath the tissue. After preparation, the anterior implant osteotomy is evaluated with a probe, especially on the facial aspect before implant placement, because labial undercuts often are present above the level of the facial flap reflection and may result in facial fenestrations during the osteotomy or implant placement process. When in doubt of a bone fenestration, the

WWW.HIGHDENT.IR

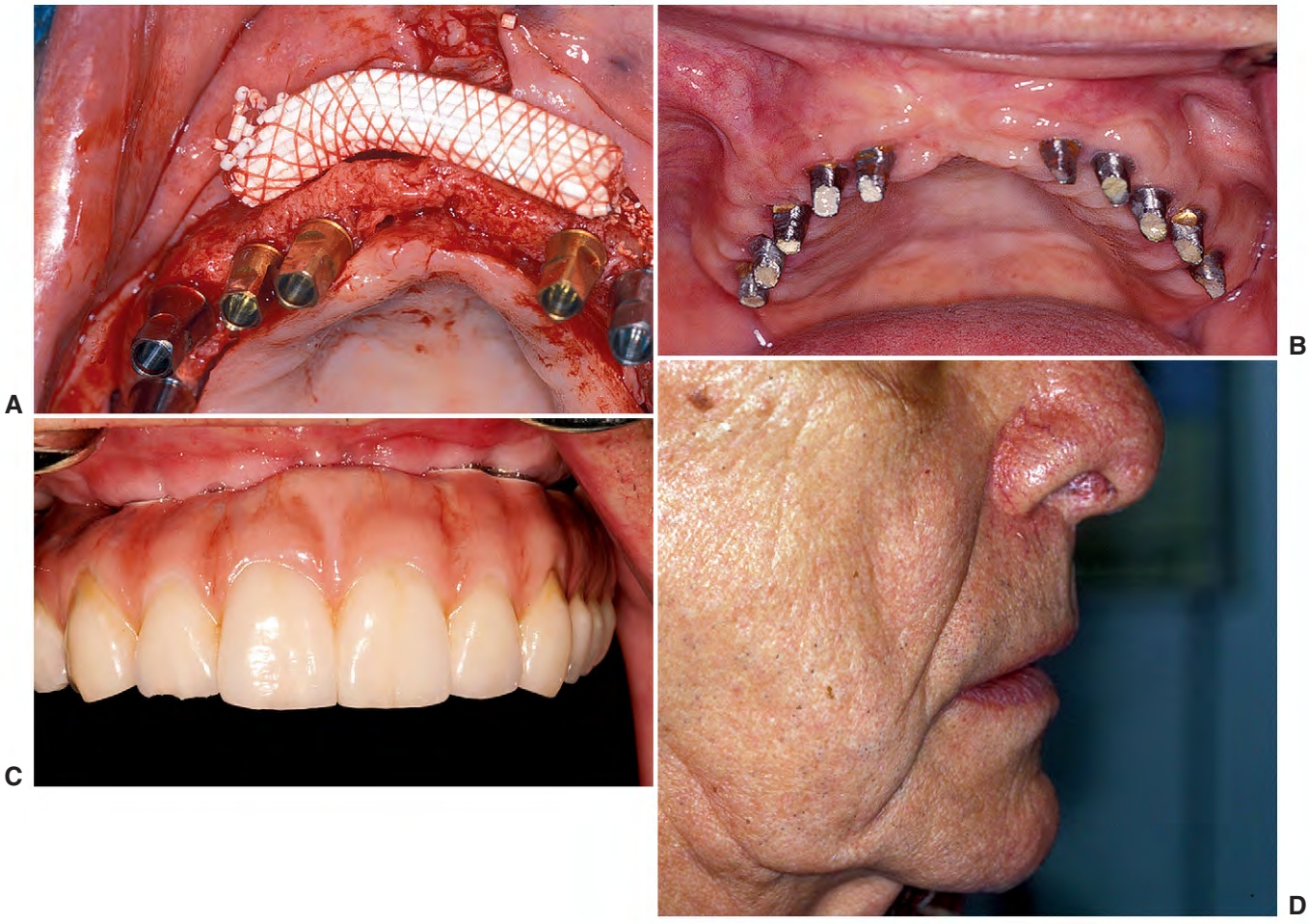


Figure 34-17 **A,** An alloplastic dense hydroxyapatite (HA) PermaRidge graft is placed on the facial aspect of the bone, above the implants, to support the maxillary lip. **B,** The soft tissue contour is improved over the dense HA graft. **C,** The alloplastic graft and FP-3 prosthesis support the maxillary lip. **D,** The final prosthesis and maxilla graft provide adequate lip support for the patient shown in **A** to **C**.

labial tissue should be completely reflected and the area evaluated.

A 2-mm end-cutting drill prepares the implant sites with the angulation determined by the incisal edge of the prosthesis. A direction indicator is then positioned into the sites. The stop on the direction indicator is often 4 mm in diameter, which may be used to evaluate the final implant position and bone volume around the final implant site. If the labial hard tissue on the facial of the implants is not greater than 1.5 mm, then bone loss after remodeling or early loading may cause a facial fenestration over the implant. When the bone is inadequate in width, bone expansion may be used to increase the facial plate of bone, which is presented later in the chapter (Figure 34-20, *N* and *O*).

When abundant bone exists 3 mm below the future implant crown and abundant soft tissue is present, an FP-1 prosthesis may be planned. The implant body should be placed approximately 3 mm below the facial free gingival margin of the future implant crown, which is level with the crestal bone (Figure 34-20, *P* to *R*). In an FP-2 or FP-3 prosthesis, the level of the soft tissue and bone relationship is not relevant. When placing implants in Division A bone to support an implant overdenture (IOD), the crown height space is evaluated to ensure a minimum of 12 mm for prosthetic components. When not present, an osteoplasty before implant insertion is indicated.

The anterior implant site should ideally have greater than 6 mm of available bone width for Division A root forms. When a less than ideal bone width does not provide a minimum of 1.0 to 1.5 mm of facial bone, a Division B root form (3.5 to 3.75 mm), a membrane ridge augmentation with a layered approach at the time of implant insertion, or a bone-spreading technique is indicated.

The soft tissue drape of an FP-1 restoration on multiple implants is easiest to obtain when the soft tissue is at the desired height of the interproximal papillae. Elevating and increasing the tissue thickness at the insertion surgery may accomplish this. Permucosal extensions 3 mm in height and an acellular dermis matrix graft (AlloDerm) may be used to elevate the tissue with primary closure over these components (Figure 34-20, *S* to *Y*).

WWW.HIGHDENT.IR

Figure 34-18 **A,** An iliac crest bone graft can help support the maxillary lip of an edentulous maxilla. **B,** The maxillary FP-3, porcelain-fused-to-metal implant prosthesis opposing a hybrid denture tooth, acrylic-to-metal framework in the mandible. **C,** A panoramic radiograph of the maxillary implants inserted into the iliac crest graft and supporting the FP-3 prosthesis. **D,** The iliac crest bone graft and the FP-3 prosthesis support the maxillary lip. A philtrum is present, and the maxillary lip is in front of the mandibular lip.

If a fenestration occurs on the apical one half of the implant during the osteotomy or implant insertion, or if a thin ridge presents inadequate bone width, then a layered bone graft of autogenous bone over the implant site, covered with a mixture of demineralized freeze-dried bone (DFDB) cortical fibers, resorbable HA, and platelet-rich plasma (PRP) and covered with a resorbable collagen or acellular dermal tissue membrane may prevent further bone loss and resultant implant thread exposure. The labial flange of a soft tissue–borne transitional prosthesis should be completely removed to decrease the risk of incision line opening when implants are placed into an edentulous premaxilla (Figure 34-20, *Z* and *AA*). A subtraction soft tissue technique recontours the tissue and creates the emergence profile of the crowns and the interdental papilla at stage II uncovery.

BONE SPREADING

The faciopalatal bone is often only 4 to 6 mm wide at the crest and/or presents a facial "hourglass" concavity. Bone spreading may be used to expand the available bone width before implant placement (Figure 34-21). Tatum[18-20] developed bone spreading in the early 1970s. He performed this technique using calibrated osteotomies ("graduated wedges"), matching the dimension of endosteal implants specifically designed for this procedure (Figure 34-22). Tatum inserted more than 5000 maxillary anterior implants with this procedure before 1985.

Over the years the bone expansion technique has been primarily used in regions of Division B bone to increase the bony width, rather than using onlay grafts. However, the easiest edentulous ridges to expand are Division A bone volume and D3 or D4 bone density. The narrower the bone, the greater the risk of fracture of the facial plate. The softer the trabecular bone quality, the lower the elastic modulus and the greater the viscoelastic nature of the ridge. Therefore the less dense the bone, the easier and more predictable the bone expansion. Maxillary edentulous Division A bone is the most ideal to expand and provides the best situation to develop a learning curve for the bone-spreading technique, which is also advantageous in Division B ridges (although more technique sensitive).

WWW.HIGHDENT.IR

Figure 34-19 **A,** An intraoral view of a RP-4 overdenture bar. **B,** A maxillary RP-4 overdenture from the previous intraoral view (in place). A labial flange is designed to support the maxillary lip. **C,** The denture teeth and flange of the overdenture support the maxillary lip.

Division A Bone Expansion

Bone expansion does not affect the facial and palatal plates equally. The thicker palatal bone is more difficult to manipulate; therefore the expansion process is primarily in the direction of the thinner facial plate. This is a benefit because the bone defect or deficit is also facial. Bone expansion provides a more normal facial contour to the region. The implant osteotomy is begun with a No. 6 bur or a 2.0-mm starter drill and positioned 0.5 mm more palatal than the usual midcrest position, to place a slightly thicker amount of bone on the facial aspect and decrease the risk of labial plate fracture during the expansion process (Figure 34-23, *A* and *B*). The No. 6 round bur can be used to prepare the osteotomy several millimeters in depth and to remove a portion of the thicker palatal plate on the lingual of the osteotomy site.

Bone expansion requires tapping on an osteotome with a surgical mallet (almost like tapping a nail into balsa to white pine wood). As a result, patient sedation is recommended because the tapping may elicit greater patient concern and response than the use of drills to prepare the osteotomy. Advantages exist to using an osteotome and mallet to prepare an implant site, even in Division A bone. The less dense bone may be compressed, which results in a denser interface after healing.[21] Less risk of causing a facial fenestration also exists compared with drilling, because the bone is being pushed facially, not removed with rotating drills.

A 2-mm end-cutting pilot drill is then used at 2500 rpm, with copious amounts of cooled sterile saline. The drill penetrates along the initial pilot hole prepared with the No. 6 bur at the angulation corresponding to the final ideal angulation, directly under the incisal edge and parallel to the cervical third contour of the adjacent natural teeth. The bone is prepared to the opposing landmark, which is often 2 to 4 mm deeper than the final implant length. The presence of a thick palatal plate tends to push the drill toward the facial plate. This occurs because the side of the end-cutting drill is in contact with dense palatal bone. Rather than removing equal amounts of bone from each side of the osteotomy, the drill, which is not designed to be side cutting, is pushed facially, and the osteotomy is only enlarged from that aspect. In such situations, the surgeon should stop and use a side-cutting drill (e.g., Lindemann drill) to enlarge the palatal side of the osteotomy before using the 2.0-mm drill to completion.

Once the 2-mm drill has prepared the bone 2 to 4 mm beyond the final implant depth, specifically designed osteotomes are used to further widen the osteotomy (Figure 34-23, *C*). The implant is round in cross section and will be inserted at the same time as the bone expansion; therefore the osteotomes are also round in cross section. The ends of the osteotomes are tapered for the last 2 to 3 mm to create a gradual bone expansion. The osteotomes are provided in increasing diameters, preferably in increments of 0.5 mm. Larger-diameter increments increase the risk of labial plate bone fracture.

The first osteotome used in the osteotomy is 2 mm in diameter. It is gently tapped with a surgical mallet into the bone 4 mm beyond the final implant length (Figure 34-23, *D*). This process should be easy because the drill already prepared an osteotomy of the same dimension. The angulation is evaluated—at least 2 mm of bone should be on the facial aspect of the instrument. If not, then the osteotome is removed, and a side-cutting drill is used to remove some bone on the palatal aspect of the osteotomy. To remove a bone-spreading osteotome, it should not be luxated as a tooth; instead it is rotated in the socket and unscrewed with a gentle axial tensile force.

A 2.5-mm round, tapered osteotome is then inserted into the osteotomy (Figure 34-23, *E*). A surgical mallet

WWW.HIGHDENT.IR

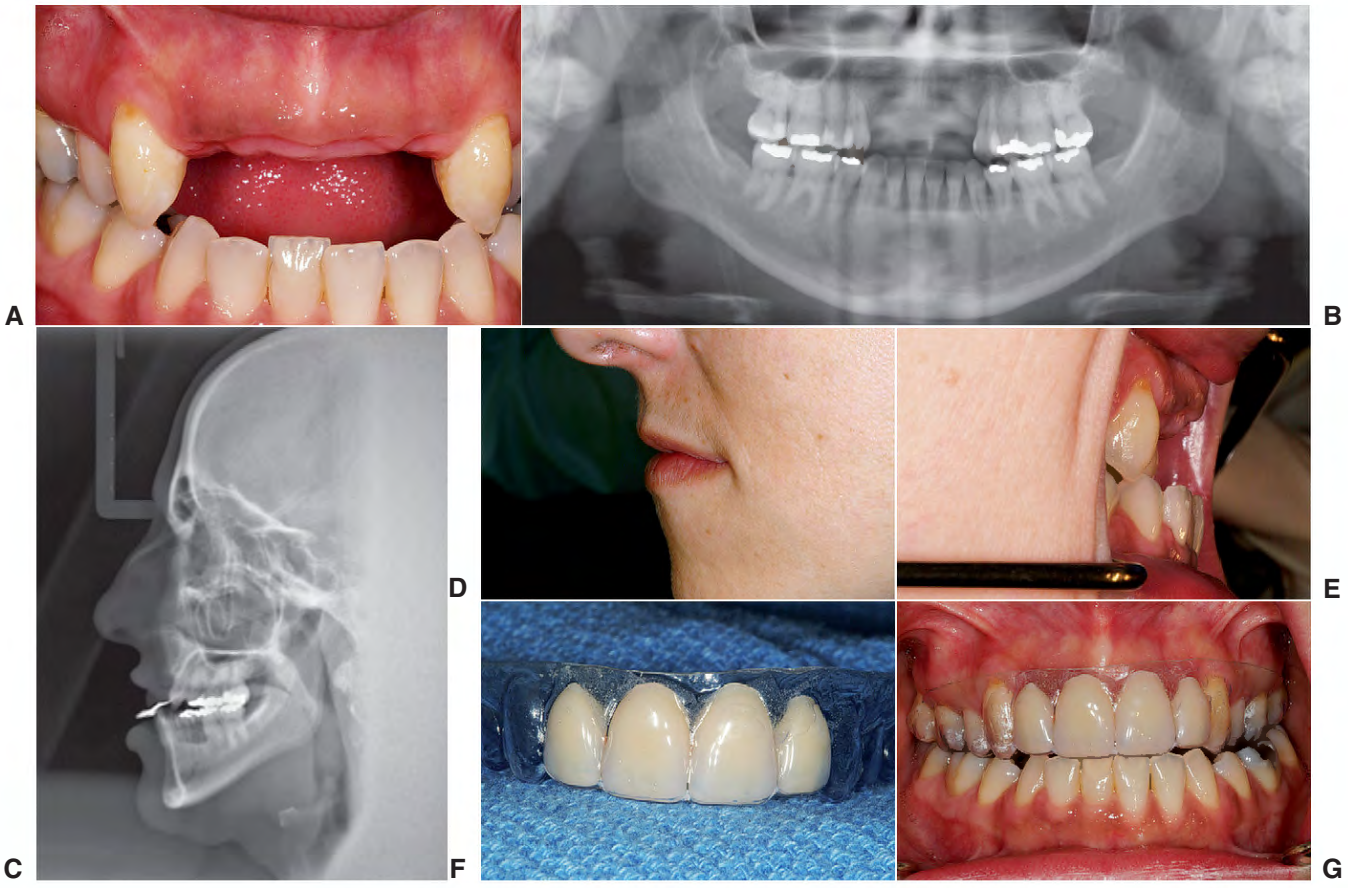


Figure 34-20 **A,** An intraoral view of a patient missing four anterior incisors. **B,** Panoramic and **(C)** cephalometric radiographs of the patient in **A**. **D,** The maxillary lip has support from the canines but is deficient in the midline. **E,** The maxillary arch is palatal to the mandibular incisor edge position and is deficient in the midline. **F,** A surgical template indicates the maxillary incisor edges and the desired tooth size. **G,** The surgical template is designed to fit on the adjacent teeth so that it is rigid when in place and allows more precise implant site preparation. *Continued*

then gently taps the osteotome so that it proceeds into the bone for about 1 mm for every tap (Figure 34-23, *F*). At each 5-mm depth, a pause of 15 to 30 seconds is allowed (the harder the tap required to proceed 1 mm, the longer the wait). Once this instrument proceeds 3 mm beyond the final length, or 0.5 to 1.0 mm less in final depth than the previous osteotome, the surgeon should pause 1 to 2 minutes before removing the instrument (Figure 34-23, *G*).

The periodic pauses are an intrinsic part of the procedure that takes advantage of the viscoelastic nature of bone, to expand it without fracture. During the pauses, the intraosseus and extraosseous fluids between the compressed trabeculae escape from the intratrabecular spaces, thus decreasing the risk of fracture of the facial plate. In addition, the biomechanical creep change in bone over time permits the bone to expand without fracture.

A 3-mm round, tapered osteotome is then tapped into position (Figure 34-23, *H*). This process is more difficult than the previous two steps. It is beneficial to have the assistant hold and direct the osteotome once it is engaged for the first 6 to 8 mm. The fingertips of the nontapping hand of the surgeon hold and brace the facial and palatal plates over the edentulous site. If pressure is felt in the fingertips while the tapping commences, then the process slows down and each step is extended in time.

The intensity of the tapping is sufficient when the osteotome proceeds 0.5 mm to 1 mm deep for each tap. A pause every 5 mm is 30 to 60 seconds in duration (Figure 34-23, *I*). The final osteotome penetrates 2 mm shorter than the original 2.0-mm osteotome. Once completely inserted, the osteotome is left in position for 2 to 4 minutes, depending on the difficulty to proceed and the pressure felt on the labial plate. A surgical forceps may be required to rotate and pull the osteotome out from the bony site.

On occasion, the osteotome handle may tend to drift facially such that the angulation of the osteotomy flares

WWW.HIGHDENT.IR

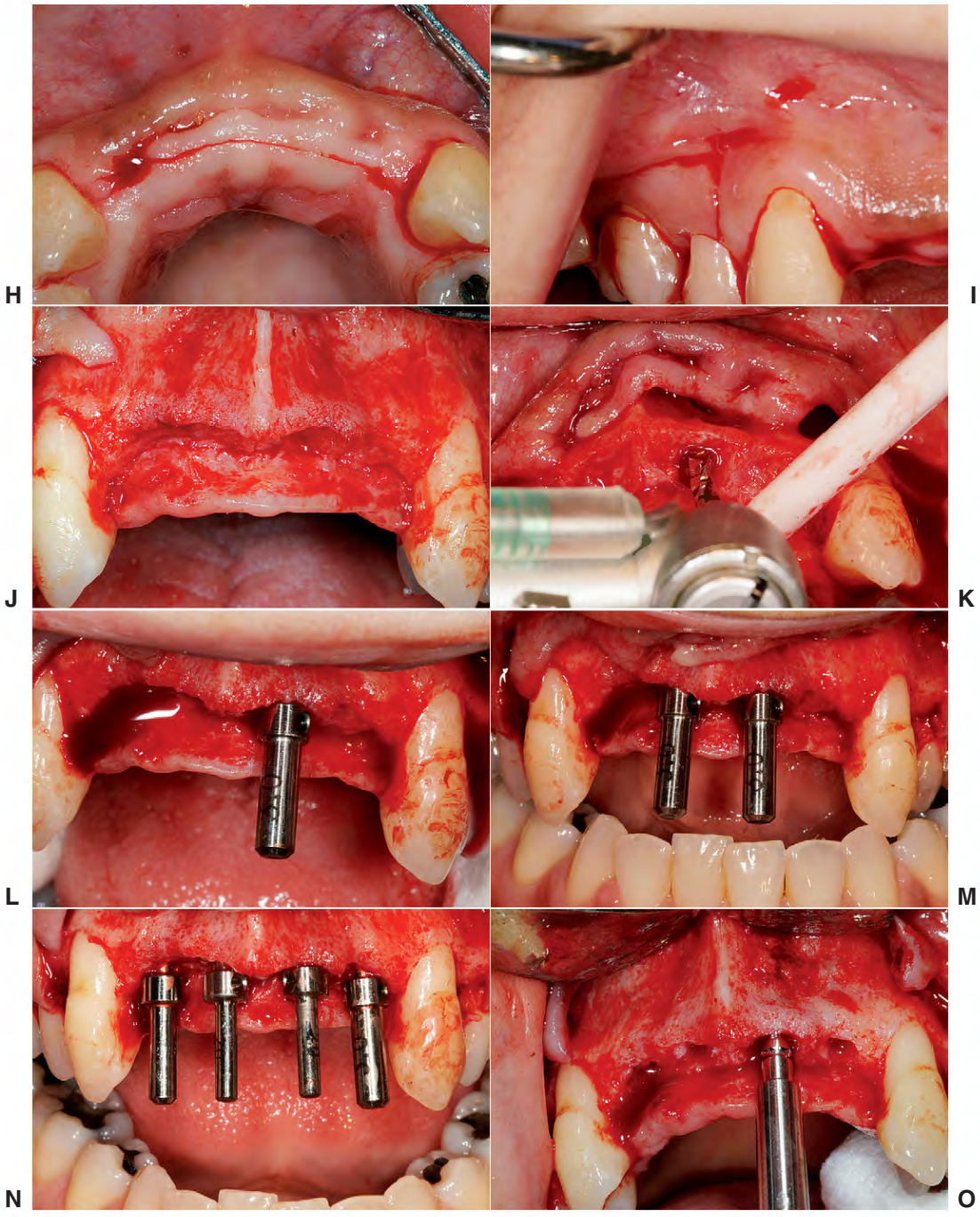


Figure 34-20, cont'd **H,** The soft tissue crestal incision is made on the palatal aspect of the ridge to increase the amount of keratinized tissue on the facial of the incision line. **I,** A vertical-release incision is made distal to the interdental papilla of the canine. In this patient the canine root is also treated for root coverage. **J,** The facial tissues are reflected, and the width of the residual ridge is evaluated. **K,** The central incisor site is prepared with a 2-mm starter drill and is 0.5 mm more distal than the midtooth position. **L,** A direction indicator is placed into the site and evaluated for angulation in site position. **M,** The other central incisor site is prepared using the surgical template and the first direction indicator as a guide. **N,** The direction indicator stops are 4 mm in diameter; therefore the implant interimplant distance amount of facial bone to the implant and angulation may be evaluated. **O,** A bone spreader is used in the central incisor site to increase the amount of bone facial to the implant.

Continued

out beyond the incisal edge position. This is caused by the presence of the thicker palatal bone resisting expansion on the palatal side of the osteotome and facially pushing the instrument. When this occurs, the osteotome is removed, and a side-cutting drill is used to remove some of the palatal bone within the osteotomy (Figure 34-23, *J*). The osteotome is reinserted and proceeds in the previous fashion.

At the 3.0-mm-diameter point, the clinical decision must be made regarding whether a 3.5- or 4.0-mm-diameter implant will be selected. If little bone is present on the facial aspect of the 3.0-mm osteotome

WWW.HIGHDENT.IR

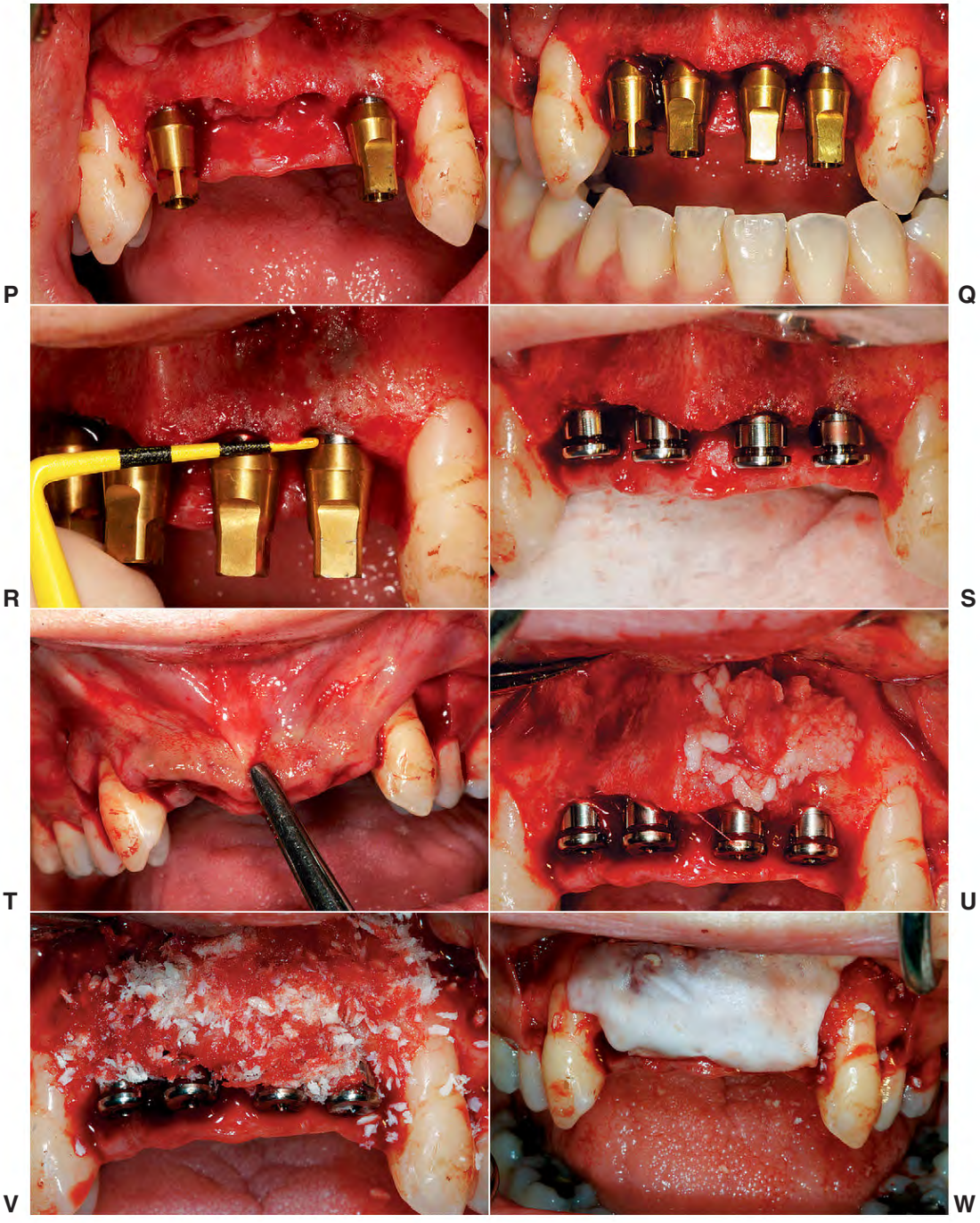


Figure 34-20, cont'd **P,** The two lateral incisor implants are inserted. The crestal bone and implants are 3 mm beyond the free gingival margin of the adjacent teeth and future implant crowns. **Q,** The maxillary anterior implants are placed 2 mm below the cement-enamel junction (CEJ) of the adjacent canines, or 3 mm below the free gingival margin of the future implant crowns. **R,** The implants are 3 mm or more apart to allow interimplant space to develop the soft tissue drape. **S,** Permucosal extensions are inserted into the implant bodies to act as a tissue tent screw. **T,** The labial soft tissue of lip is advanced so it may obtain primary closure over the implants. **U,** Autologous bone is primarily placed over the implant that had bone spreading to increase the width. **V,** A layer of demineralized freeze-dried bone (DFDB) (30%) and mineralized freeze dried bone (70%) is placed over the facial bone. **W,** An acellular tissue graft (AlloDerm) is placed over the large bone graft to act as a barrier membrane and increase tissue thickness. *Continued*

and/or the bone was difficult to prepare, then a 3.5-mm implant is selected. The final drill for the 3.5-mm-diameter implant (usually 3.0 mm in diameter) is used to finalize the osteotomy after the last osteotome. Many maxillary ridges, however, can be further spread to receive a larger 4.0-mm-diameter implant.

When the larger diameter implant is selected, a 3.5-mm round, tapered osteotome is then inserted into the osteotomy and proceeds in a similar fashion (Figure 34-23, *K*). The final depth of the 3.5-mm osteotome is 1 mm shorter than for the 3.0-mm-diameter osteotome, similarly to a "step back" approach in canal preparation

WWW.HIGHDENT.IR

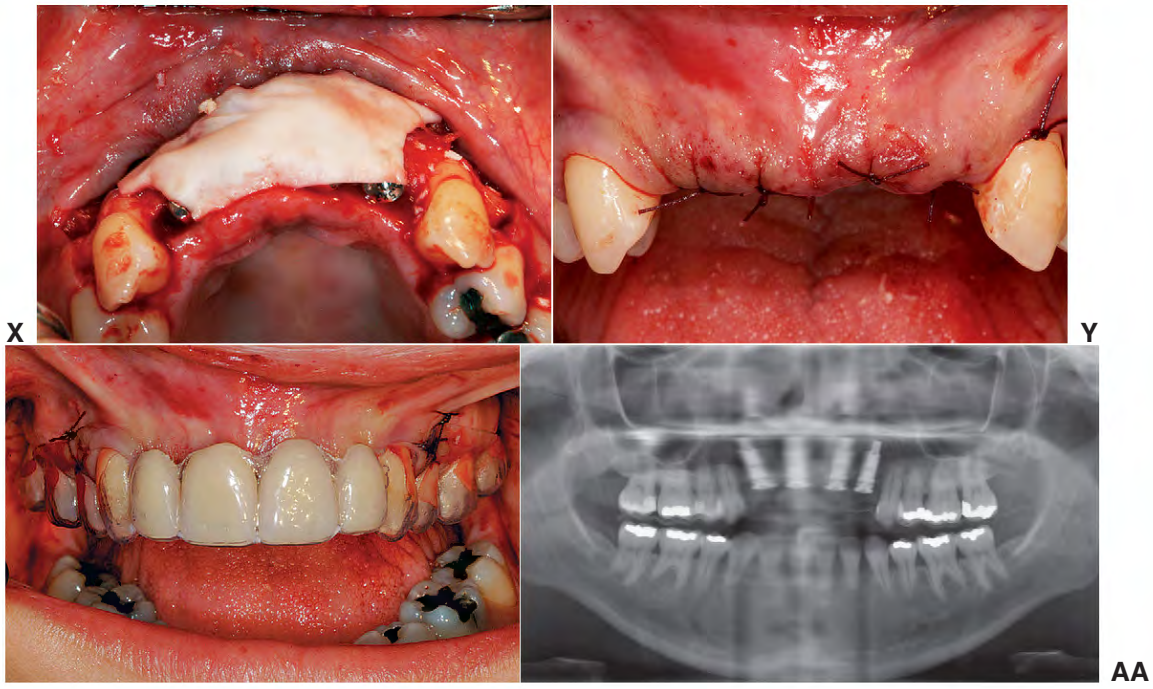


Figure 34-20, cont'd **X,** The acellular tissue graft (AlloDerm) is draped over the permucosal extensions to increase the tissue thickness. **Y,** The soft tissue flap is advanced and covers the layered bone graft and permucosal extensions for primary closure. The canine roots surfaces are also covered with soft tissue. **Z,** A tooth-borne Essix transitional prosthesis is delivered, which places no pressure on the soft tissue. **AA,** Postoperative panoramic radiograph.

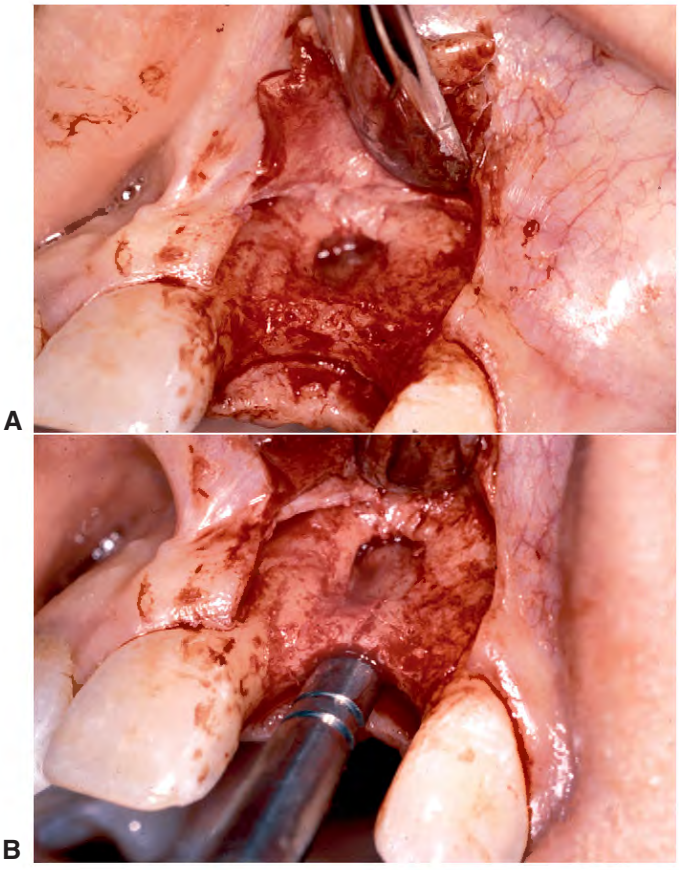


Figure 34-21 **A,** A bone defect is found at the apical aspect of the implant site of the left lateral incisor. **B,** A bone-spreader osteotome and bone expansion technique places bone on the facial aspect of the apical defect.

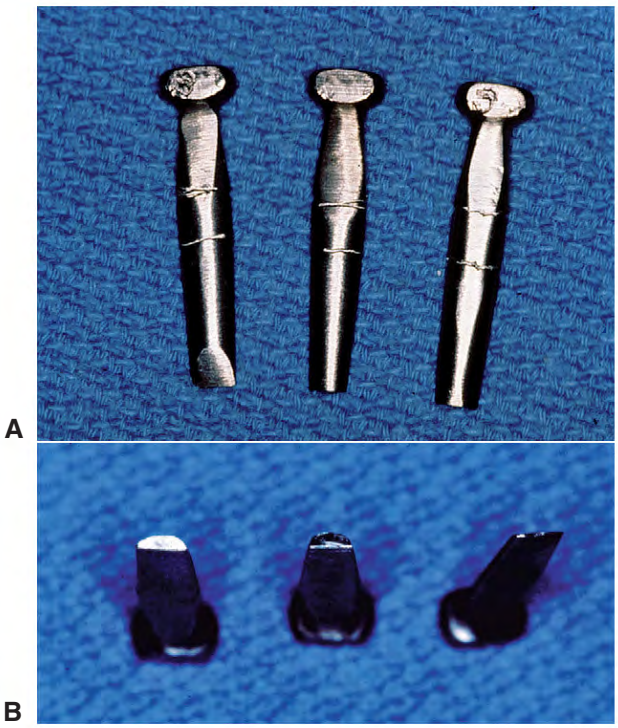


Figure 34-22 The bone spreaders of Tatum were wedge-shaped devices that gradually increased in width *(from left to right).* Their shape was similar to the final endosteal implant shape.

WWW.HIGHDENT.IR

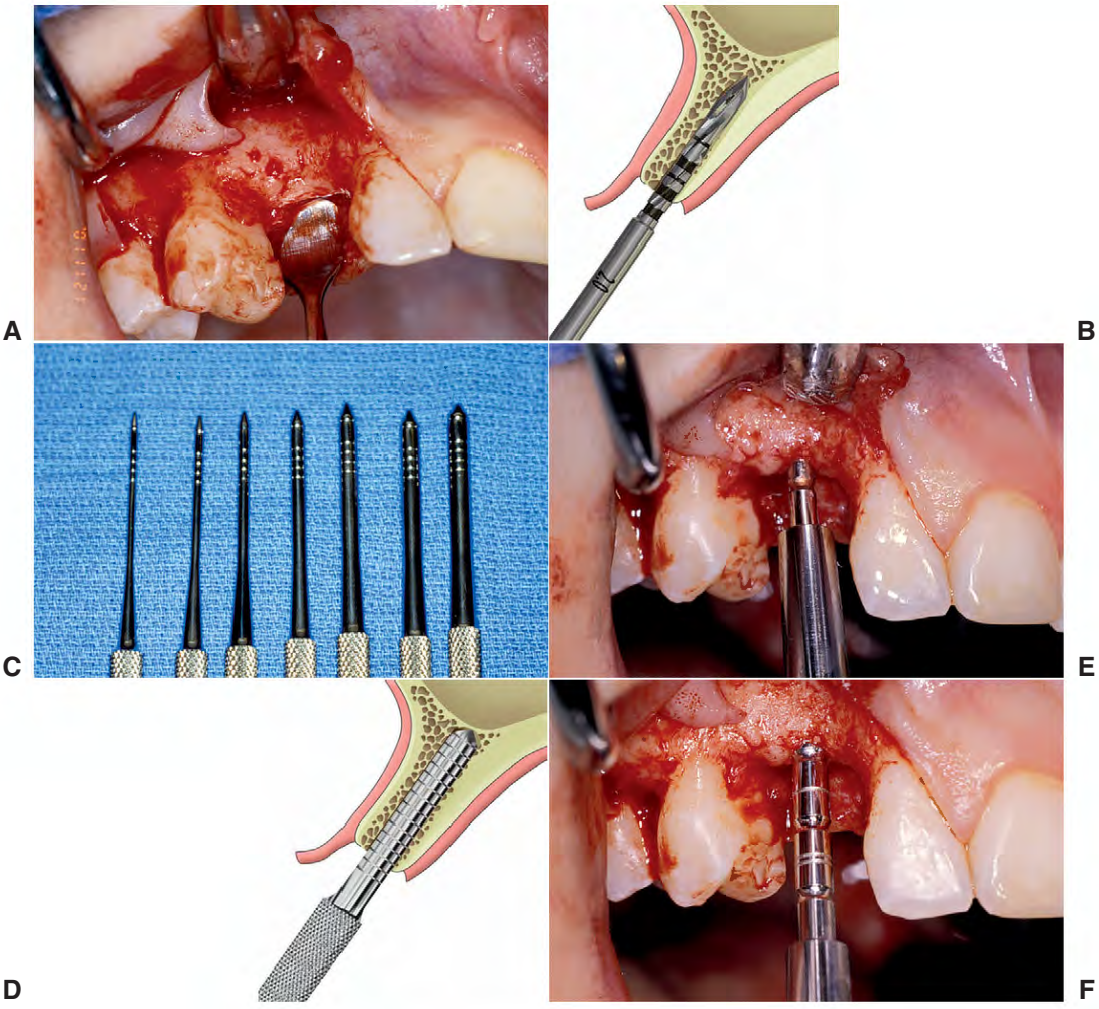


Figure 34-23 **A,** A Division A site that is D3 bone is a good site to develop the technique of bone spreading. **B,** A starter drill begins the osteotomy 0.5 mm toward the palate to increase the facial bone volume. **C,** The osteotomes for bone spreading and implant insertion are round, proceed with 0.5-mm-diameter increments, and have a tapered apical 2- to 3-mm end. **D,** A 2.0-mm bone-spreading osteotome is tapped into position in 3- to 5-mm increments. This process proceeds without difficulty because the 2.0-mm twist drill has already prepared the site. **E,** A 2.5-mm osteotome is tapped into position in 3- to 5-mm increments. Every 3 to 5 mm, a waiting period of 30 to 60 seconds permits bone to expand without fracture. **F,** A 2.5-mm bone-spreading osteotome continues to be tapped into the site in 3- to 4-mm increments and 30- to 60-minute waiting periods.

Continued

for endodontics (Figure 34-23, *L*). The reader should remember that the depth of the initial osteotome is 3 mm deeper than the desired implant length. Each successive, larger osteotome is inserted 0.5 mm shorter than the preceding instrument. This expands the base of the osteotomy in a V shape, rather than a U shape, and it is less likely to fracture the labial cortical plate. When conditions warrant, additional pauses and extended time intervals for each 5-mm-deep segment should be incorporated.

The final diameter drill of the manufacturer can now be used for the 4.0-mm-diameter threaded implant. This diameter often approaches 3.2 to 3.4 mm (depending on implant design and bone density). Although a 3.5-mm osteotome was used last, the bone will relapse back to a smaller diameter, shortly after each bone-spreading instrument is used. By drilling the final osteotomy size and extracting bone, rather than just using the expansion technique that only compresses the bone, the final osteotomy will be of the proper width and depth (Figure 34-23, *M*). Because the bone most often is wider at its base, compared with the crestal region, even when the gradually increasing tapered osteotomes do not expand the apical region, the end-cutting drill can prepare the site without consequence.

The labial tissue is palpated during the actual bone preparation to feel whether the drill can be detected. The final implant osteotomy is also inspected with a probe, especially on the facial aspect before implant placement. Labial undercuts or fractures may cause fenestrations. Once the labial tissues have been reflected, the blood supply to the labial porous compact bone is diminished. The mesiolingual aspect of a central incisor implant is also checked to ensure the lack of communication

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

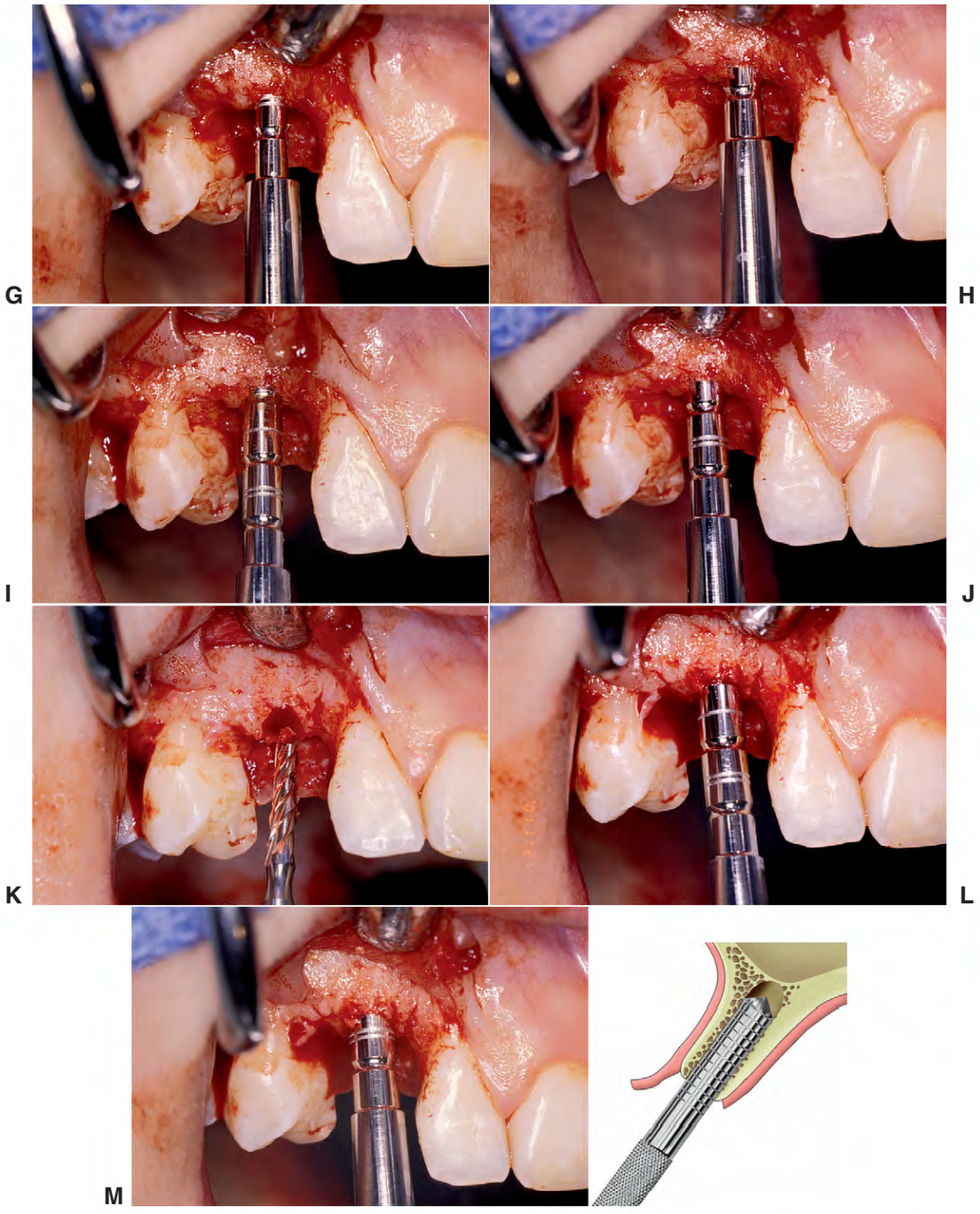


Figure 34-23, cont'd **G,** The 2.5-mm bone-spreading osteotome is tapped into the implant site 3 mm beyond the length of the implant. **H,** A 3.0-mm bone-spreading osteotome is placed into the site and tapped in 3- to 5-mm increments. **I,** The 3.0-mm bone-spreading osteotome requires a firmer tap, shorter increments, and longer waiting periods. **J,** A side-cutting Lindemann drill used to remove some of the palatal bone of the osteotome is pushed to the facial. **K,** A 3.5-mm osteotome is used to prepare the final osteotomy diameter for a 4.0-mm implant. **L,** A 3.5-mm bone-spreading osteotome is tapped to the final implant length. **M,** Because the bone has a modulus of elasticity and is not expanded beyond permanent deformation, the size of the expanded osteotomy rebounds to a smaller diameter after each osteotome. Therefore after the final osteotome *(left)* a 3.4-mm drill is used to prepare the final diameter for the implant.

Continued

with the incisive canal. When in doubt, the labial tissue should be reflected and the area inspected. If a perforation is present, then an autologous graft and/or barrier membrane is used to augment the site on top of the cortical plate. When the facial plate is not violated but is less than 1.5 mm thick, a resorbable calcium phosphate and a resorbable membrane are placed over the grafted region to prevent further bone loss from remodeling and causing subsequent implant thread exposure.

WWW.HIGHDENT.IR

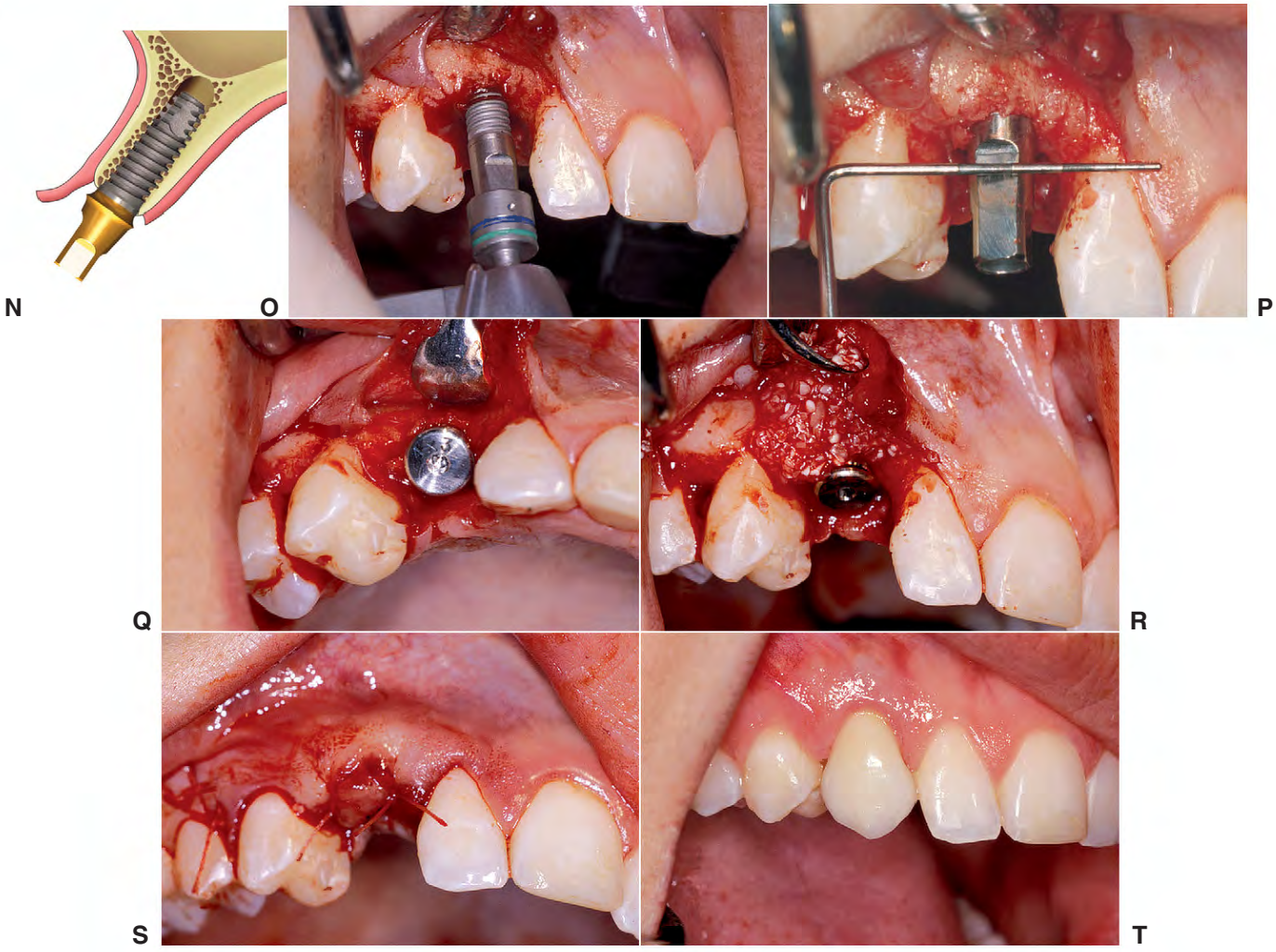


Figure 34-23, cont'd **N, O,** The implant is threaded into position with a hand piece at 30 rpm after a bone tap prepared the threads within the bone. **P,** The implant is seated 3 mm below the facial free gingival margin of the future implant crown. **Q,** A 3-mm-high permucosal extension (PME) is inserted into the implant. **R,** A layered bone graft of autograft, then mineralized and demineralized bone, is placed over the facial bone to decrease the risk of thread exposure after bone spreading. **S,** The soft tissue covers the PME and bone graft. This augments the facial bone and the soft tissue for the interproximal tissue. **T,** After a subtraction technique for stage II uncovery and the development of proper emergence profile with a temporary crown, the final crown is delivered.

A crestal bone drill is rarely indicated with the bone expansion technique. However, a bone tap is often used to prepare the dense palatal bone for a threaded implant. When a tap is not used before implant insertion, a threaded implant may be pushed through the facial softer bone, rather than threading the more dense palatal bone. The bone tap helps prevent the less efficient threads of the implant from being pushed facial and fenestrating the facial plate.

The final implant may be threaded into position using a slow-speed, high-torque hand piece (Figure 34-23, *N*). The implant is seated at the depth appropriate for the prosthesis type (3 mm below free gingival margin of the future implant crown for FP-1 restoration) (Figure 34-23, *O*). When the soft tissue in the interproximal area requires augmentation, a 3-mm PME may be placed into the implant (Figure 34-23, *O*). This device may act as a tenting screw to elevate the tissue. A layered bone graft with an autograft, then demineralized and mineralized bone, is placed on the facial bone over the implant, concentrating on the crestal region (Figure 34-23, *P*). The graft procedure decreases the risk of crestal bone loss after the bone expansion and also aids in the modeling process of bone to change bone volume. The soft tissue is approximated over the PME (Figure 34-23, *Q*). This elevates the interproximal tissue and is a benefit to the layered graft. The final implant uncovery may use a subtraction technique to carve the interdental papillae and develop the soft tissue drape. The final crown develops the emergence contour for acceptable esthetics (Figure 34-23, *R*).

Bone spreading may be performed in either arch; in general, it is most often used in the maxilla. It may be used in several sites or one site only. The soft tissues

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

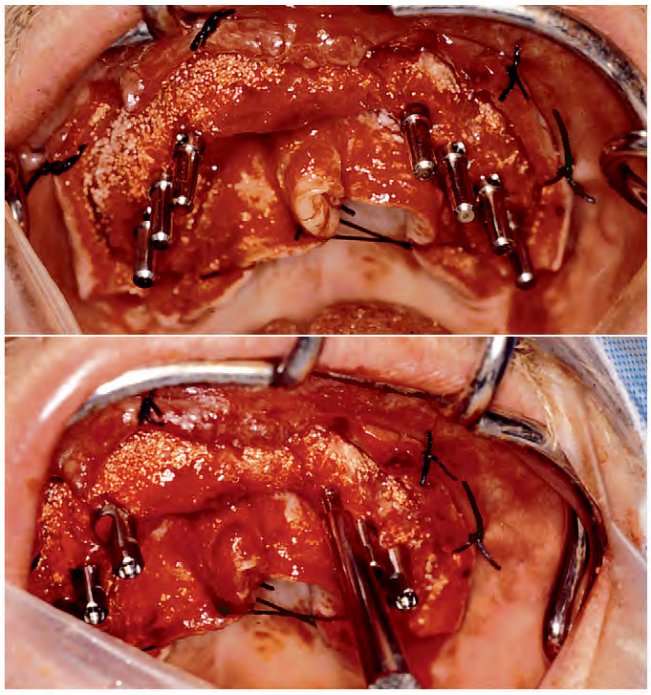


Figure 34-24 **A,** The maxillary soft tissue is reflected and a 2.0-mm-diameter drill is followed by 2.0-mm-diameter direction indicators. The stop on the direction indicator is 4.0 mm and can be used to evaluate the thickness of the bone around the site. The patient's left lateral site is too narrow for the implant. **B,** A bone-spreading osteotome is used in the lateral incisor site.

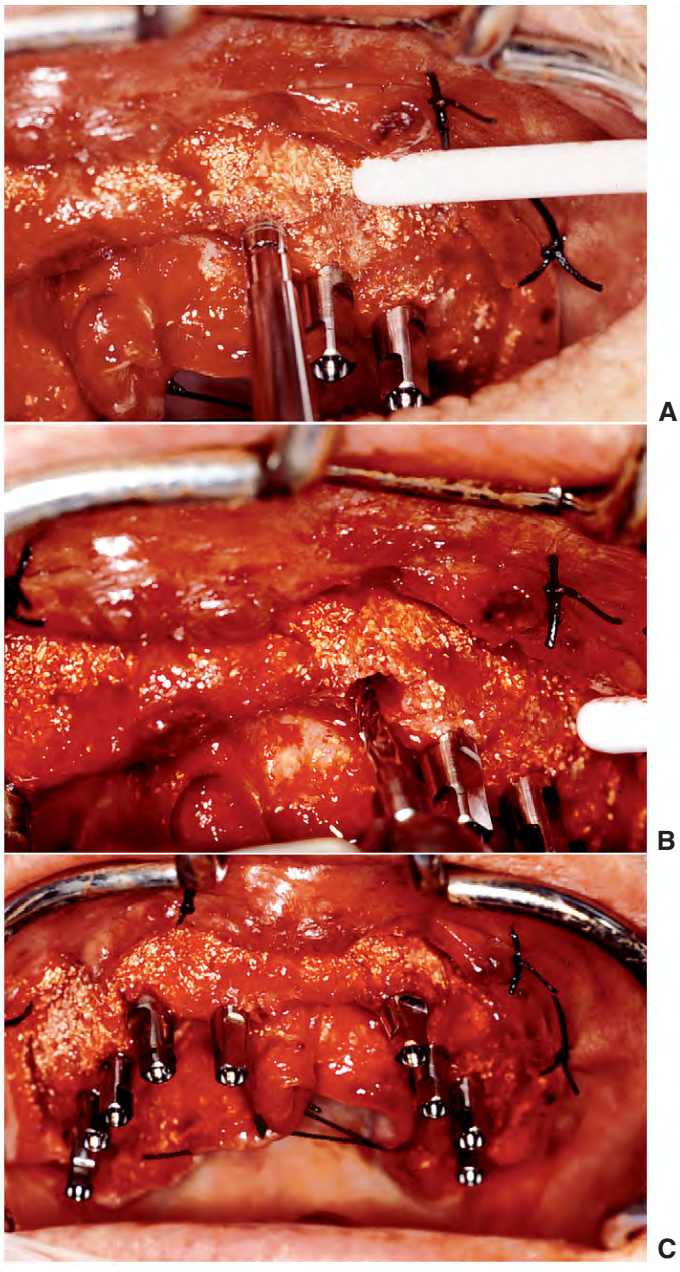


Figure 34-25 **A,** A 3.0-mm osteotome is used in the lateral site. **B,** A side-cutting Lindemann drill is used to prepare the palatal aspect of the osteotomy. **C,** A 3.5-mm implant is positioned into the lateral incisor implant site.

are reflected, and the initial implant osteotomy is made with a 2-mm-diameter starter drill. Direction indicators are then placed into the prospective implant site (Figure 34-24, *A*). The stops on the direction indicators are 4 mm in diameter, represent the diameter of the implant, and may be used to evaluate the facial and palatal bone thickness next to the 4-mm stop. When inadequate, bone spreading is a surgical option to allow implant insertion with more bone positioned to the facial (Figure 34-24, *B*). The site is expanded in a similar fashion to the previous discussion (Figure 34-25, *A*). A Lindemann side-cutting drill is often used on the palatal aspect of the bone-spreading osteotomy (Figure 34-25, *B*). The implants may then be inserted with additional bone on the facial to decrease risk of crestal bone loss after loading (Figure 34-25, *C*).

Division B Bone Spreading

When D2 bone is present in Division A or B ridges, labial bone fracture is more likely during bone expansion. To decrease this risk, bone spreading is accomplished after a narrow channel-shaped osteotomy is made with a 700 XXL surgical bur, similar to a plate form (blade implant) osteotomy (Figure 34-26). The narrow horizontal channel osteotomy is made the complete mesiodistal dimension of the implant site. In other words, if the mesiodistal implant site is 7 mm, then the horizontal groove is 7 mm wide. It is more predictable to spread a Division B width ridge that is 7 mm long, compared with a Division B ridge that is 4 mm long. When adjacent implant sites require bone spreading, the horizontal groove is made continuously from one implant site to the other. It is more predictable to spread a 14- to 21-mm mesiodistal ridge with a horizontal groove

WWW.HIGHDENT.IR

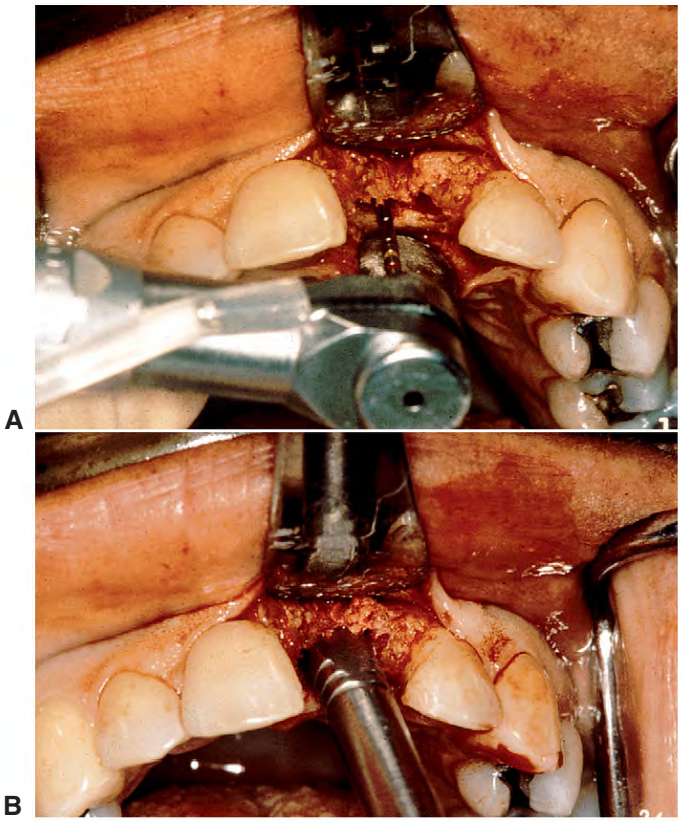


Figure 34-26 **A,** A Division B volume ridge is easier to expand when a mesiodistal horizontal slot is made with a 700 XXL surgical bur. **B,** A bone-spreading osteotome is tapped gently into the horizontal slot, and the facial bone is expanded.

than it is to spread two 4-mm adjacent sites within a 14- to 21-mm space. The horizontal osteotomy is also made several millimeters deeper than the length of the desired implant. It is more predictable to spread a deeper site than to spread one the same depth as the implants (see Figure 34-28).

When a Division B maxillary ridge is 4 to 5 mm, several options are available to the practitioner, including bone expansion and simultaneous implant placement, which is often performed when the bone is D3 or D4 in density. The Tatum approach for bone expansion limits periosteal reflection to the crest of the ridge to maintain the blood supply to the labial cortex. However, this surgical approach does not permit direct evaluation of the labial bone for potential splitting. In addition, the bone on the labial aspect of the implant is rarely 1.5 mm thick. As a result, crestal bone loss is more evident after healing and occlusal loading. Therefore after bone spreading and implant insertion is performed, a layered bone graft and resorbable membrane are most always placed on the labial aspect of the residual ridge. To this effect, a full reflection of the labial flap and soft tissue expansion for primary closure is almost always required.

Another option for Division B bone with D2 or D3 bone quality is an onlay graft, which requires a labial periosteal reflection. A tent screw is placed before the graft to maintain the space under the membrane. A layer of autogenous bone is placed on the labial surface of the recipient site, then a mix of resorbable HA with DFDB cortical fibers is placed on the facial of the ridge. A resorbable barrier membrane is then positioned over the top of the graft to assist in guided bone regeneration (GBR). A cortical block graft may also be used without a barrier membrane for augmentation in Division B sites but usually requires a mandibular donor site (see Chapter 39).

Complications

The most common complication of bone spreading, especially in Division B bone that is D2 quality, is splitting the facial plate during the procedure. Once this occurs, the surgeon must decide whether to continue, place the implant, and perform a barrier membrane layered bone graft, or abort the procedure and only place a bone graft. The implant may be inserted when the following factors are positive: (1) the implant is rigid at the proper depth, (2) the implant is in a favorable angulation, and (3) the facial plate is farther facial than the implant (it is fractured, but expanded). Under these conditions, the barrier membrane layered graft procedure is predictable to restore the facial bone and the implant is not compromised. If one of these three factors is negative, then it is more prudent to remove the implant, harvest additional autograft, and perform the bone graft without the implant in situ.

Another complication of spreading is the dehiscence of the labial plate after healing and bone remodeling around the implant (Figure 34-27, *A*). Because of its modulus of elasticity and because the expansion of the labial plate was not beyond the permanent deformation, the bone does not fracture, but it attempts to rebound to its original size during remodeling. As a result, during bone remodeling the bone does not heal in its expanded position; instead it returns to its initial narrow dimension, and the implant fenestrates the labial plate. This is not seen on a radiograph because it does not affect the interproximal bone. As a result, when bone expansion was performed at implant placement, a stage II uncovery with reflection of the facial soft tissue is advantageous. When a dehiscence is observed, a barrier membrane with layered graft approach are indicated to restore the facial plate (Figure 34-27, *B* and *C*).[22] Because the implant is integrated to the remaining bone, the implant may be progressively loaded after a 3- to 4-month period, rather than waiting 6 to 9 months, as with augmentation by barrier grafts alone.

The third complication of bone expansion is a poor implant position, usually more facial than ideal. The thicker palatal cortical plate pushes the osteotomes to the facial; if the surgeon is unaware, then the implant angulation slowly becomes too facial. Constant attention

WWW.HIGHDENT.IR

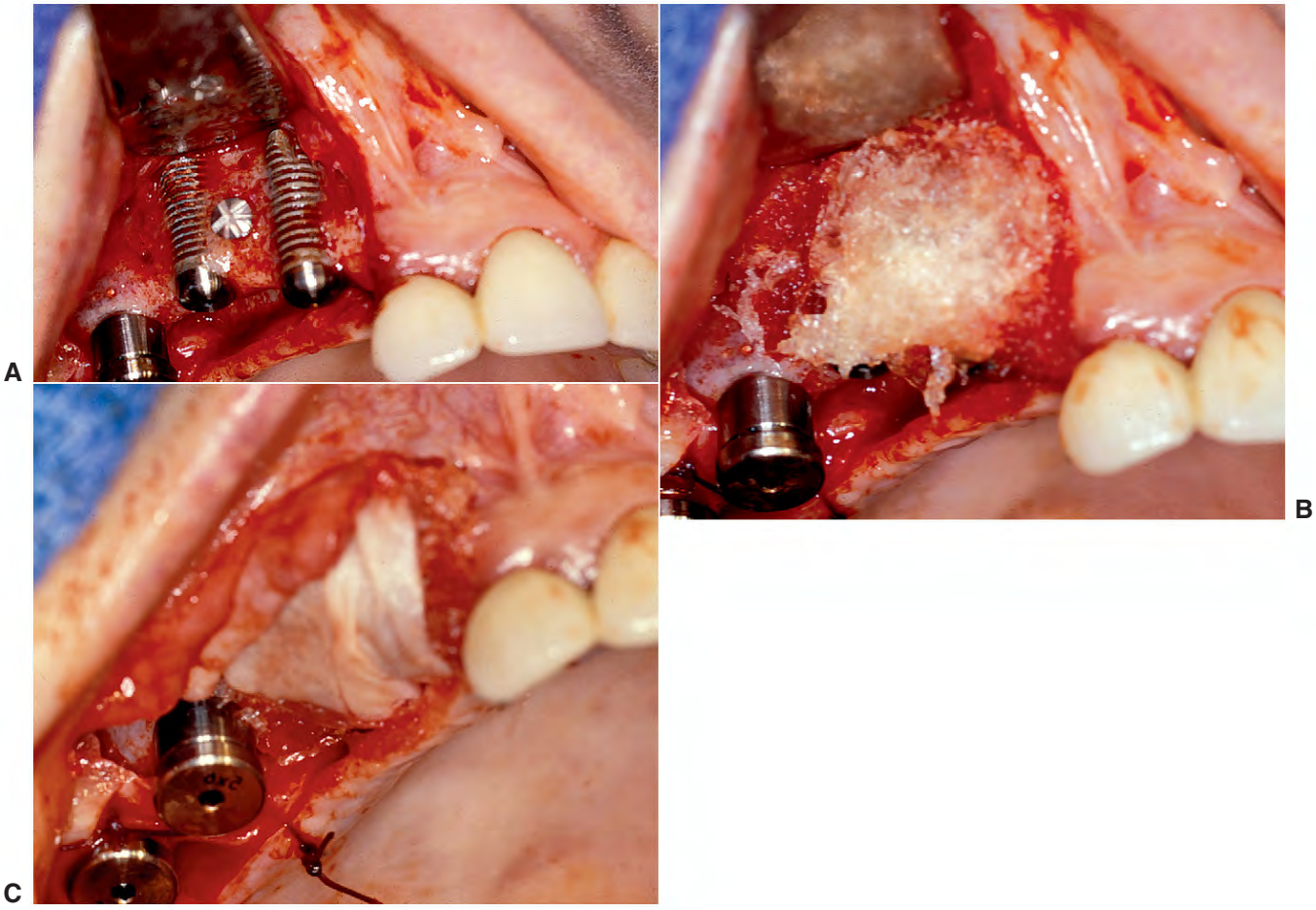


Figure 34-27 **A,** One of the more common complications of bone spreading is facial dehiscence of the implant after initial bone healing. When this occurs, a tent screw and layered bone graft is suggested to augment the width deficient ridge. **B,** Autologous bone, covered with mineralized and demineralized bone, is placed on the facial of the implants. **C,** A collagen barrier membrane or acellular dermal tissue graft (AlloDerm) is placed over the layered bone graft. The area is left to heal for a few additional months.

to angulation and modification of the palatal bone with side-cutting drills is necessary to prevent this problem. The final prosthesis should not be compromised for the sake of placing the implant during the surgery. Bone augmentation and reentry 6 months later often improves the implant position and, as a result, the final restoration (Figure 34-28).

SUBNASAL ELEVATION

The C–w bone volume ridge in the maxilla often does not increase in width after osteoplasty until inadequate remaining height is present. However, when anatomy permits, the osteoplasty of C–w bone may improve the crestal width to a Division C–h ridge. The C–h edentulous patient with a compromised anterior maxillary region has an available bone height of 7 to 11 mm, with a crown height/residual bone height ratio greater than 1.

A C–h premaxilla most often requires a removable overdenture, because the maxillary lip requires greater support than a fixed restoration can render. The partially edentulous patient with a C–h premaxilla who desires a FP-1 fixed restoration often requires an extraoral autogenous bone graft. Full-arch onlay grafts can be used to increase vertical height or bone, reduce the crown height space, and improve the arch's size and shape, which may be required for a fixed restoration.

The osteoplasty technique eliminates the need for autologous bone grafts and the associated healing time, discomfort, and cost of the procedure. The ideal dimensions required in a maxillary anterior C–h bone include a ridge width of greater than 6 mm, allowing at least 1 mm of bone on the lateral aspects of the implant. When the residual crest in the anterior maxilla is adequate in width but only 7 to 11 mm in height, a Division C–h root form and subnasal elevation and/or lateral piriform rim modification may be considered to provide adequate height for implant placement.[10,23-25]

Subnasal elevation may be used in the C–h premaxilla, because the nasal floor can be elevated from 0 to 4 mm in the central or lateral incisor region and up to 4 mm in the canine area.

WWW.HIGHDENT.IR

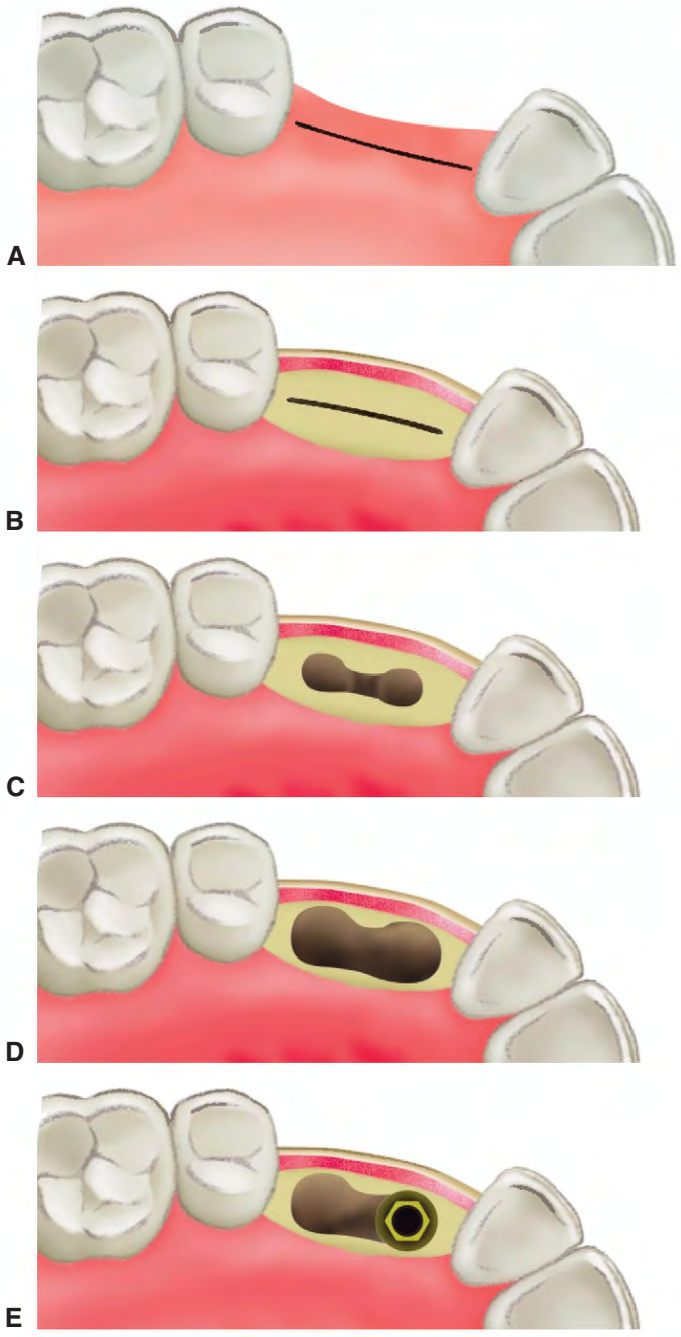


Figure 34-28 **A,** A midcrestal incision is made with keratinized tissue on the facial aspect. **B,** A narrow osteotomy is prepared with a surgical bur. **C** and **D,** The edentulous ridge is gradually expanded facially with round-shaped osteotomes. **E,** The implant may be inserted; however, if the facial plate breaks, then a layered graft with a membrane is added on the facial aspect in addition to an intrapositional bone graft placed after bone expansion to increase the width.

Anatomical Considerations

The anterior nasal spine has an anterior component that averages 4.1 mm (0 to 9 mm) in adults.[26] Posterior and lateral to it, two flat processes, the alae of the premaxilla, project superiorly and laterally (Figure 34-29). The piriform aperture is bounded below and laterally by the maxilla. The breadth of the piriform aperture

Figure 34-29 The natural canine root is distal to the lateral piriform rim of the nose. However, a nasal recess extends distal, behind the piriform rim and over the canine site of a resorbed maxilla, which is more palatal than the tooth root position. The inferior concha of the nose is 4 to 6 mm superior to this recess region.

in adults ranges from 20 to 28 mm and averages 23.6 mm.[26,27] The lower border of inferior piriform rim may be sharp or rounded. This border often rises from the premaxillary bone and ends anteromedially in the anterior nasal spine. The anatomy of the nasal floor is variable in relation to the inferior turbinate and is typically situated 5 to 9 mm below the level of this structure.[28]

When maxillary anterior teeth are present or have maintained the residual bone, the inferior piriform rim is usually level or a few millimeters above the floor of the nose in the central and lateral region.[29] The inferior piriform rim above the nasal floor forms a prenasal fossa, found in 12% of patients.[27,30] In these cases a shallow depression extends toward the alveolar arch behind a sharp border of the inferior piriform rim. As a result, when the inferior piriform rim is used as a guideline for the height of the opposing cortical plate from the crest of the ridge in the maxilla during surgery to determine implant length, the lowest portion of the nasal floor and nasal mucosa may be inadvertently perforated during the implant osteotomy and placement. Instead, the nasal floor may be elevated and grafted before implant placement.

The canine tooth position is immediately distal to the lateral piriform rim. The canine eminence in this area is lost after several years of edentulism, and the crest of the residual C–h bone volume ridge is palatal to the original tooth position. The nasal cavity is usually above, medial, and palatal to the canine position in the dentate patient. However, a nasal recess is present behind the lateral piriform rim. This nasal recess corresponds to the apical region of the canine position in the C–h maxilla, which has resorbed palatally and is now under the nasal cavity. Implants placed in the canine position of a C–h bone volume may extend more superior than the natural canine root and penetrate into the nasal cavity because of the nasal recess behind

WWW.HIGHDENT.IR

the lateral piriform rim. One study reported that during healing, no undesirable side effects occurred secondary to penetration of titanium screws into the nasal cavity.[31] However, because the whole implant length is not placed in bone, soft tissue will encapsulate the apical end, and the functional surface area of the implant is unknowingly reduced.

The lateral and inferior piriform rim may be identified and the nasal mucosa elevated up to 4 mm off the rim and the floor of the nasal cavity or within the recess distal to the lateral rim, creating a subnasal pocket for augmentation. This procedure has also been performed in conjunction with bone augmentation from the iliac crest or with mandibular blocks.[32-37]

The arterial blood supply to the nose is derived from both the external and internal carotid arteries. The terminal branch of the maxillary artery (a branch of the external carotid) supplies the sphenopalatine artery, which supplies the lateral and medial wall of the nasal chamber. The anterior and posterior ethmoid arteries (branches of the ophthalmic artery) supply the nasal vestibule and the anterior portion of the septum. In addition, a few vessels from the greater palatine artery pass through the incisive canal of the palate to reach the anterior part of the nose.

At the junction between the squamous epithelium of the nasal vestibule and the respiratory epithelium of the nasal cavity lies a strip about 1.5 mm wide covering a region of wide and long capillary loops, known as *Kiesselbach's plexus*.[38] It extends to the lower and central part of the cartilaginous septum and is a common region for nose bleeds. The subnasal graft is positioned anterior to this region.

The fifth cranial nerve innervates the region of the subnasal graft. Infiltration anesthesia has been successfully used; however, a more significant regional anesthesia is achieved when the secondary division of the maxillary nerve (V2) is blocked. Using this technique, anesthesia of the hemimaxilla, side of the nose, cheek, lip, and maxillary sinus can be achieved. The use of a long-acting anesthetic (e.g., bupivacaine 0.5% with epinephrine 1:200,000) is preferred, because it may also decrease postoperative discomfort. Xylocaine 2% with 1:100,000 epinephrine is infiltrated into the labial mucosa and palatal region to evaluate the V2 block and may enhance local hemostasis.

SURGICAL TECHNIQUE

A full-thickness incision is made on the crest of the edentulous premaxilla, from the distal end of the edentulous site on one side to the distal end of the contralateral edentulous site. Vertical lateral relief incisions are then made at the distal aspect of the flap. A full-thickness mucoperiosteal reflection is accomplished first on the palatal aspect, then a similar flap exposes the facial aspect of the maxilla, the nasal spine, and the inferior and lateral piriform rim.

C–h Maxillary Canine Position Implant

The bone on the distal and inferior to the lateral piriform rim is identified. The nasal mucosa in the nasal recess is elevated using a curved soft-tissue curette, in a manner similar to elevation of the sinus mucosa in sinus graft procedures (Figure 34-30, *A*). The back side of the curette is positioned on the soft tissue, and pressure on the blunt aspect of the instrument pushes the nasal mucosa palatal, until the sharp portion of the curette is positioned on the lip of the lateral piriform rim. Because the nasal mucosa is generally thicker and more tear resistant, it is easier to elevate from the bony walls than its maxillary sinus counterpart. However, increased pressure against the bone is required, compared with membrane elevation in maxillary sinus procedures, because of the presence of elastic fibers in the periosteum, which adhere more tenaciously to the underlying bone. The curette then slides down the lateral aspect of the nasal fossa and along the distal aspect of the inferior piriform rim. Depending on the depth of the depression behind the lateral piriform rim, the nasal mucosa is elevated approximately 3 to 5 mm distally and superiorly.

Once the periosteum of the prenasal fossa is elevated, a sharp probe is used to aggressively scratch the floor and lateral walls of the nasal cavity in the pocket created to induce a regional acceleratory phenomenon (RAP). The pocket created for the lateral piriform rim elevation is approximately 5 × 5 mm, which is relatively small; therefore an autograft usually can fill the region (Figure 34-30, *B*). Autogenous bone has long been considered the gold standard and remains the material of choice for the subnasal graft because of its osteogenic, osteoinductive, and osteoconductive properties, which enhance bone formation and provide greater initial density after bone modeling. Because the donor site is usually chosen depending on the volume and type of bone desired, the subnasal graft generally requires less than 2 cc unilaterally and is easily filled with all autogenous bone from a maxillary harvest (Figure 34-30, *C* and *D*). It can usually be harvested from maxillary intraoral sites, maxillary tuberosity, exostoses, and/or debris from implant osteotomies. The cortical and trabecular autogenous bone chips are applied from the most posterior regions of the nasal space to the most anterior labial region of the piriform rim.

The implant may be inserted in conjunction with the subnasal elevation, because the augmentation only concerns the apical region of the implant.

When implants are inserted simultaneously with the nasal augmentation, the implant osteotomy in the canine position stops 1 to 2 mm short of the nasal

WWW.HIGHDENT.IR

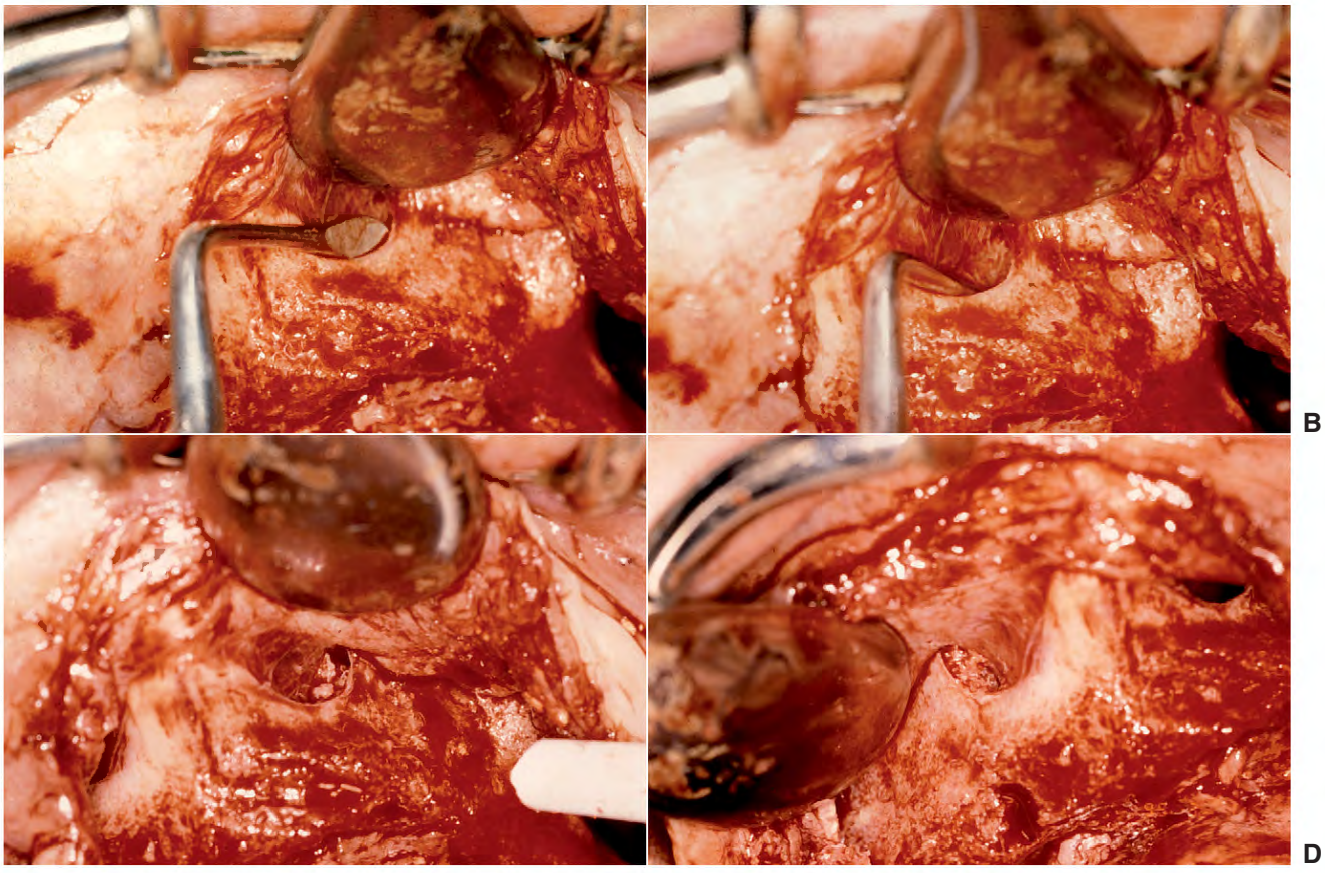


Figure 34-30 **A,** A soft tissue currette is used to elevate the nasal mucosa off the left canine recess region of the premaxilla. **B,** The soft tissue curette creates a submucosal space approximately 4 mm in height, 5 mm deep, and extends to the vertical wall of bone on the distal of the canine recess site. **C** and **D,** An autograft (tuberosity) is used to fill the left and right canine recess site.

floor (similar to an SA2 sinus lift approach). The final diameter of the implant osteotome is obtained in the usual sequence of drill diameters. A flat-ended osteotome, similar to the sinus lift design, is used to create a greenstick fracture to the floor (this also enhances the RAP phenomenon). Bleeding (although rarely a concern during the subnasal elevation) may be controlled with pressure, collagen, or electrocautery. If bleeding originates from the Kiesselbach's plexus, then pressure is applied through a nasal packing for 15 to 30 minutes. A cotton roll, lightly coated with petroleum jelly, is a convenient nasal-packing material. A string of dental floss may be tied around one end of the cotton roll to allow the removal of the sponge with ease.

During installation of an implant in the canine position, the osteotomy may inadvertently penetrate the maxillary sinus, because this structure often interfaces with the lateral piriform rim and nasal cavity in the long-term edentulous patient. A sinus graft procedure is most often performed with posterior implants inserted, which decreases this risk when the sinus floor is filled to the anterior wall. Rigid fixation of the implants should always be confirmed at the time of surgery, and any implant that is mobile or not adequately stabilized should be removed because it may perforate the nasal mucosa, become dislodged into the nares, and ultimately end up in the trachea (Figures 34-31 to 34-33).

C–h Maxillary Central to Lateral Incisor Implant

The maxillary central to lateral incisor region in C–h cases presents 7 to 11 mm of vertical bone and more than 6 mm of available bone width. The crest of the residual ridge is palatal to the original incisor position, and the prosthesis should most often be an overdenture. The subnasal elevation procedure is performed in a fashion similar to the C–h canine implant (Figure 34-34). The elevation takes place from the inferior piriform rim and onto the floor of the nasal cavity (Figure 34-35). During subnasal elevation in the incisal regions, the soft tissue curette should stay against the bone lip and follow the depression when evident. The nasal depression below the inferior piriform rim may drop as much as 2 to 4 mm. When this occurs, the nasal floor augmentation is more predictable, because a facial bony wall improves the number of walls of host bone for the new bone formation. The subperiosteal space in the nasal floor is usually 5 mm deep and 2 to 4 mm

WWW.HIGHDENT.IR

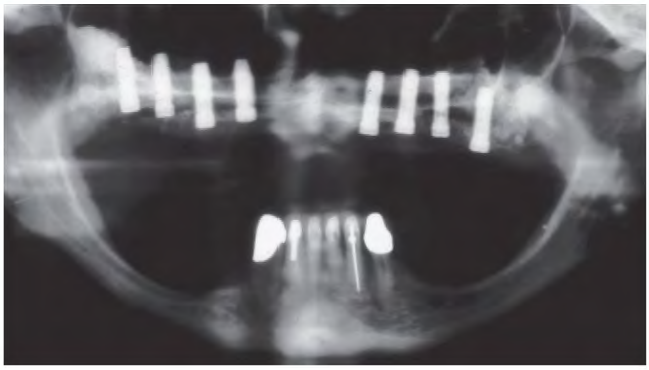


Figure 34-31 Postoperative panoramic radiograph of the implants in the canine sites after the nasal elevation and graft.

Figure 34-32 The eight healed implants in the edentulous maxilla with the canines in a C–h bone volume after nasal elevation and augmentation.

high. Autologous bone is harvested from the maxilla and fills the space. The implant osteotomy is prepared to the final drill diameter for the implant, 2 mm short of the nasal floor (not the piriform rim) (Figure 34-36). An implant 11 to 13 mm may then be placed into the osteotomy, which extends 2 to 4 mm above the original nasal floor (Figure 34-37). The prenasal fossa augmentation procedure may also be performed in conjunction with autogenous bone grafts to decrease the height requirement for the onlay graft before the placement of implants.

Postoperative Instructions for Subnasal Elevation

The postoperative instructions for subnasal grafts and/or implant insertion are similar to those for most oral surgical procedures. In addition, the patient is instructed not to place fingers in the nose to prevent retrograde pressure and implant mobility.

Complications

The unique complications associated with nasal elevation and implant insertion include tearing the nasal

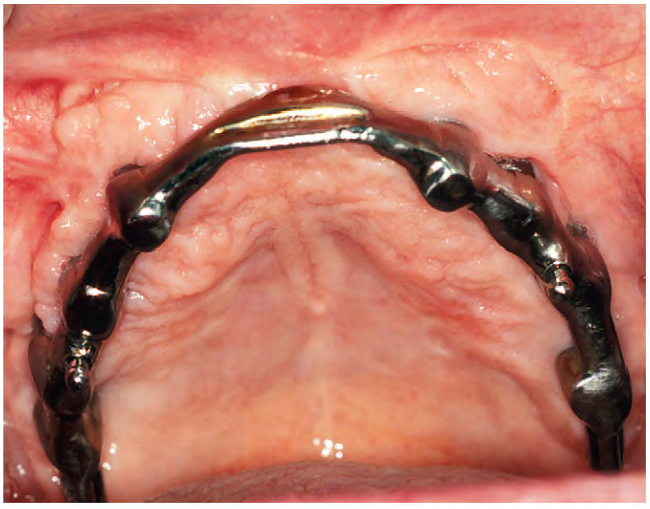


Figure 34-33 The eight implants are splinted together with a bar. A keratinized tissue graft was placed on the facial aspect of the canine implants.

Figure 34-34 A nasal recess area often exists behind the inferior piriform rim of the nose. This region may be elevated 2 to 4 mm in a C–h anterior maxilla for additional implant length.

mucosa and the implant extending into the nares proper. When the nasal mucosa is torn, bacteria from the nasal cavity may infect the implant site. As a result, the nasal mucosa tear should be closed with a horizontal mattress suture and a water-tight closure. No tension should exist on the suture line over the graft and implant, which may result in a delayed incision opening. This is more complicated, because the healing mucosa may feel inflamed and patients often rub the area with their fingers, resulting in reopening of the tear.

On occasion, the implant does not perforate the nasal mucosa during the surgery; however, years later the implant may become exposed in the nares proper. The threaded design may capture mucous or nasal scabs and contribute to odor or even rhinosinusitis. A bone graft before implant insertion decreases this risk. However, when this occurs to a rigid implant, consideration

WWW.HIGHDENT.IR

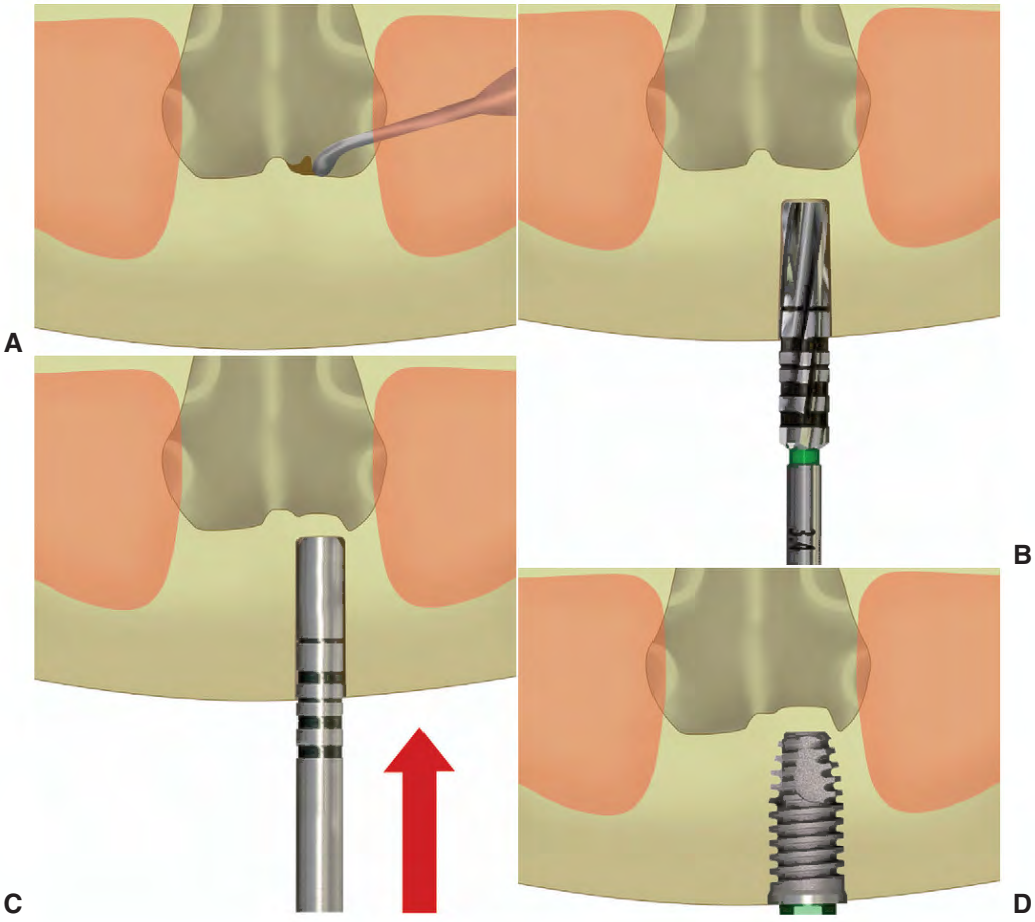


Figure 34-35 A diagram of the central-lateral site nasal elevation. **A,** The nasal mucosa is elevated from the nasal recess, and submucosal pocket is created 2 to 4 mm high, 4 mm deep, and 5 mm wide. **B,** A bone drill prepares the implant site 1 to 2 mm below the floor of the nose. **C,** A flat-ended osteotome is inserted and greenstick fractures the apical bone into the recess site. **D,** An implant is threaded into the site and extends 2 to 4 mm beyond the original nasal floor.

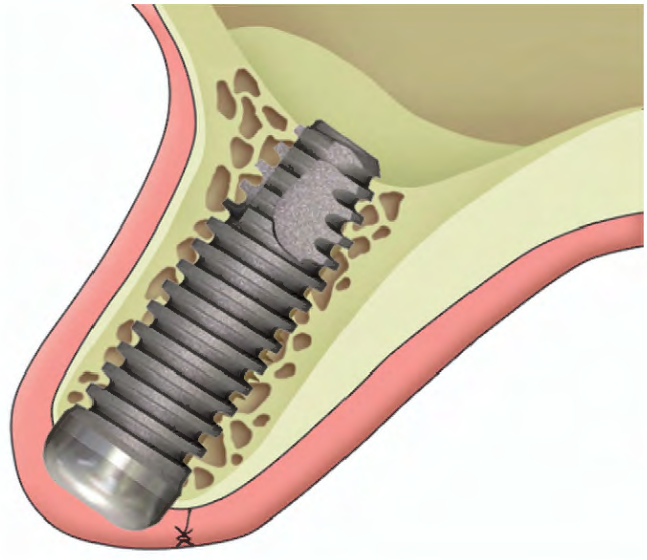


Figure 34-36 The implant is inserted in the central-lateral position and extends through the nasal floor 2 to 4 mm into the subnasal graft.

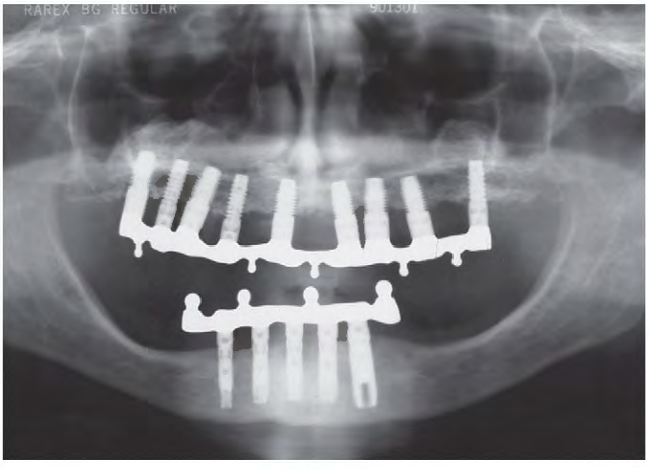


Figure 34-37 A panoramic radiograph of a C–h maxilla with sinus graft and canine and central implants, with subnasal elevation and augmentation. These nine implants are splinted together for an RP-4 overdenture.

WWW.HIGHDENT.IR

may be given to perform an implant apicoectomy and remove the protruding aspect of the implant.

Incisive Foramen Implant

The incisive foramen region, rather than a central incisor site, may also be used to insert an endosteal implant, especially when an overdenture is the intended final prosthesis.[39] The incisive canal ranges in length from 4 to 26 mm and is directly related to the height of bone in the premaxilla.

As alveolus height is resorbed, the canal reduces in length; therefore Division A, B, and C–w bone has greater canal length than Division C–h and D. The incisive canal has an average axis of 70 degrees with a range of 57.0 to 89.5 degrees from the horizontal plane.[30] This structure contains terminal branches of the nasopalatine nerve, the greater palatine artery, and a short mucosal canal (i.e., Stensen's organ). However, the reflection of the palate rarely results in bleeding (and almost never arterial bleeding); although an artery may be present, it appears that a venous return structure rather than an arterial source is present to the palate. A vertical projection above the incisive canal along the nasal floor is called the *premaxillary wing*. The nasal process of the maxillary premaxilla rises 2 to 3 mm above the nasal floor. As a result, when 7 to 11 mm of bone is present below the nasal floor, a large osteotome may create a greenstick fracture in this process above the foramen and permit the placement of a 9- to 14-mm implant. The foramen is usually 4 to 6 mm in diameter at the crest and narrows down to 4 mm at the apex. Therefore implants inserted at the same time as the soft tissue is curetted are usually 5 to 6 mm in diameter.

The incisive foramen is first reflected and identified, and a periodontal probe evaluates the angle and depth of the bony canal to ensure a minimum length of 9 mm. The soft tissues in the incisive canal are curetted from the canal site, which is approximately 4 mm in diameter at its apex. A large round drill may be used to this effect.

Once the soft tissue is removed, drills progressively increase the diameter to the final implant osteotomy diameter 2 mm below the final height of the canal. A blunt osteotome and gentle, sudden impact force with a mallet then prepares the apical 2 mm of the implant site. A large-diameter threaded implant (>5 mm) is generally used and should be greater than the diameter of the foramen (Figure 34-38). When the foramen diameter is greater than that of the implant available, the canal is augmented with an autologous bone graft, and the implant insertion is delayed for several months (Figure 34-39).

Complications

Some unique complications may be found with an incisive canal implant: two surgical complications, one short-term complication, and one long-term complication.

The first surgical complication of an incisive foramen implant is related to the implant that is too small for the foramen and not properly fixated. The implant may be inadvertently pushed through the incisive canal and into the nares proper (Figure 34-40). Because the patient is lying on their back during the surgery, the implant may fall back into the soft plate, then into the trachea or esophagus. If the implant disappears from the oral site, the patient's head should be turned to the side immediately, then down and forward. A nasal speculum and tissue forceps may then be used to recover the implant.

A second surgical complication may include bleeding from the incisive foramen. Although this complication is very rare, it is possible. When reflection of the palatal tissue off the incisive canal is associated with arterial bleeding, a blunt bone tap (mirror handle) may be placed over the canal and a mallet used to hit the instrument firmly, crushing the bone over the artery. After several minutes the procedure may continue, and the implant insertion will obdurate the site and arrest the bleeding.

The short-term complication of an incisive foramen implant is associated with enucleation of the soft tissue from the foramen. Although the author has not witnessed this complication, neurological impairment of the soft tissues in the anterior palate may exist. These complications may be paresthesia to the soft tissue or a dysesthesia as burning sensation. It is logical to include this risk in an informed consent. If it should occur, then removal of the implant for dysesthesia is warranted, whereas paresthesia of the palate most likely is a condition the patient can tolerate without significant issues.

A long-term complication that has been observed twice by the author is the regeneration of the soft tissue in the incisive canal, resulting in bone loss around the implant (Figure 34-41). When the implant is removed and the soft tissue biopsied, nerve fibers can be seen reinvading the site. This most likely occurs because the implant was too small for the size of the foramen, and the soft tissue can reform around the implant. Treatment of this complication includes removing the implant and, if necessary for the treatment plan, then regrafting and/or reimplantation.

SUMMARY

The maxillary surgical considerations for multiple implants in Division A bone are very similar to implant insertion for single-tooth implants. However, more often FP-3 or RP-4 prostheses are fabricated. As such, implant insertion is less critical, and the soft tissue drape is more often restored with the prosthesis. However, when the

WWW.HIGHDENT.IR

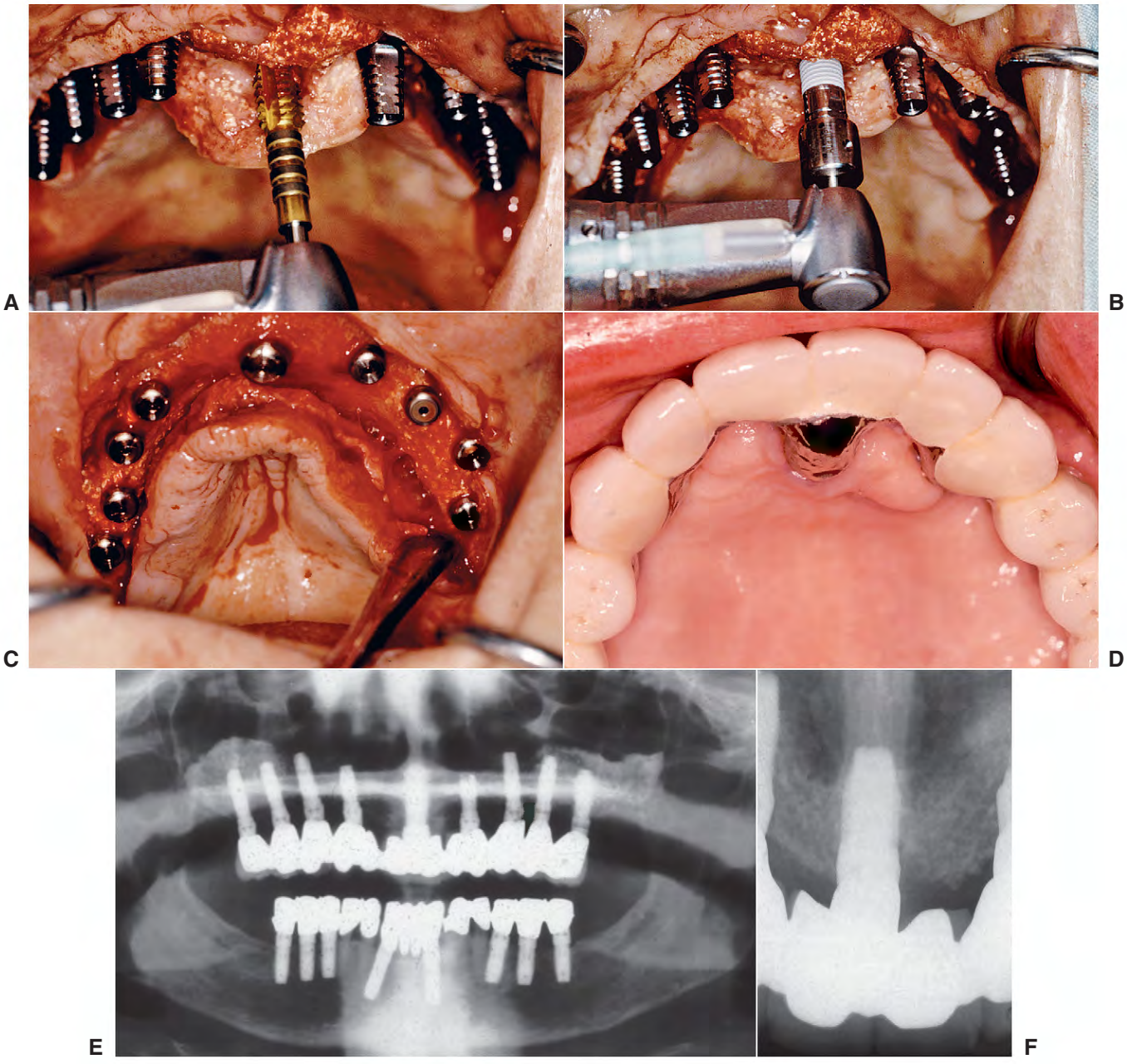


Figure 34-38 **A,** The incisive foramen was excavated and prepared for an implant. The site may be bone tapped before implant insertion. **B,** A 5-mm-diameter implant is inserted into the incisive foramen after implant preparation. **C,** A stage II reentry reveals bone around the incisive foramen implant. **D,** On occasion, a fixed prosthesis may be fabricated with the anterior implants in the canine and incisive foramen position. **E,** A panoramic radiograph of the incisive foramen implant, bilateral sinus grafts, and nine implants (including the canine positions). **F,** A periapical radiograph of an incisive foramen implant after 5 years of function.

patient desires an FP-1 restoration, multiple adjacent implants make this goal more difficult than for a single tooth.

The maxillary lip often requires support when multiple anterior teeth are missing. When a fixed prosthesis is indicated, ridge augmentation is often required to support the lip above the prosthesis. This modality presents unique surgical requirements. Division B bone volume in the maxilla often may be addressed by bone-spreading techniques in conjunction with implant insertion. These procedures require a learning curve that is best developed in Division A bone with D3 or D4 bone density.

The C–h bone volume in the premaxilla may be treated with unique surgical techniques to alter the floor and lateral aspects of the nose or permit implant placement in the incisive foramen. These procedures are most often used for RP-4 or RP-5 restorations.

WWW.HIGHDENT.IR

Figure 34-39 An incisive foramen implant, canine implants, bilateral sinus grafts, and posterior implants splinted together for an RP-4 prosthesis.

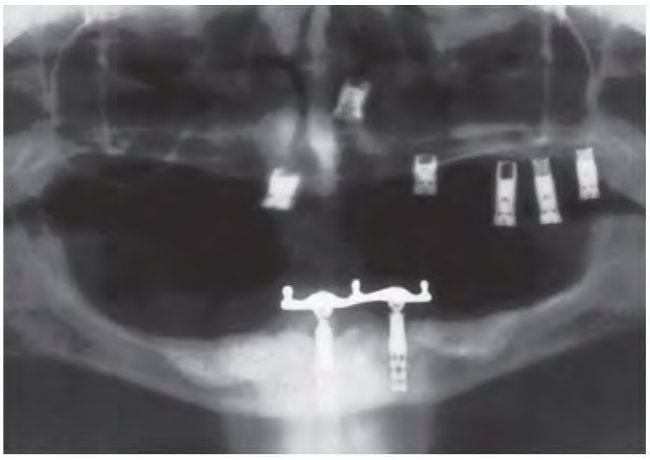


Figure 34-40 A surgical complication of incisive foramen implant surgery is the implant becoming mobile and falling into the nares. This implant can then fall back into the pharynx, esophagus, or trachea.

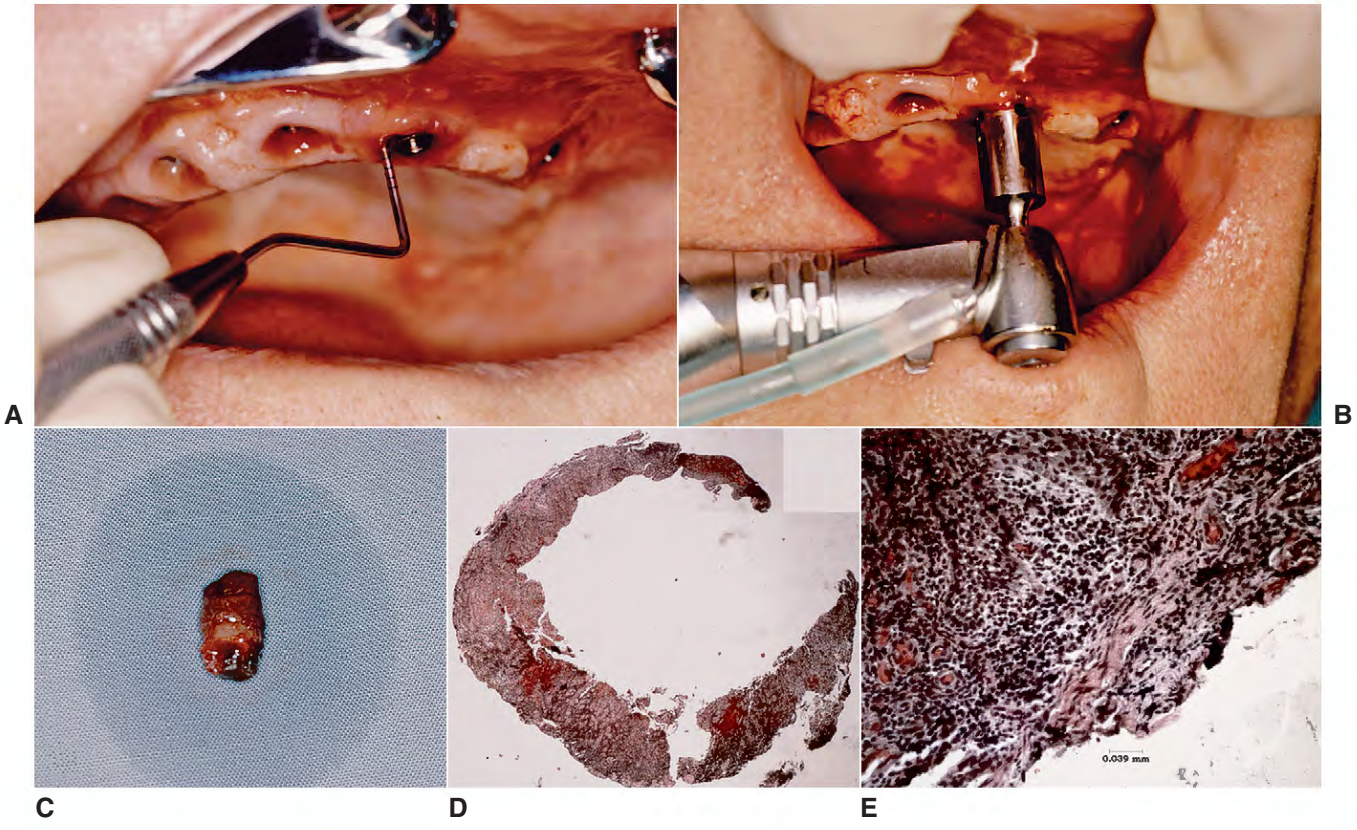


Figure 34-41 **A,** A long-term complication of an incisive foramen implant may be bone loss around the implant that extends the full length of the implant. **B,** When more than 50% of the implant has been lost, it should most often be extracted. A trephine bur may remove the integrated portion of the implant. **C,** The implant removed is surrounded by soft tissue. **D,** Histologic examination of the soft tissue around the implant reveals the contents of the incisive canal are reforming around the implant. **E,** Histologic examination demonstrates nerve fibers in the soft tissue around the implant.

References

1. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent* 90:121-132, 2003.
2. Pietrokowski J: The bony residual ridge in man, *J Prosthet Dent* 34:456-462, 1975.
3. Tatum H: Maxillary and sinus implant reconstruction, *Dent Clin North Am* 30:207-229, 1986.
4. Atwood DA, Coy WA: Clinical cephalometric and densitometric study of reduction of residual ridges, *J Prosthet Dent* 26:200-295, 1971.
5. Gruber H, Solar P, Ilm C: Anatomie und Atrophie bedingte veranderungen der kiefer knochen. In Watzek G,

WWW.HIGHDENT.IR

editor: *Enossale implant in der orale chirurgie,* Berlin, 1993, Quintessence.
6. Botticelli D, Berglundh T, Lindhe J: Hard-tissue alterations following immediate implant placement in extraction sites, *Clin Periodontol* 31:820-828, 2004.
7. Schropp L, Wenzel A, Kostopoulos L et al: Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study, *Int J Periodontics Restorative Dent* 23:313-323, 2003.
8. Misch CE: Bone character, second vital implant criterion, *Dent Today* 7:39-40, 1988.
9. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing and progressive bone loading, *Int J Oral Implantol* 6:23-31, 1991.
10. Misch CE: Treating the edentulous premaxilla. In *Misch Implant Institute manual,* Dearborn, Mich, 1991, Misch Implant Institute.
11. Tarnow DP, Cho SC, Wallace SS: The effect of interimplant distance on the height of interimplant bone crest, *J Periodontol* 71:546-569, 2000.
12. Degidi M, Misch CE: Vertical and horizontal perimplant bone loss: relevance of interimplant spacing [submitted for publication].
13. Tarnow D, Elian N, Fletcher P et al: Vertical distance from the crest of bone to the height of the interproximal papilla between adjacent implants, *J Periodontol* 74:1785-1788, 2003.
14. Tarnow DP, Magner AW, Fletcher P: The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal papilla, *J Periodontol* 63:995-996, 1992.
15. de Oliveira RR, Novaes AB, Papalexiou V et al: Influence of interimplant distance on papilla formation and bone resorption: a clinical-radiographic study in dogs, *J Oral Implantol* 32:218-227, 2006.
16. Ivanoff CH, Grondahl K, Bergstrom C et al: Influence of bicortical or monocortical anchorage on maxillary implant stability: a 15-year retrospective study of Brånemark system implants, *Int J Oral Maxillofac Implants* 15:103-110, 2000.
17. Kiener P, Oetterli M, Mericske E et al: Effectiveness of maxillary overdentures supported by implants: maintenance and prosthetic complications, *Int J Prosthodont* 4:133-140, 2001.
18. Tatum OH: The Omni implant system. In *Surgical manual,* 1981.
19. Tatum OH: The Omni implant system. In Hardin J, editor: *Clark's clinical dentistry,* vol 5, Philadelphia, 1984, Lippincott.
20. Tatum OH: Maxillary and sinus implant reconstruction, *Dent Clin North Am* 30:209-229, 1986.
21. Nkenke E, Kloss F, Whitfag J et al: Histomorphometric and fluorescence microscopy analysis of bone remodeling after installation of implants using an osteotome technique, *Clin Oral Implants Res* 13:595-602, 2002.
22. Dahlin C, Lekholm U, Becker W et al: Treatment of fenestration and dehiscence bone defects around oral implants using the guided tissue regeneration technique: a prospective multicenter study, *Int J Oral Maxollofac Implants* 10:312-318, 1995.
23. Keller EE, Tolman CE, Eckert SE: Maxillary antral-nasal inlay autogenous bone graft reconstruction of compromised maxilla: a 12-year retrospective study, *Int J Oral Maxillofac Implants* 14:707-721, 1999.
24. Keller EE, Eckert SE, Tolman DE: Maxillary antral and nasal one-stage inlay composite bone graft: preliminary report on 30 recipient sites, *J Oral Maxillofac Surg* 52:438-447, 1994.
25. Lundgren S, Nystrom E, Milson H et al: Bone grafting to the maxillary sinuses, nasal floor and anterior maxilla in the atrophic edentulous maxilla: a two-stage technique, *Int J Oral Maxillofac Surg* 26:128-434, 1997.
26. Lang J, Baumeister R: Uber das postnatale Wachtumder Nasenhohle, *Gegenbaurs Morphol Jahrb* 128:354-393, 1982.
27. Lang J: *Clinical anatomy of the nose, nasal cavity and paranasal sinuses,* New York, 1989, Thieme.
28. Bell WH, Proffit WR, White RP: *Surgical corrections of dental facial deformities,* vol 1, Philadelphia, 1980, WB Saunders.
29. Blitzer A, Lawson W, Friedman W, editors: *Surgery of the paranasal sinuses,* Philadelphia, 1985, WB Saunders.
30. Hovorka O: *Die aussere nase,* Vienna, 1893, Hohler.
31. Brånemark PL, Adell R, Albrektsson T et al: An experimental and clinical study of osseointegrated implants penetrating the nasal cavity and maxillary sinus, *J Oral Maxillofac Surg* 42:497-505, 1984.
32. Kahnberg KE, Nystrom E, Bartholdsson L: Combined use of bone grafts and Brånemark fixtures in the treatment of severely resorbed maxillae, *Int J Oral Maxillofac Implants* 4:297, 1989.
33. Higuchi K: Bone grafting into the nasal floor. In Worthington P, Brånemark PI, editors: *Advanced osseointegration surgery, applications in the maxillofacial region,* Chicago, 1992, Quintessence.
34. Jensen J, Sindet-Pedersen S, Oliver AJ: Varying treatment strategies for reconstruction of maxillary atrophy with implants: results in 98 patients, *J Oral Maxillofac Surg* 52:210, 1994.
35. Jensen J, Krantz-Simonsen E, Sindet-Pedersen S: Reconstruction of the severely resorbed maxillary with bone grafting and osseointegrated implants—a preliminary report, *J Oral Maxillofac Surg* 48:27-32, 1990.
36. Misch CE, Dietsh F: Endosteal implants and iliac crest grafts to restore severely resorbed, totally edentulous maxillae—a retrospective study, *J Oral Implantol* 20:100-110, 1994.
37. Misch CE: *Extraoral bone grafts.* Lecture presented at the American College of Oral Implantology, Tarpon Springs, Fla, January 1998.
38. Kiesselbach W: Uber Nasenbluten, *Wien Med* 2:501, 1885.
39. Scher ELC: Use of the incisive canal as a recipient site for root form implants: preliminary clinical reports, *Implant Dent* 3:38-41, 1994.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 35

Immediate Load and Restoration in Implant Dentistry: Rationale and Treatment

Carl E. Misch, Gerard M. Scortecci

Predictable formation of a direct bone-implant interface is a consistent treatment goal in implant dentistry. The two-stage surgical protocol established by Brånemark et al.[1] to accomplish osseointegration consisted of several prerequisites, including the following: countersinking the implant below the crestal bone, obtaining and maintaining a soft tissue covering over the implant for 3 to 6 months, and maintaining a nonloaded implant environment for 3 to 6 months. The primary reasons cited for the submerged, countersunk surgical approach to implant placement were to reduce and minimize the risk of bacterial infection, to prevent apical migration of the oral epithelium along the body of the implant, and to reduce and minimize the risk of early implant loading during bone remodeling. After this procedure, a second-stage surgery is necessary to uncover these implants and place a prosthetic abutment. A high degree of long-term, clinical rigid fixation has been observed with this protocol in completely and partially edentulous patients.[2,3]

During the last 15 years, several authors have reported that root form implants may osseointegrate, even though they reside above the bone and through the soft tissue during early bone remodeling.[4-6] This surgical approach has been called a *one-stage* or *nonsubmerged implant procedure* and eliminates the second-stage implant uncovery surgery. As a result, the tissue discomfort and healing associated with second-stage surgery are eliminated for the patient. The dentist also eliminates the surgical time for uncovery and suture removal. In addition, the soft tissue is already mature before fabrication of the final prosthesis.

Immediate loading of a dental implant not only includes a nonsubmerged, one-stage surgery but also actually loads the implant with a provisional restoration at the same appointment or shortly thereafter. Immediate loading was the initial protocol suggested with dental implants. These implants yielded a wide range of clinical survival.[7-11] On occasion, a direct bone interface could be developed and maintained for more than 20 years[12] (Figure 35-1).

TERMINOLOGY

Immediate restoration of dental implants not only includes a nonsubmerged first-stage surgery but also implies the occlusal surfaces and implants are loaded with a provisional or definitive restoration. Discussions have involved whether a restoration must be delivered at the time of surgery for this description. Because the restoration is not truly loaded immediately after implant insertion, regardless of the technique, an agreement should be established as to what guidelines and language may be acceptable to a majority of the profession. Misch et al.[13] suggested a terminology for immediate restoration and/or occlusal loading. The *immediate occlusal-loading* protocol is an implant-supported temporary or definitive restoration in occlusal contact within 2 weeks of the implant insertion. *Early occlusal loading* refers to an implant-supported restoration in occlusion between 2 weeks and 3 months after implant placement and may use the time period in parentheses (i.e., early [5-week] occlusal loading). *Delayed* or *staged occlusal loading* refers to an implant prosthesis with an occlusal load after more than 3 months after implant insertion. The delayed occlusal-loading approach may use either a two-stage surgical procedure that covers the implants with soft tissue or a one-stage approach that exposes a portion of the implant at the initial surgery. *Nonfunctional immediate restoration* describes an implant prosthesis with no direct occlusal load within 2 weeks of implant insertion and is primarily considered in partially edentulous patients. *Nonfunctional early restoration* describes a restoration in a partially edentulous patient delivered between 2 weeks and 3 months after the implant insertion.

WWW.HIGHDENT.IR

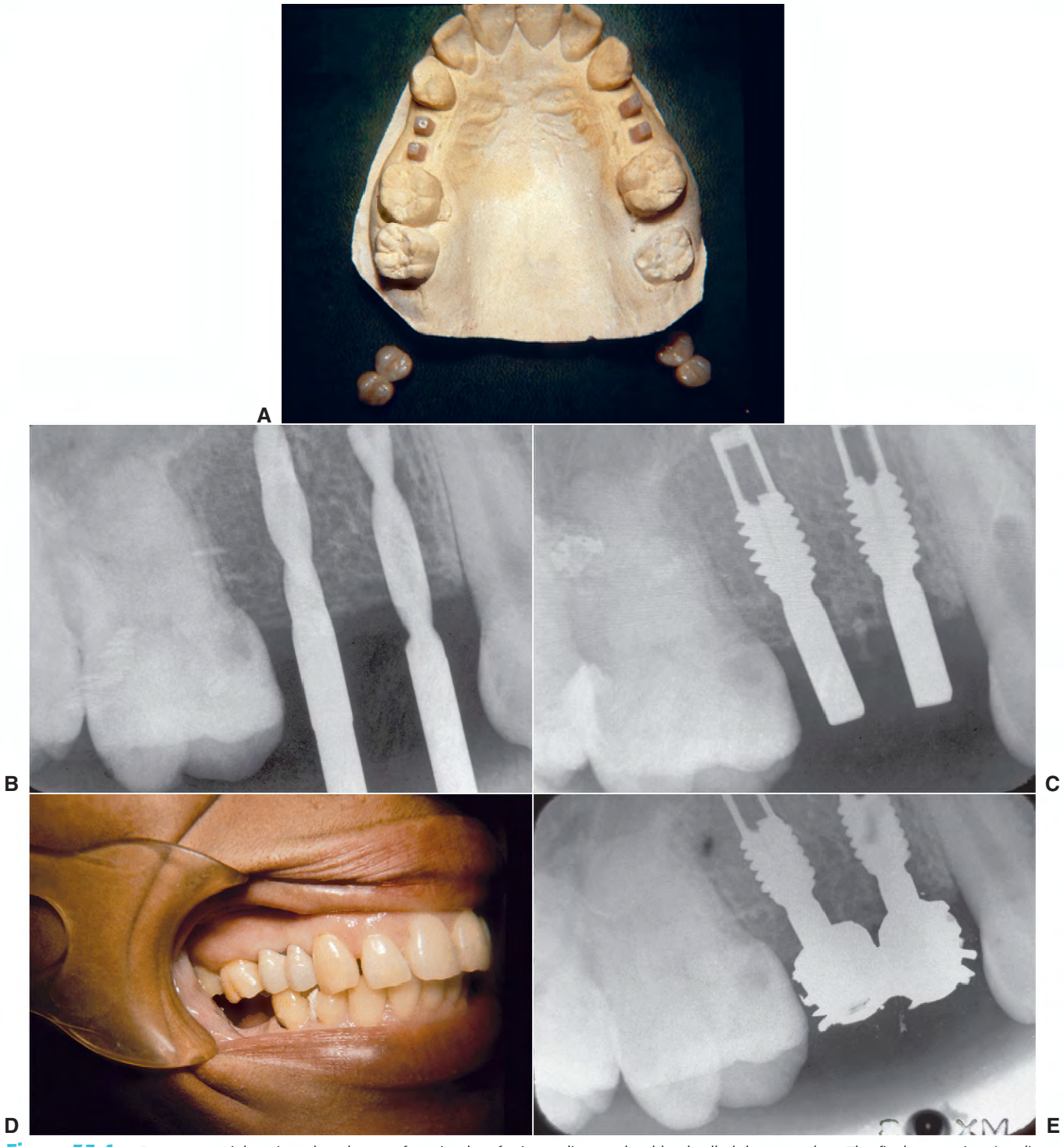


Figure 35-1 **A,** In 1963, Linkow introduced a root form implant for immediate occlusal load called the *vent-plant.* The final restoration (acrylic to metal) was made with implant analogs in the predetermined position. In this patient model, implants will be inserted into the bilateral premolar positions. **B,** The final drills confirmed the location of the two implants. **C,** Two vent-plant implants in position. **D,** The final restoration is relined and delivered in full occlusion. **E,** A postoperative radiograph of two immediate occlusal loaded implants after 10 years in function. ***(A-D,*** *Courtesy L.I. Linkow.)*

COMPLETELY EDENTULOUS PATIENTS

Early studies in immediate loading with root form implants, with a primary goal of a direct bone-implant contact, have been proposed and have shown encouraging results.[14] In general, two different approaches have emerged. The first approach involves placing several more implants than the usual treatment plan for a conventional healing period (Figure 35-2, *A*). Selected implants around the arch (three or more) then are loaded immediately with a transitional prosthesis (Figure 35-2, *B*). Enough implants are left submerged

WWW.HIGHDENT.IR

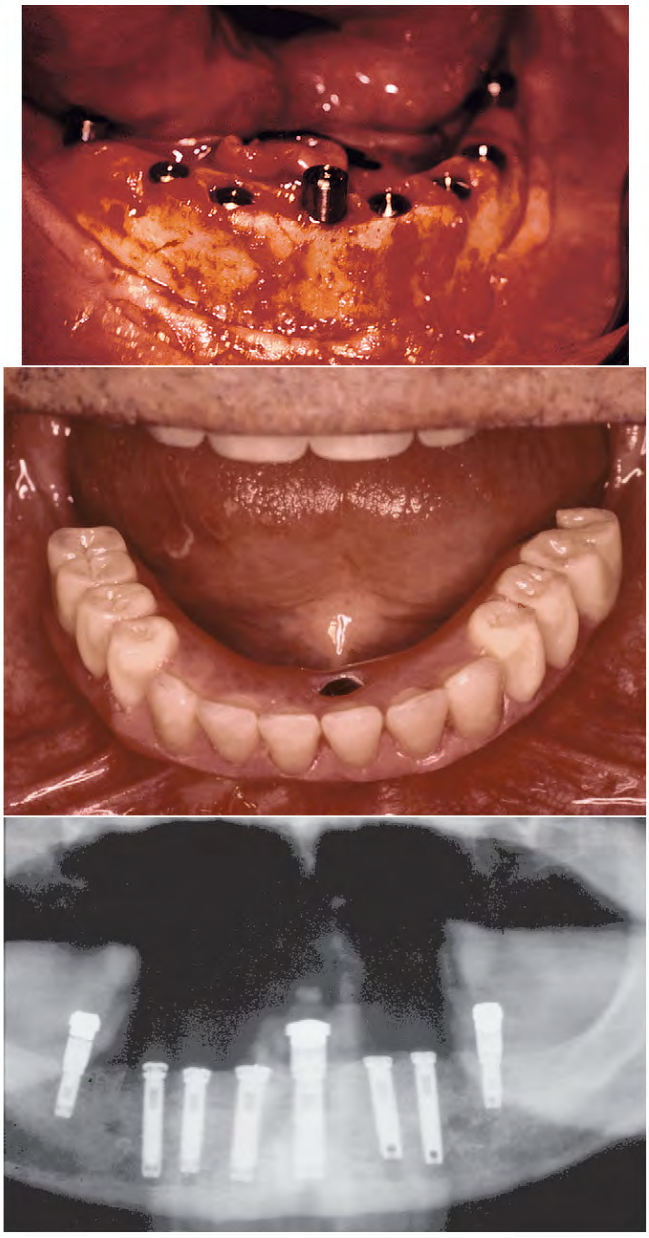


Figure 35-2 **A,** Five implants are inserted between the mental foramens with a two-stage approach. Three additional implants were positioned for immediate occlusal loading of a transitional restoration. **B,** The transitional restoration is connected to the three additional implants for immediate loading. **C,** The panoramic radiograph after 4 months of immediate occlusal loading. *(**A,** Courtesy Craig Misch.)*

for a regular healing period to allow delivery of a fixed prosthesis, even if all immediately loaded implants fail (Figure 35-2, *C*). If any of the implants survive, then they also are used in the final restoration. This protocol was first published by Schnitman et al.[15] in 1990, using 28 screw-shaped implants in 10 mandibles to support a fixed transitional prosthesis. Schnitman et al.[16] suggested that this procedure be used only in the completely edentulous mandible, where moderate to abundant bone was present posterior and anterior to the mental foramens. Using this approach, 100% of the submerged unloaded implant survived, whereas three immediately loaded implants failed before 6 months and one implant failed 18 months after surgery (84% survival) over 9 years.

In 1997, Tarnow et al.[17] reported on immediate loading with a fixed prosthesis, using threaded implants in 10 consecutive completely edentulous cases over 5 years. Sixty-six of 69 implants were integrated in six mandibular and four maxillary completely edentulous arches (96% survival) using a total of 10 to 13 implants in each arch for the final prosthesis. Like Schnitman et al.,[15,16] Tarnow et al. did not immediately load all the implants for the transitional prosthesis.

The other protocol for immediate occlusal loading of dental implants initially loads all of the implants inserted. The implants are splinted together, which decreases the stresses on all the developing interfaces and increases the stability, retention, and strength of the transitional prosthesis during the initial healing phase (Figure 35-3). Often additional implants also are used with this technique compared with the traditional healing method.[18-22]

The immediate-load concept provides all the advantages of the one-stage surgical approach. In addition, implants are splinted together, which decreases the risk of overload because of a greater surface area and improved biomechanical distribution. The patient does not need to wear a removable restoration during initial bone healing, which greatly increases comfort, psychological factors, function, and stability during the transition period. Over the last few years, authors have reported on immediate loading in the completely edentulous patient, with 95% to 100% success rates. However, the influence of immediate loading on crestal bone loss has few animal and clinical reports so as to compare the differences of immediate loading to a more traditional bone-healing time with no functional load.

Rationale for Implant Immediate Loading

Surgical Trauma

The immediate implant–loading concept challenges the conventional healing time of 3 to 6 months of no loading before the restoration of the implant. Often the risks of this procedure are perceived to be during the first week after the implant insertion surgery. In reality, the bone interface is stronger on the day of implant placement compared with 3 months later[23] (Figure 35-4).

The surgical process of the implant osteotomy preparation and implant insertion cause a regional acceleratory phenomenon of bone repair around the implant interface.[24] As a result of the surgical placement, organized, mineralized lamellar bone in the preparation site becomes unorganized, less-mineralized, woven bone of repair next to the implant.[25] The implant-bone interface is weakest and most at risk of overload at

WWW.HIGHDENT.IR

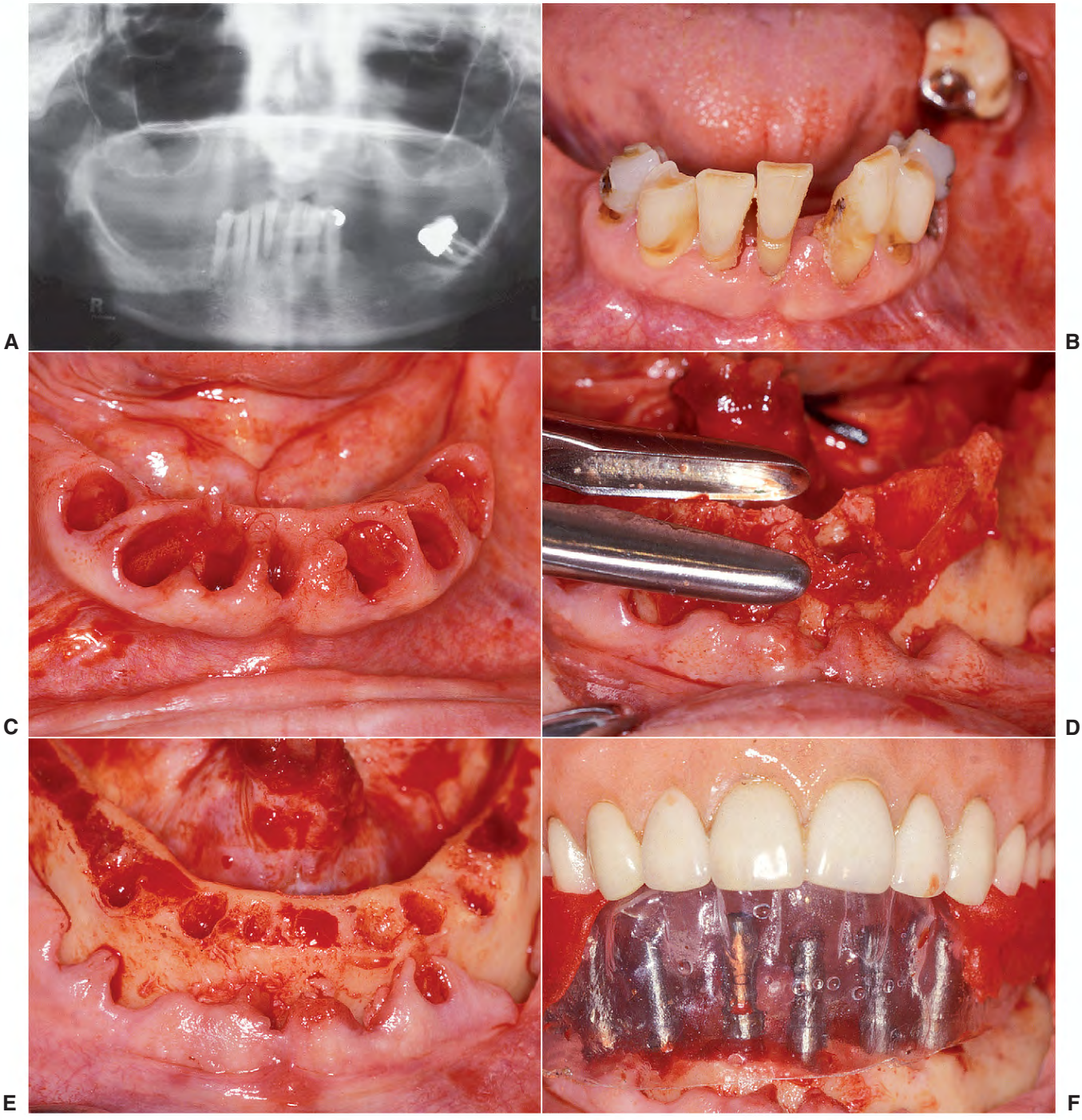


Figure 35-3 **A,** A preoperative panoramic radiograph of eight failing mandibular teeth. **B,** An intraoral view of the eight failing teeth in the mandible. **C,** The eight mandibular teeth are extracted. **D,** A rongeur is used after the tissues are reflected to perform an osteoplasty to the anterior mandible. **E,** The reflected mandibular arch after osteoplasty. **F,** A surgical template indexed to the upper teeth to evaluate the position of six guide pins in the initial implant osteotomies.

Continued

3 to 6 weeks after surgical insertion because the surgical trauma causes bone remodeling at the interface that is least mineralized and unorganized during this time frame. A clinical report by Buchs et al.[26] found immediately loaded implant failure occurred primarily between 3 and 5 weeks after implant insertion from mobility without infection. At 4 months the bone is still only 60% mineralized, organized lamellar bone.[27] However, this has proved to be sufficient in most bone types and clinical situations for two-stage healing and delayed implant loading.

One method to decrease the risk of immediate occlusal overload is to decrease the surgical trauma and amount of initial bone remodeling at implant placement. Roberts et al.[28] reported a devital zone of bone for 1 mm or more around the implant as a result

WWW.HIGHDENT.IR

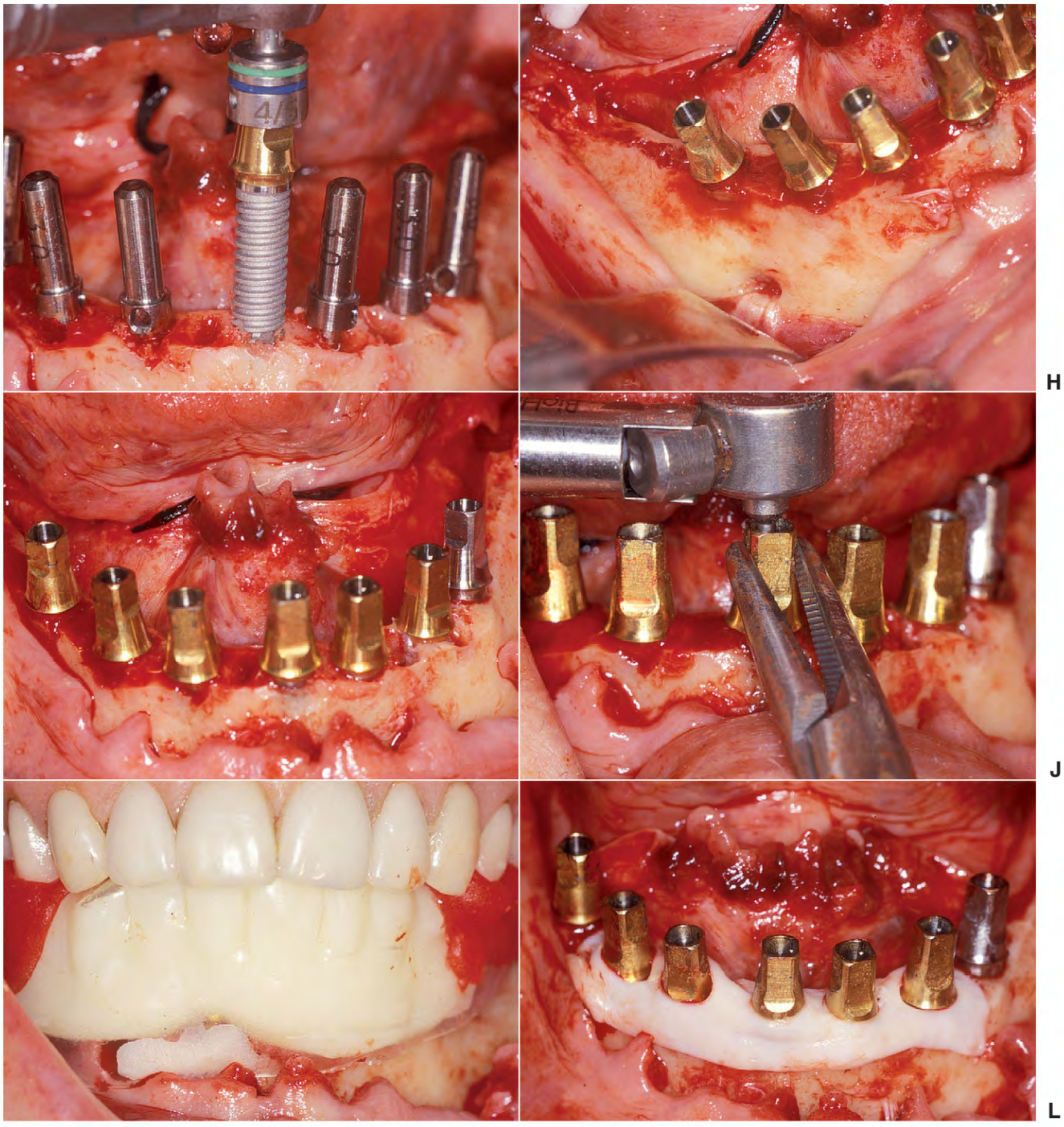


Figure 35-3, cont'd **G,** BioHorizons Maestro implants (BioHorizons; Birmingham, Ala.), which are 15 mm long and 4 mm in diameter, are inserted between the foramens. Longer implants are used when possible for immediate occlusal loading. **H,** An implant is positioned over each foramen to increase the number of implants, increase the anterioposterior (A-P) distance, and decrease the cantilever length of the final prosthesis. **I,** The completed surgery demonstrates seven BioHorizons implants: five between the mental foramens and two above the foramens. **J,** A torque wrench is used to tighten the abutment screws to 30 N-cm to decrease abutment screw loosening during the initial loading period. **K,** A light-cured material (e.g., Triad, Dentsply) is used for the transitional restoration to eliminate acrylic monomer contact on the bone and decrease restorative material shrinkage during setting. **L,** An acellular dermal matrix (AlloDerm; LifeCell, Branchburg, N.J.) is used around the implants to act as a barrier membrane for the extraction sites and to develop a zone of nonmobile tissue around the implants.

Continued

of the surgical trauma (Figure 35-5). Causes of trauma include thermal injury and microfracture of bone during implant placement. Excessive surgical trauma and thermal injury may lead to osteonecrosis and result in fibrous encapsulation around the implant.[29] Eriksson and Albrektsson[30] have reported bone cell death at temperatures as low as 40° C. Sharawy et al.[31] have shown that the amount of heat generated in the bone next to the implant drills depends on the drill design and speed. The temperature next to the drill ranged from

WWW.HIGHDENT.IR

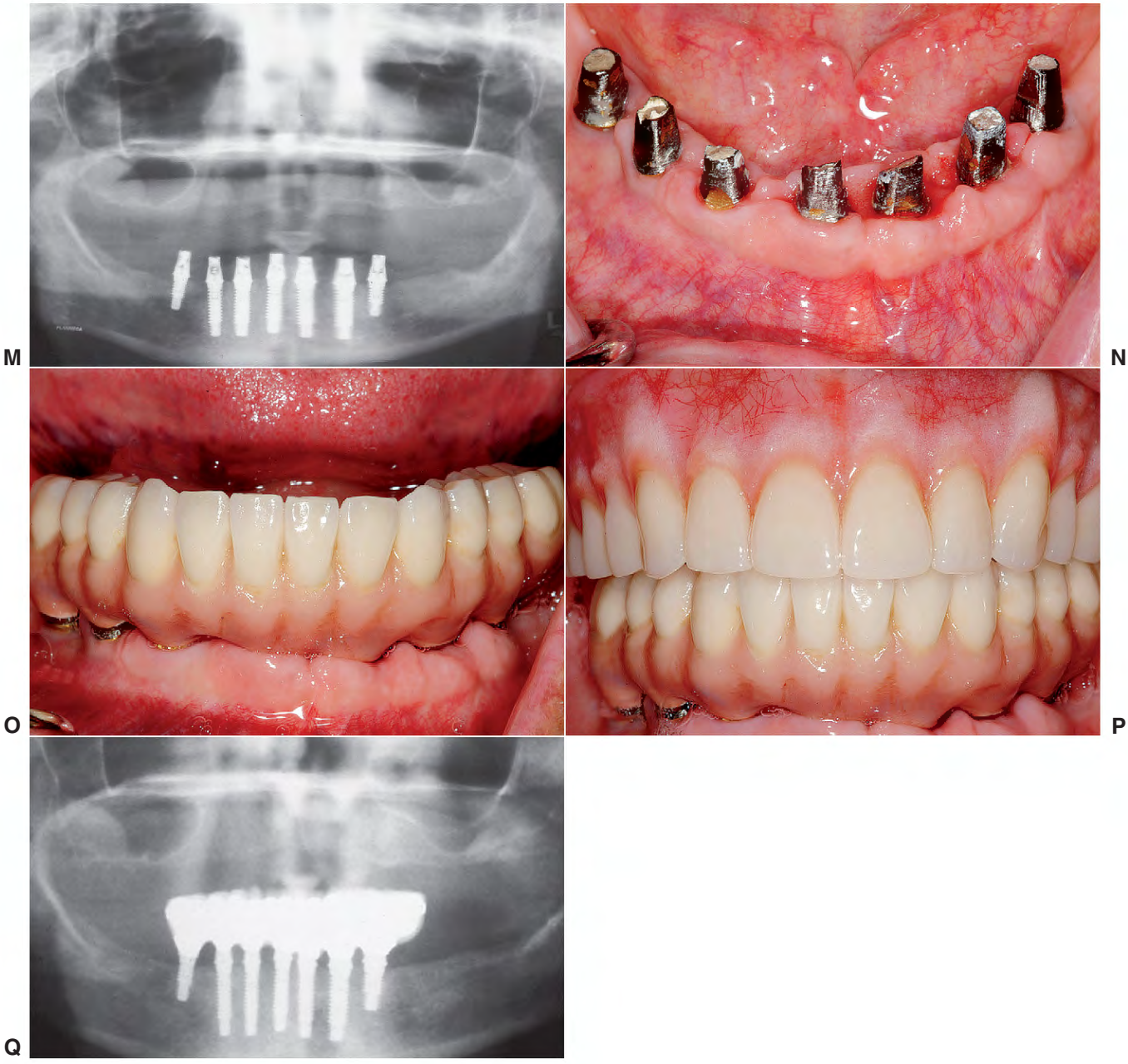


Figure 35-3, cont'd **M,** A panoramic radiograph is obtained to evaluate implant position at the conclusion of the surgery. **N,** After 4 months the immediate-loaded transitional prosthesis is removed and the implants evaluated. **O,** A full-arch fixed, porcelain-metal cemented prosthesis is delivered. **P,** A maxillary complete denture opposes the mandibular fixed prosthesis. **Q,** A postoperative panoramic radiograph evaluates the seven immediate-loaded implants and final fixed prosthesis.

38° C to more than 41° C, from a baseline of 37° C, and required 34 to 58 seconds to return to baseline. In this report, the two implant drill systems with internal-cooled drills cut at a higher temperature than the two systems tested with external irrigation. Other factors related to heat generated to bone include the amount of bone prepared, drill sharpness, depth of the osteotomy, and variation in cortical thickness.[32,33]

A self-tapping implant causes greater bone remodeling (woven bone) around the implant during initial healing compared with a bone tap and implant placement technique.[34] The implant should be nonmobile on insertion, but excess strain from additional torque also may increase the risk of microdamage at the interface. The dentist may believe a protocol for immediate load is to tighten the implant within the bone to 45 to 60 N-cm. Although this concept helps ensure that the implant has rigid fixation and is in good-quality bone, the additional torque used to secure or evaluate fixation of an implant in bone actually may increase the strain at the interface and therefore increase the amount of remodeling, which decreases the strength of the bone-implant interface. Therefore it is prudent to minimize factors related to thermal injury and surgical trauma when considering immediate load to the implant interface.

WWW.HIGHDENT.IR

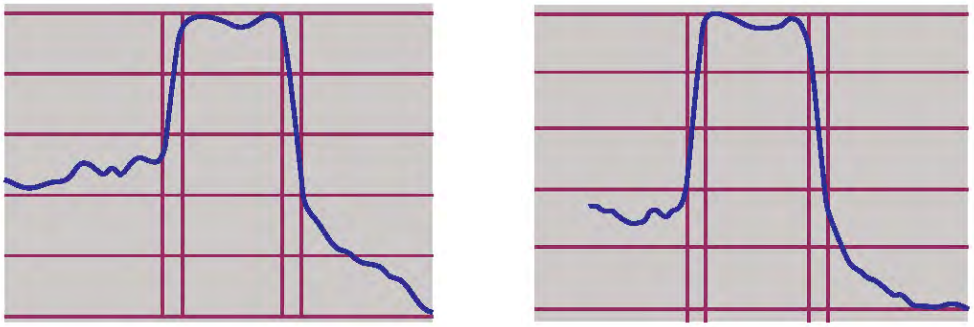


Figure 35-4 **A,** A densitometry profile of an implant 10 days after insertion. The two parallel lines at the interface represent the bone-implant contact. **B,** After 3 months the densitometry profile was repeated. The implant interface is weaker at this time than the initial radiograph showed. *(Data from Strid KG: Radiographic results. In Brånemark PI, Zarb GA, Albrektsson T, editors:* Tissue integrated prostheses*, Chicago, 1985, Quintessence.)*

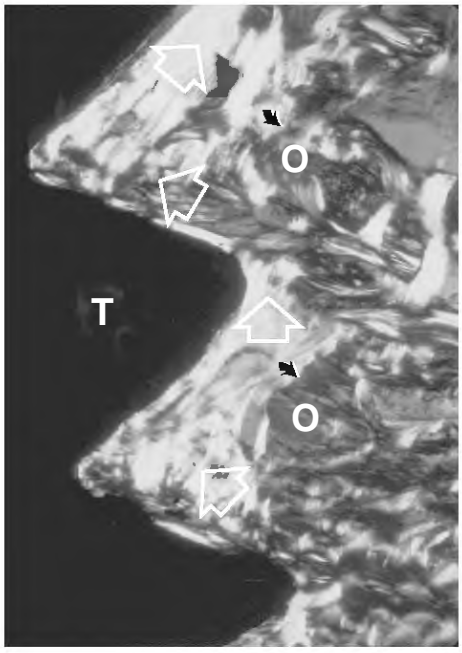


Figure 35-5 Bone remodeling around an implant after surgery replaces a 1-mm or more devital zone of bone. *Arrows* indicate the devital zone of bone replacement. *T,* Implant; *O,* original bone.

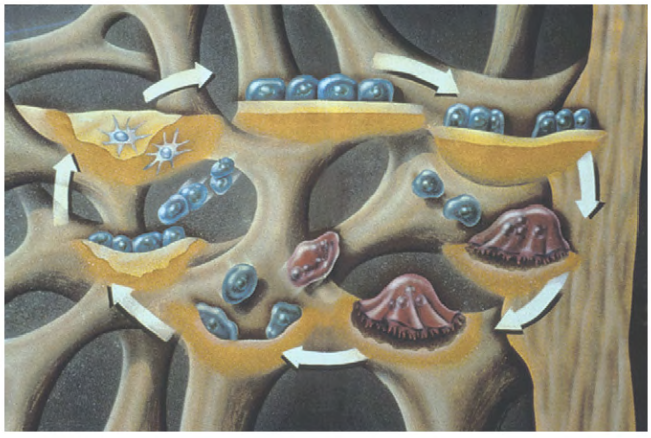


Figure 35-6 Bone remodeling replaces the existing bone with new bone and is controlled primarily by the amount of microstrain within the bone. The rate of the bone remodeling also is related directly to the amount of microstrain.

Bone-Loading Trauma

Cortical and trabecular bone may be modified by modeling or remodeling.[25] Remodeling, or bone turnover, permits the repair of bone after trauma or allows the bone to respond to its local mechanical environment. The bone most often is lamellar but may become woven bone during the repair or remodeling process, so the bone may respond more rapidly to the current situation. Lamellar bone and woven bone are the primary bone tissue types found around a dental implant. Lamellar bone is organized, highly mineralized, is the strongest bone type, has the highest modulus of elasticity, and is called *load-bearing bone*. By comparison, woven bone is unorganized, less mineralized, weaker, and more flexible (lower modulus of elasticity). Woven bone may form at a rate of 60 µm per day, whereas lamellar bone forms at a rate of 1 to 5 µm per day.[27] The classic two-stage surgical approach to implant dentistry permitted the surgical repair of the implant to be separated from the early loading response by 3 to 6 months. Therefore the majority of the woven bone that formed to repair the initial surgical trauma was replaced with lamellar bone. Lamellar bone is stronger and able to respond to the mechanical environment of occlusal loading. Therefore a rationale for immediate loading is not only to reduce the risk of fibrous tissue formation (which results in clinical failure) but also to minimize woven bone formation and promote lamellar bone maturation to sustain occlusal load. The woven bone of surgical trauma has been called *repair bone*, and the woven bone formed from the mechanical response may be called *reactive woven bone*.[35] Remodeling also is called *bone turnover* and not only repairs damaged bone but also allows the implant interface to adapt to its biomechanical situation (Figure 35-6). The interface-remodeling rate is the period of time for bone at

WWW.HIGHDENT.IR

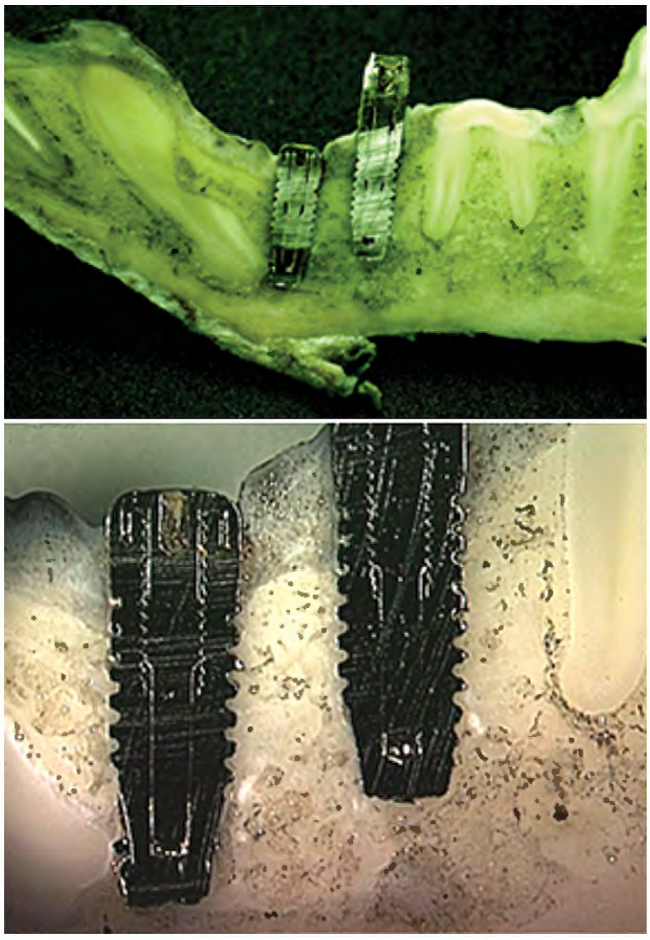


Figure 35-7 Paired BioHorizons Maestro implants inserted into a canine model, with one implant not loaded and the other immediately placed into function for 4 weeks.

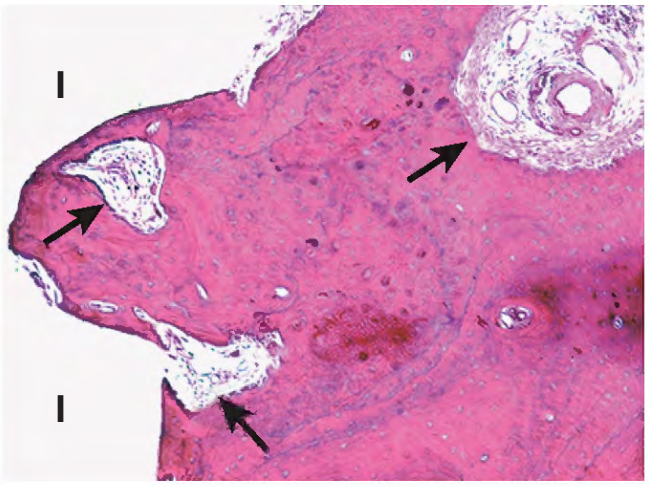


Figure 35-8 No statistical difference in bone-implant contact percent and the volume fractions of the interface were found between the implants immediately loaded and those with no load for 1 month.

the implant interface to be replaced with new bone. Once the bone is loaded by the implant prosthesis, the interface begins to remodel again. However, this time the trigger for this process is strain, rather than the trauma of implant placement. *Strain* is defined as the change in length of a material divided by the original length and is measured as the percentage of change. When the surgical trauma is too great or the mechanical situation is too severe, fibrous tissue may form rather than bone. Fibrous tissue at an implant interface may result with clinical mobility rather than rigid fixation.

Histologic Evaluation: Short Term

General agreement is that excess stresses to an implant interface may cause overload and implant failure. However, immediate loading of an implant does not necessarily result in excessive stresses. The initial histologic response of bone at the implant interface has been evaluated on immediately loaded implants. A direct bone-implant contact with favorable bone quality around the implants is reported. For example, Romanos et al.[36] demonstrated no statistical difference between immediate- and delayed-loaded implants. Sharawy[37] evaluated the immediate- versus delayed-healing interface of 20 BioHorizons dental implants in five adult beagle dogs (Figure 35-7). All implants were inserted into premolar grafted bone defect sites. The implants were paired, so one half the implants were submerged and the adjacent implants received an abutment and were subjected to immediate function for 4 weeks. The implants then were evaluated with histometric analyses of plastic embedded calcified sections. No statistically significant difference ($p > 0.05$) was found in the bone-implant contact ratios between the submerged and loaded implants (Figure 35-8). Similarly, the volume fractions of the interface bone were not significantly different. The bone next to the implants appeared mature and showed evidence of remodeling.

Suzuki et al.[38] performed a clinical and histological evaluation of immediate-loaded posterior implants in nonhuman primates. After loading 10 implants for 90 days, they were compared with 5 control implants with no loading. The bone-implant contact percent (BIC) ranged from 50.3% to 64.1%, with an average of 56.3% for the controls. The immediate-loaded group had one implant failure, seven implants with an average of 67.6% BIC, and two implants with 43.2% and 45.6% BIC, respectively. Therefore the study demonstrated immediate-loaded implants may have a higher BIC than nonloaded implants, most likely a response to the strain conditions in the bone. However, three implants had less BIC or failure when compared with controls. Although benefits exist related to immediate loading, it appears some risks are involved in the procedure.

Testori et al.[39] reported on the histologic interface of two implants in human beings that were immediately loaded after 4 months. The bone contact ranged from

WWW.HIGHDENT.IR

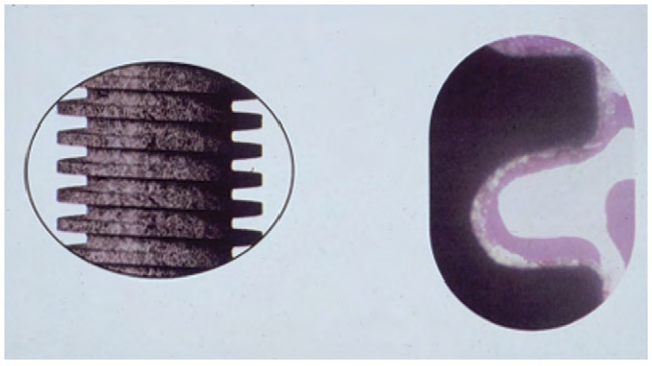


Figure 35-9 A BioHorizons Maestro D4 immediate-loaded implant for 1 year in function from a posterior maxilla in a human being. The bone-implant contact was 83%. *(Courtesy M. Degidi and A. Piatelli.)*

78% to 85%, with no epithelial migration. Therefore immediately loading an implant interface apparently does not necessarily place the interface at increased risk of fibrous tissue formation.

Histologic Evaluation: Long Term

Piatelli et al.[40] evaluated bone reactions and the bone and titanium interface in early loaded implants in monkeys compared with unloaded implants in the same arch several months after immediate loading. No statistically significant differences were detected in the BIC after 8 months. However, loaded implants had less marrow spaces and more compact bone. A later study by the same group demonstrated greater bone contact in immediately loaded implants at 9 months.[41] No fibrous tissue was found at the interface. After 15 months, unloaded and immediately loaded implants were compared, and loaded implants exhibited greater (almost twice) direct bone contact at the interface. In particular, early loaded screws demonstrated thicker lamellar and cortical bone than unloaded implants.[42] This finding suggests that early occlusal loading may enhance bone remodeling and further increase bone density.

Randow et al.[43] evaluated the bone interface in a human patient after 18 months in an immediate-loading situation. They noted a direct bone-implant interface. Ledermann[44] confirmed these results in a 95-year-old patient who had an immediately loaded, bar-connected overdenture in function for 12 years (Figure 35-9). Thus a long-lasting direct bone-implant contact relationship appears possible.

Immediate Occlusal Loading: Factors That Decrease Risks

Bone Microstrain

Loaded bone changes its shape. This change may be measured as strain. Microstrain conditions 100 times less than the ultimate strength of bone may trigger a cellular response. Frost[45] has developed a microstrain language for bone based on its biological response at different microstrain levels (Figure 35-10). Bone fractures at 10,000 to 20,000 microstrain (με) units

Rights were not granted to include this figure in electronic media.
Please refer to the printed publication.

Figure 35-10 Frost has reported on four distinct microstrain patterns within the bone. The acute disuse window results in atrophy, the adapted window is the physiologic response of organized bone, the mild overload zone corresponds to fatigue fractures with reactive woven bone formation, and the pathologic overload zone causes bone resorption. *(Data from Frost HM: Mechanical adaption of Frost's mechanostat theory. In Martin DB, Burr DB, editors:* Structure, function and adaption of compact bone, *New York, 1989, Raven Press.)*

WWW.HIGHDENT.IR

(1% to 2% strain). However, at levels of 20% to 40% of this value, bone already starts to disappear or form fibrous tissue and is called the *pathologic overload zone*. The ideal microstrain for bone is called the *physiologic* or *adapted zone*. The remodeling rate of the bone in the jaws of a dentate canine or human being that is in the physiologic zone is about 40% each year.[46] At these levels of strain, the bone is allowed to remodel and remain an organized, mineralized lamellar bone. This is called the *ideal load-bearing zone* for an implant interface. The mild overload zone corresponds to an intermediate level of microstrain between the ideal load-bearing zone and pathologic overload. In this strain region, bone begins a healing process to repair microfractures, which are often caused by fatigue. Histologically, the bone in this range is called *reactive woven bone*. Rather than the surgical trauma causing this accelerated bone repair, the microstrain causes the trauma from overload. In either condition, the bone is less mineralized, less organized, weaker, and has a lower modulus of elasticity.

One goal for an immediately loaded implant-prosthesis system is to decrease the risk of occlusal overload and its resultant increase in the remodeling rate of bone. Under these conditions the surgical regional acceleratory phenomenon may replace the bone interface without the additional risk of biomechanical overload.

When strain is placed on the horizontal axis and stress is positioned on the vertical axis, the relationship between these two mechanical indexes results in the flexibility or modulus of elasticity of a material. Therefore the modulus conveys the amount of deformation in a material (strain) for a given load (stress) level. The lower the stress applied to the bone (force divided by the functional surface area that receives the load), the lower the microstrain in the bone (Figure 35-11). Therefore one method to decrease microstrain and the remodeling rate in bone is to provide conditions that increase functional surface area to the implant-bone interface.[47] The surface area of load may be increased in a number of ways: implant number, implant size, implant design, and implant body surface conditions. The force to the prosthesis also is related to the strain and may be altered in magnitude, duration, direction, or type. Methods that affect the amount of force include patient conditions, implant position, and direction of occlusal load.

Increased Surface Area

Implant Number

The dentist may increase the functional surface area of occlusal load at an implant interface by increasing implant number.[48] Therefore rather than three to five implants to support a fixed restoration, use of additional implants when immediate loading is planned is more prudent. Immediate-loading reports in the literature with the lowest percentage survival correspond to fewer implants loaded.[15,16,49] Alternatively, a clinical system has been developed specifically for immediate loading with a definitive prosthesis using only three mandibular implants. A 3-year study demonstrated 98% implant survival rates.[50] However, if even one implant fails in this approach, then the patient treatment may require extended time for bone grafting and implant reinsertion before the fabrication of another fixed restoration.

When 10 to 13 implants were inserted and splinted together per arch, implant survival may be greater than 97%.[19-22] The increased number of implants also increases the retention of the restoration and reduces the number of pontics (Figure 35-12, *A*). The increased retention minimizes the occurrence of partially unretained restorations during healing, which can overload the implants still supporting the restoration. The decrease in pontics may decrease the risk of fracture of the transitional prosthesis, which also may be a source of overload to the remaining implants supporting the prosthesis (Figure 35-12, *B*). More implants typically are used in the maxilla (eight to 12) compared with the mandible (five to nine) (Figure 35-13). This approach helps compensate for the less dense bone and increased directions of force often found in the upper arch.

Implant Size

The dentist also may increase the surface area of implant support by the size of the implant. The length of the implant in most systems increases in increments of 2 to 4 mm. Each 3-mm increase in length can improve surface area support by more than 20%.[51] The benefit of increased length is not found at the crestal bone interface but rather in initial stability of the bone-implant interface. Most of the stresses to an implant-bone interface are concentrated at the crestal bone, so the increased implant length does

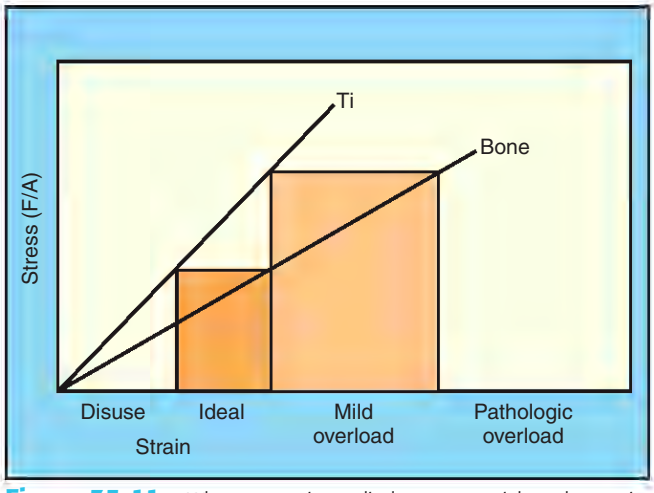


Figure 35-11 When stress is applied to a material, a change in shape occurs (strain). The modulus of elasticity of a material represents the interaction of stress and strain. Titanium (Ti) has a higher modulus of elasticity than bone. When stress is applied to both of these materials, the microstrain difference between the two in the Frost microstrain zone at the interface at 50 units or less is disuse atrophy. When the microstrain difference is 50 to 2500 units, the ideal loading zone is present. Between 2500 and 4000 units, the zone is in mild overload. At more than 4000 units, the zone is in pathologic overload.

WWW.HIGHDENT.IR

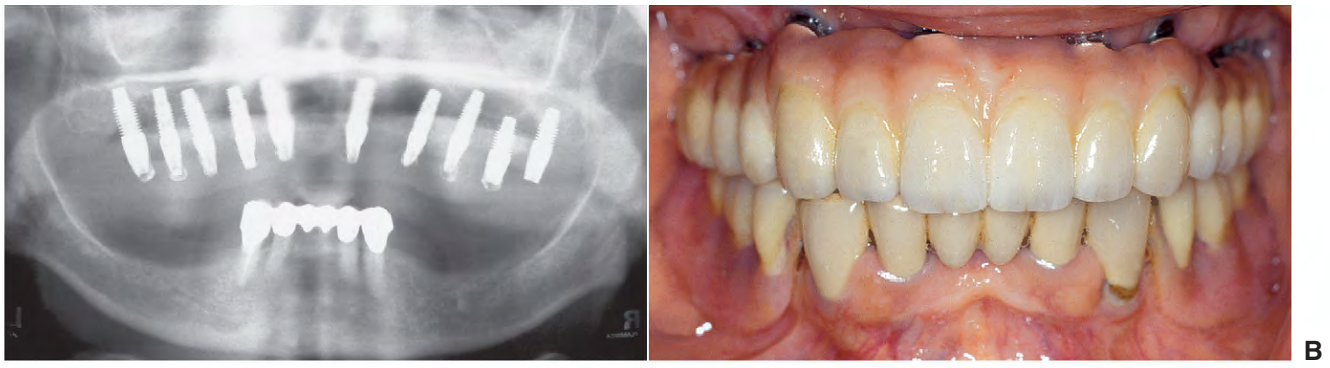


Figure 35-12 **A,** A panoramic radiograph of 10 BioHorizons Maestro implants, inserted and immediately occlusal loaded in the maxillary arch. **B,** An intraoral view of the final porcelain-metal fixed prosthesis after 6 months of function with a transitional restoration.

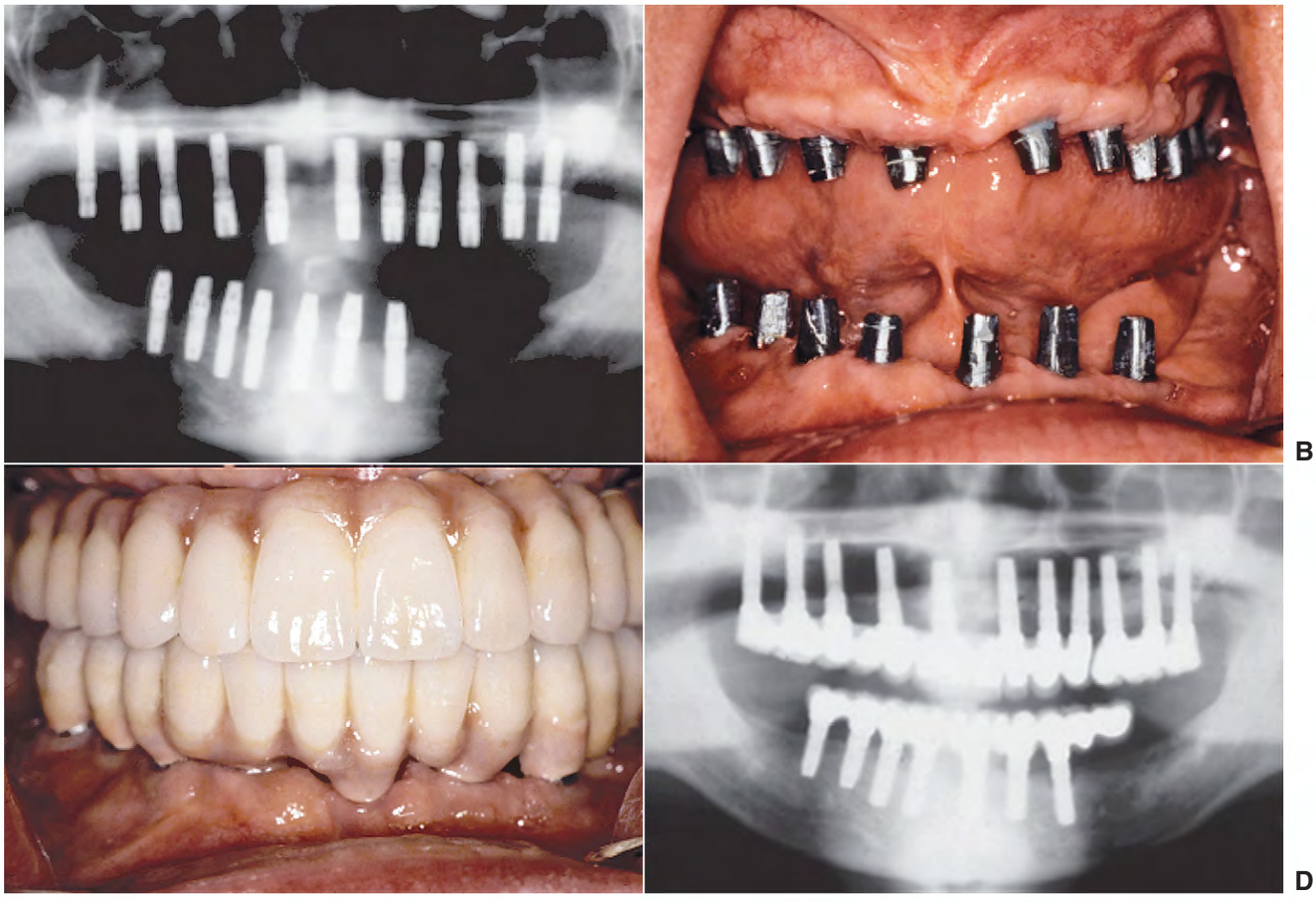


Figure 35-13 **A,** A panoramic radiograph the day of surgery with 11 maxillary implants and seven mandibular implants. More implants generally are used in the completely edentulous maxilla for immediate loading compared with the mandible. **B,** After 6 months the transitional restorations are removed and the final prostheses are fabricated. **C,** The final maxillary and mandibular fixed prostheses (porcelain to metal) supported by 18 implants that were loaded immediately. **D,** A panoramic radiograph of 11 BioHorizons Maestro implants in the maxilla and seven implants in the mandible supporting fixed prostheses.

little to decrease the stress that occurs at the transosteal region around the implant.[52] Therefore length is not an effective method to decrease stress because it does not address the problem in the functional surface area region of the bone-implant interface. However, because the implant is loaded before the establishment of a histologic interface, implant length is more relevant for immediate-load applications, especially in softer bone types. The additional implant length also may permit the implant to engage the opposing cortical plate, which further increases implant initial stability.

The functional surface area of each implant support system is related primarily to the width and the design of the implant. Wider root form implants provide a greater area of bone contact than narrow implants (of similar design). The crest of the ridge is where the occlusal stresses are greatest. As a result, width is more

WWW.HIGHDENT.IR

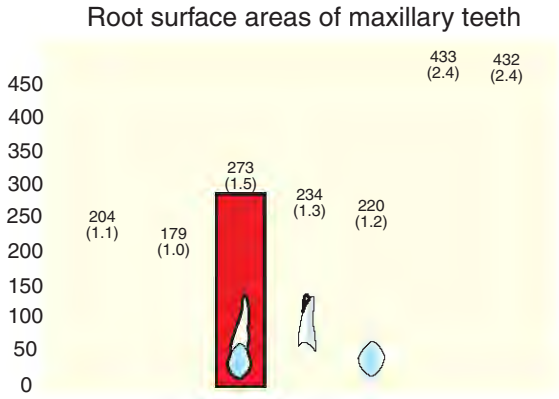


Figure 35-14 The natural dentition root surface area is two times greater in the molar region compared with any other tooth position. Treatment plans for immediate loading should consider implant size or number to increase surface area in this region, especially in the maxilla.

Figure 35-15 The implant body design is more specific for immediate load because the bone does not have time to attach to the surface conditions of the implant before occlusal load. A press-fit implant (the cylinder design on the far right) is least indicated for immediate-load application.

important than length of implant (once a minimum length has been obtained for initial fixation). Bone augmentation in width may be indicated to increase implant diameter when forces are greater, as in cases of moderate to severe parafunction. The major increase in tooth size occurs in the molar regions for natural teeth, where root surface area doubles compared with the rest of the dentition (Figure 35-14). Therefore implant diameter often is increased in the molar region. When a larger-diameter implant is not possible without additional augmentation surgery, more implants may be inserted (i.e., two for each molar), which also is a method to double the overall surface area in the posterior region.

Implant Body Design

The implant body design should be more specific for immediate loading because the bone has not had time to grow into recesses or undercuts in the design or attach to a surface condition before the application of occlusal load. For example, a press-fit implant with a cylinder design does not have bone integration the day of implant placement (Figure 35-15). An implant body with a series of horizontal plates that is tapped or pressed into place does not have bone present between the plates the day of surgical placement. Macrospheres on an implant surface do not have bone present within or around the porous surfaces of the implant the day of implant placement. In general, press-fit implants exhibit decreased initial stability and have an inherent design flaw for immediate-load applications. The surface condition of the implant, although vital for a cylinder implant, is less relevant during immediate-load protocols.

For a threaded implant, bone is present in the depth of the threads from the day of insertion. Therefore the functional surface area is greater during the immediate-load format. The number of the threads also affects

Figure 35-16 A threaded implant design has bone between the threads at the initial surgery. The number of threads per unit length is related to the initial functional surface area of the implant. The implant on the far right has more threads and the most surface area to resist the occlusal loads. The implant on the far left has less surface area.

the amount of area available to resist the forces during immediate loading. The greater the number of threads, the greater the functional surface area at the time of immediate load (Figure 35-16). Some threaded implants have a 1.2-mm distance between the threads (e.g., ITI dental implants), whereas others have a 0.4-mm distance (e.g., BioHorizons dental implants). The smaller the distance between the threads, the greater the thread number and corresponding surface area.[52]

The thread depth varies in implant design. The greater the thread depth, the greater the functional surface area for immediate-load application. The thread depth of some threaded implants is 0.2 mm, whereas the thread depth of other implant designs may reach

WWW.HIGHDENT.IR

0.42 mm.[52] Therefore one threaded implant may have more than two times the overall functional surface area compared with other implants of similar length and width (Figure 35-17).

The functional surface area of an implant body may affect the remodeling rate of bone during loading. A macrosphere implant with reduced surface area may have twice the remodeling rate of a typical threaded implant design. A square-threaded implant design, with deeper threads in greater number, is reported to have a tenfold reduction in remodeling rate under similar loading conditions and approximates 50% per year. The higher the remodeling rate, the weaker the bone interface. The teeth have a bone-remodeling rate of 40% per year, which maintains lamellar bone at the interface.[47]

Figure 35-17 The thread depth of the implant body is related to the initial functional surface area for immediate load. Not all implant designs have the same depth of thread. For example, some designs (i.e., Steri-Oss, Nobel Biocare) have a thread depth of 0.22 mm. BioHorizons Maestro implants have a thread depth up to 0.44 mm. Pictured are two BioHorizons implants. The implant on the right (Maestro D4) is better suited for immediate loading because it has more threads per unit length and is longer. The shorter implant on the left is better designed for a conventional two-stage healing sequence.

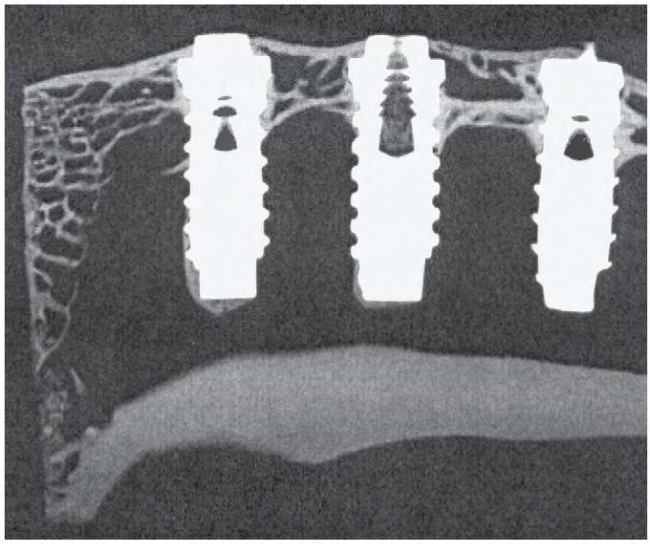


Figure 35-18 In evaluating the initial healing of 72 implants in rabbits, one third of the implants had a V-shaped thread *(left)*, one third a square thread *(center)*, and one third a reverse buttress thread *(right)*. The implants all had the same number of threads, same surface condition, and same size in length and width. The square thread shape resulted in higher bone-implant contact percent and greater reverse-torque values. These two conditions are more conducive for immediate-load applications. (*Courtesy J. Steigenga.*)

The thread geometry also may affect the strength of early osseointegration and the bone-implant interface. Steigenga[53] placed 72 implants into 12 rabbits and reverse-torque tested the unloaded implants after 12 weeks (Figure 35-18). One third of the implants had a V-thread, one third had a reverse buttress shape, and one third had a square thread. The number and depth of threads were the same, as were the width and length of each implant. The V and reverse buttress thread geometry yielded similar values for reverse-torque and BIC values. The square thread demonstrated statistically significantly higher values for both of these evaluations.

Implant thread design may affect the bone turnover rate (remodeling rate) during occlusal load conditions. For a V-shaped thread design, a tenfold greater shear force is applied to bone compared with a square thread shape.[52] Bone is strongest to compression and weakest to shear loading.[54] Compressive forces decrease the microstrain to bone compared with shear forces. Therefore the thread shape and implant design may decrease the early risks of immediate loading while the bone is repairing the surgical trauma.

Implant design affects functional surface area more than implant size. A larger-diameter cylinder implant has less surface area than a smaller-diameter threaded implant. As a result, threaded implants present considerable advantages compared with press-fit implants for immediate-load protocols, because their design features do not require histologic integration to resist loads, and they have greater surface area to resist occlusal forces.

A tapered-implant design presents some disadvantages for immediate-load applications. When the tapered osteotomy is prepared using tapered drills, the implant does not engage the bone physically until the implant is seated almost completely into the bone site. This reduces the initial fixation. In addition, the tapered implant has less overall surface area compared with a parallel-walled, threaded implant. Many tapered-design implants have less thread depth near the apical portion of the implant. This further reduces surface area and initial fixation. The tapered implant is also less likely to engage lateral cortical bone in the apical half of the implant. If the tapered implant should be unthreaded slightly to decrease the depth of placement or change the prosthetic platform position, then the tapered screw becomes less fixated. Therefore, at least in theory, the tapered design has fewer advantages for immediate-load application.

WWW.HIGHDENT.IR

A few clinical trials have compared immediate loading with different implant thread designs and tapered-implant bodies in the completely edentulous patient. The short-term clinical reports indicate a high success rate, regardless of implant design. As a result, overall shape and thread geometry apparently may not be the most important aspects for immediate occlusal load survival. Implant number, implant position, and patient factors most likely are more relevant components of success. Future studies in this area certainly are needed.

Implant Surface Conditions

Implant surface conditions may affect the rate of bone contact, lamellar bone formation, and the percentage of bone contact. The surface condition that allows bone formation in greatest percentage, higher BIC with higher mineralization rate, and fastest lamellar bone formation would be of benefit in immediate loading. These factors have been noted in delayed- and immediate-loading environments with hydroxyapatite (HA) coatings.[55] HA also has been shown to decrease the remodeling rate during occlusal loading.[56] Therefore if the bone is not of ideal density (D4) for immediate loading, then HA may decrease the risk of overload. However, Sullivan et al.[57] found that peak insertion torque and resonance frequency at initial implant insertion were related to implant design rather than surface condition in type 2 and 3 bone. Sirota et al.[58] compared various calcium phosphate ($CaPO_4$)–coated implants to noncoated titanium plasma–sprayed (TPS) implants in immediate-load applications and found a high BIC after 30 days of functional loading in both groups when in good-quality bone. Evans et al.[59] agree that HA-coated and noncoated screws have similar bone contact using a two-stage surgical approach in the mandible. The coating or surface condition of the implant is most beneficial during the initial healing and early loading conditions.

Implant design and surface condition are independent issues that use a different mechanism to reduce the risk of overloading. For example, an HA surface may be applied to a cylinder or a threaded implant. The thread design would be more beneficial to an immediate-load application. However, the HA or roughened surface also may be of benefit during the following healing period, especially at the 3 to 5 weeks when the bone is weakest. The majority of clinical studies have been made with threaded-implant designs. Surface conditions are more difficult to ascertain in the literature and have included a smooth machined surface, a roughened TPS surface, and an HA surface condition without a clear difference in clinical survival. However, evidence is increasing that the machine surface condition is inferior in softer bone types.

Decreased Force Conditions

The dentist may evaluate forces by magnitude, duration, direction, and type. The dentist should reduce conditions that magnify the noxious effects of these forces.

Patient Factors

The greater the occlusal force applied to the prosthesis, the greater the stress at the implant-bone interface and the greater the strain to the bone. Therefore force conditions that increase occlusal load increase the risks of immediate loading. Parafunction such as bruxism and clenching represents significant force factors because magnitude of the force is increased, the duration of the force is increased, and the direction of the force is more horizontal than axial to the implants with a greater shear component.[60] Balshi and Wolfinger[49] reported that 75% of all failure in immediate occlusal loading occurred in patients with bruxism. In their report, 130 implants were placed in 10 patients, with 40 implants immediately loaded and 90 implants following the traditional two-stage approach. The authors reported an 80% survival for immediately loaded implants compared with 96% for the traditional protocol. Grunder[61] appraised immediate loading in eight edentulous patients, four of whom exhibited bruxism. Overall success rates were 87% in the maxilla and 97% in the mandible, with five of the seven implant failures in the bruxism group. Parafunctional loads also increase the risk of abutment screw loosening, unretained prostheses, or fracture of the transitional restoration used for immediate loading (Figure 35-19). If any of these complications occur, then the remaining implants that are loaded are more likely to fail.

Figure 35-19 **A,** An immediate-load prosthesis resulted in overload failure of the left canine implant. This patient has moderate to severe bruxism. **B,** Bruxism caused the transitional prosthesis to fracture into several pieces, and the canine implant became overloaded and failed.

WWW.HIGHDENT.IR

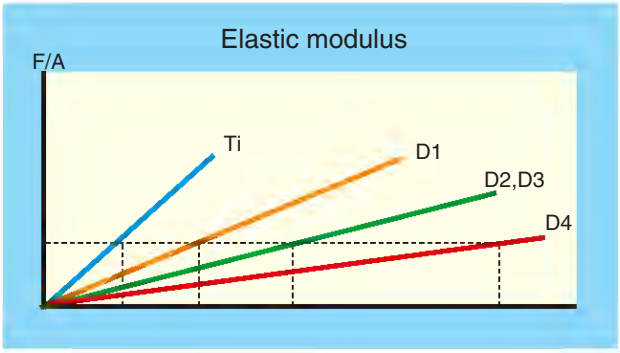


Figure 35-20 The modulus of elasticity is related to the bone density. Therefore the microstrain mismatch between titanium (Ti) and division 4 (D4) bone is greater than that between titanium and D1 bone, even when the stress amount is the same.

Occlusal Load Direction

The occlusal load direction may affect the remodeling rate. An axial load to an implant body maintains more lamellar bone and has a lower remodeling rate compared with an implant with an offset load. In an animal study, Barbier and Schepers[62] observed osteoclasts and inflammatory cells at the interface of offset-loaded implants and noted lamellar bone and a lower remodeling rate around axially loaded implants in the same animal. Therefore the clinician should eliminate posterior cantilevers in the immediate-load transitional restoration because they magnify the detrimental effects of force direction.

Implant Position

Dental implants have been used widely to retain and support cross arch fixed partial dentures (FPDs).[2,63,64] Implant position is often as important as implant number. For example, elimination of cantilevers on two implants supporting three teeth is recommended, rather than positioning the implants next to each other with a cantilever.[48] The cross arch splint forming an arch is an effective design to reduce stress to the entire implant support system. Therefore the splinted-arch position concept is advantageous for the immediate-load transitional prosthesis in completely edentulous patients.

Implant position is one of the more important factors in immediate loading for completely edentulous patients. The mandible may be divided into three sections around the arch: the canine-to-canine area and the bilateral posterior sections. Several clinical reports discuss immediate load in a mandible with only three implants, as long as the implants are positioned in the midline and each posterior region[15,16,50] (see Figure 35-2, *A* to *C*).

The maxilla requires more implant support than the mandible because the bone is less dense and the direction of force is outside of the arch in all eccentric movements. The maxilla is usually divided into five sections, depending on the intensity of the force conditions and the shape of the arch.[65] The minimum five sections include the incisor region, the bilateral canine areas, and the bilateral posterior regions. At least one implant should be inserted into each maxillary section and splinted together during the immediate-loading process.

Concerns have been raised regarding cross arch splinting in the mandible because of mandibular flexure and torsion distal to the mental foramens. Clinical reports indicate the acrylic used in the transitional prosthesis is flexible enough to alleviate these concerns. However, the final restoration should be fabricated in at least two independent sections when implants are placed in both posterior molar positions.[66]

Mechanical Properties of Bone

The modulus of elasticity is related to bone quality (Figure 35-20). The less dense the bone, the lower the modulus. The amount of BIC is also less for less dense bone. The strength of the bone also is related directly to the density of the bone. The softer the bone, the weaker the bone trabeculae.[65-68] In addition, the remodeling rate of cortical bone is slower than that of trabecular bone. As such, the cortical bone is more likely to remain lamellar in structure during the immediate-loading process compared with trabecular bone.

The bone in the anterior regions of the jaw may have cortical bone at the crestal and apical region of the root form implant, whenever the implant is long enough to engage both cortexes. The anterior root form implants should attempt to engage the opposing cortical plate when immediate load is contemplated. The improved biomechanical condition of the cortical bone and the additional implant surface area are advantageous. The maxillary cortical bone is thin compared with the mandibular counterpart at the crestal region and the opposing landmark. In the posterior regions, the maxillary sinus and mandibular canal usually negate the apical engagement of the opposing cortex of bone, which is also thin in the maxilla.

Cortical bone is also present on the lateral aspects of the residual ridge. Root form implants do not typically engage these plates unless the edentulous ridge is narrow. A disk-implant developed by Scortecci[18] is a three-dimensional implant designed to engage the lateral cortical bone plates and the crestal bone region. As such, the implant may have some particular advantage for immediate-load applications, especially in the posterior regions of the mouth when bone grafting is contraindicated or inadequate for traditional root form implant designs.

Bone grafting must depend on several factors to be predictable. Adequate blood supply and a lack of micromovement are two important conditions. The developing bone is woven bone and more at risk of overload. The bone graft in the region of the implant body may lead to less fixation and lower initial BIC. Bone augmentation is more predictable when soft tissue

WWW.HIGHDENT.IR

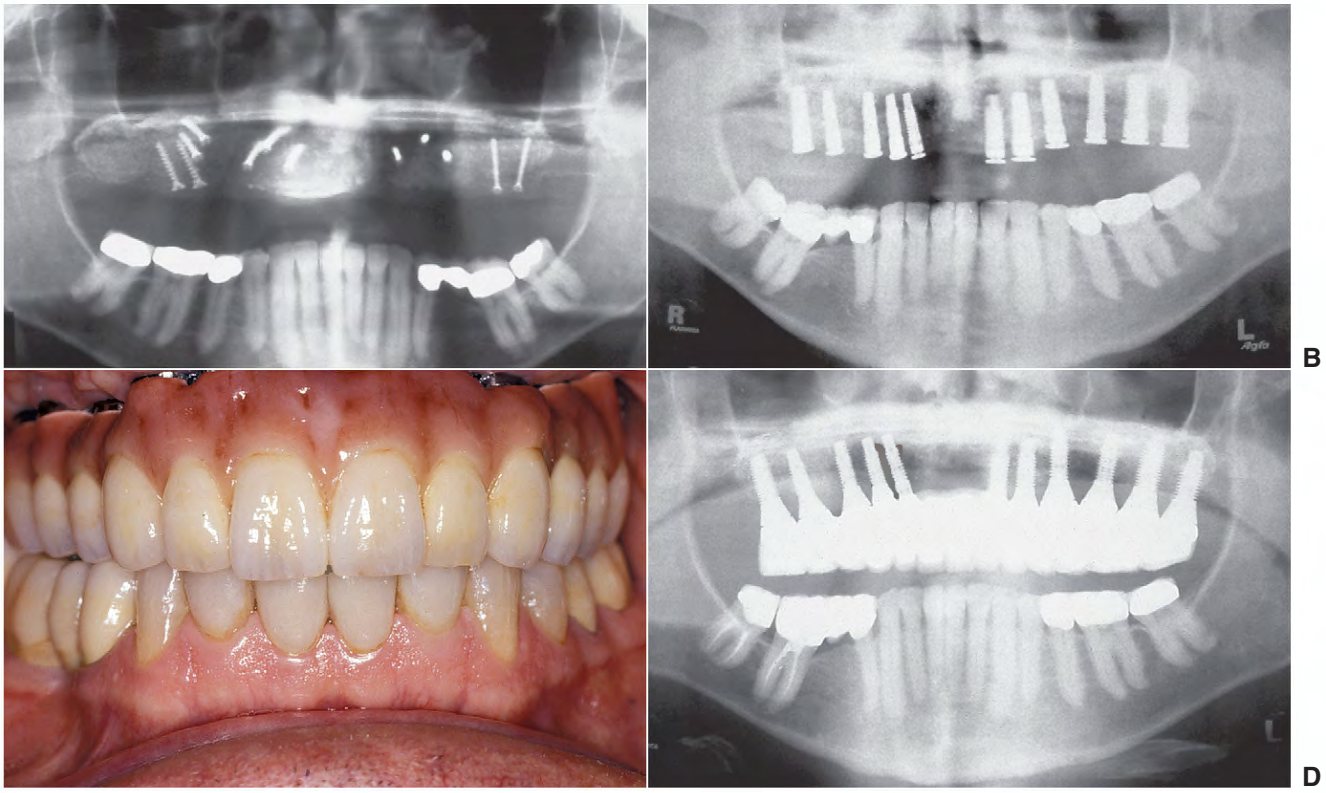


Figure 35-21 **A,** An iliac crest bone graft to the maxilla restores the bone volume of this type 2 C minus height (C–h), D maxilla. **B,** Eleven BioHorizons Maestro implants were inserted, and an impression was made for the delivery of the temporary restoration at the suture removal appointment. **C,** The final restoration is fabricated after at least 6 months. This intraoral photograph illustrates the definitive maxillary porcelain-metal restoration. **D,** A panoramic radiograph of the final maxillary restoration and the corrected mandibular occlusal plane.

completely covers the graft (and membranes when present). All of these conditions make bone grafting, implant insertion, and immediate loading more at risk. Therefore the suggestion is that implants that are immediately loaded be placed in an existing bone volume adequate for early loading and the overall proper prosthetic design. Bone grafting, before implant placement and immediate loading, is suggested when inadequate bone volume is present for proper reconstructive procedures (Figure 35-21).

Immediate-Loading Procedures for Fixed Prostheses

Primarily two different options are available for immediate occlusal loading for the completely edentulous patient desiring a fixed prosthesis. The first option loads the implants the same day as the surgery. The second option is to place the implants and make an impression at surgery. Then at the suture removal appointment 7 to 12 days later, the dentist delivers the transitional fixed prosthesis.

Option 1

Before the surgical appointment, the dentist fabricates a surgical template for implant insertion. For delivery of the restoration the day of surgery, the dentist may take one of two different approaches. The first is to fabricate a denture on the edentulous arch using impressions, baseplate and wax rims, and denture teeth try-in procedures similar to fabrication of a new denture. The second approach modifies the patient's existing denture. In either case, in the region of the future implants, the dentist hollows the restoration to create a shell. If more than two pontics are designed in the transitional restoration, then a metal-reinforced structure is suggested. No posterior cantilevers are fabricated for the transitional restoration when they are out of the esthetic zones during smiling or speech.

The dentist inserts the implants into the preestablished positions (Figure 35-22, *A* to *F*). Implants in good-quality bone, with no crestal bone grafts at insertion, are included in the transitional restoration. The more implants that are loaded at this appointment, the lower the risk of complications. The dentist initially places at least three ideal implant positions in the mandible (bilateral premolars and central) and four ideal implant positions in the maxilla (bilateral molars, bilateral canines) into function. The more additional implants that are inserted, the lower the risk of overload failure, early crestal bone loss, unretained restoration, restoration fracture, and abutment screw loosening.

Once the implants are inserted, the dentist positions the final abutment and tightens it to a torque of

WWW.HIGHDENT.IR

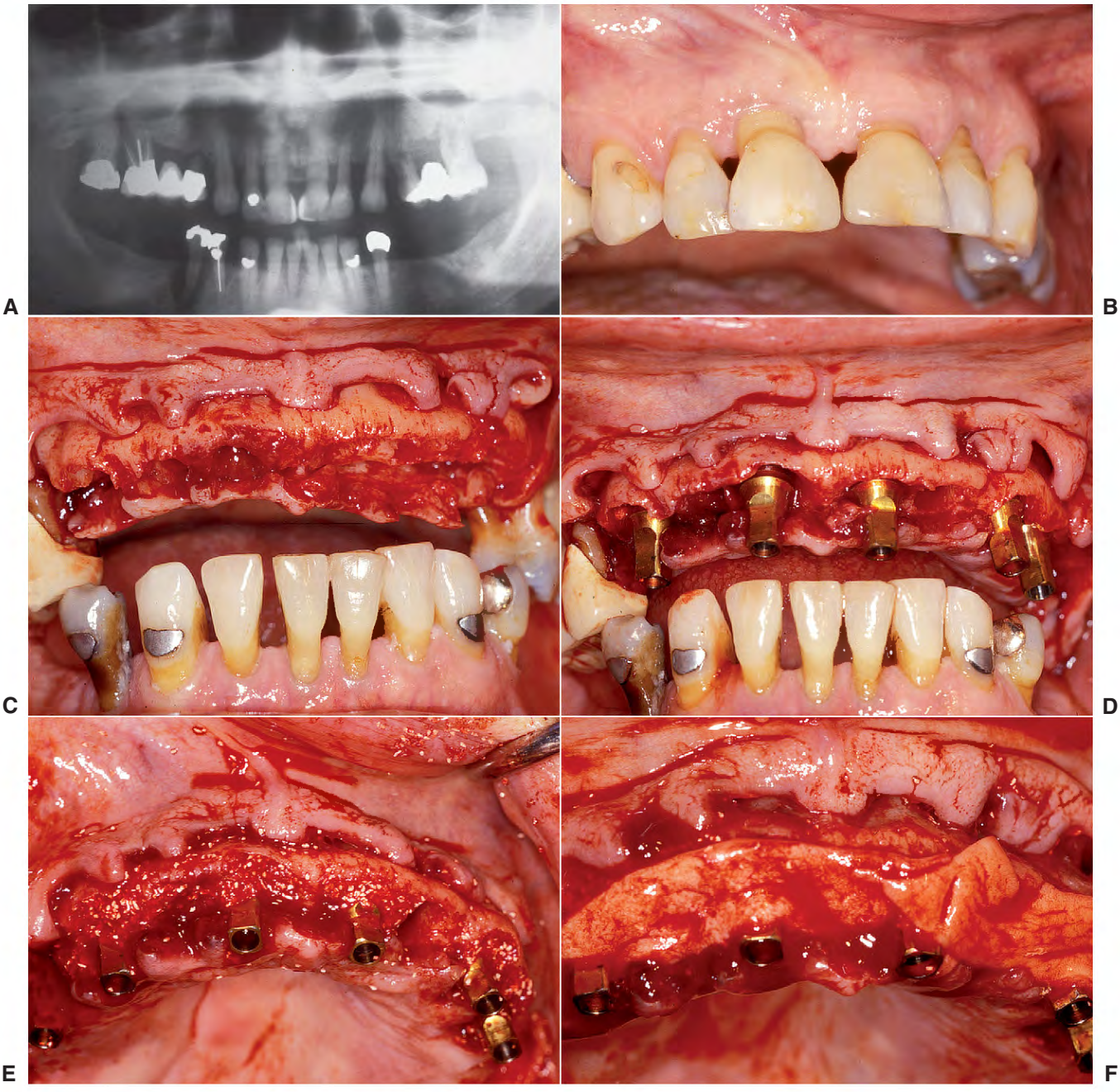


Figure 35-22 **A,** A panoramic radiograph of a maxillary arch with failing natural dentition. **B,** An intraoral view of the failing maxillary teeth. **C,** The six maxillary anterior teeth are extracted and the tissues reflected. **D,** Initially, five BioHorizons Maestro implants are inserted into the mandible. **E,** The rest of the maxillary teeth are removed and a total of six maxillary implants are inserted. A bone graft material is placed in the extraction sockets around the implants. Sinus graft is performed in the left maxilla. **F,** An acellular dermal matrix is placed around the implants to act as a barrier membrane.

Continued

30 N-cm or more. Abutment screw loosening of any individual implant may increase the risk of other implants in the prosthetic-implant system. The dentist prepares the final abutments intraorally for parallelism and proper height requirements. The dentist relines the transitional prosthesis with a light-cured composite (e.g., a urethane methacrylate–based product [Triad; Dentsply, York, Pa.) to eliminate toxic monomer contact with the bone. This material also exhibits less dimensional change compared with acrylic (4% versus 17%), and no heat generation occurs during setting (which might cause bone injury). In addition, the risk of "locking" the prosthesis into place onto unparallel implant abutments is less, because the restoration may be removed and reinserted several times during the initial light-cure process.

The dentist adjusts the temporary prosthesis to fit the abutment before approximation of the tissues. In this way the dentist may inspect the interproximal and margin region thoroughly before soft tissue closure.

WWW.HIGHDENT.IR

Figure 35-22, cont'd **G,** A transitional restoration is delivered and cemented with a definitive crown and bridge cement. **H,** A postoperative panoramic radiograph of the maxillary arch with six anterior implants (first premolar to first premolar) and a sinus graft in the left posterior region. **I,** A panoramic radiograph after the posterior left sinus graft and right socket graft have matured and four additional implants have been inserted. **J,** The maxillary implants after 8 months of immediate occlusal loading and removal of the transitional prosthesis removed. **K,** Ten maxillary implants exist in this arch (mirror image). More implants were inserted into the left sinus graft region, because the bone was less dense. **L,** A porcelain-metal fixed prosthesis is fabricated.

Continued

The dentist approximates the tissues before cementing the prosthesis (see Figure 35-22, *G* and *H*). The dentist places sutures more than 5 mm away from the incision line margin to facilitate their removal at a later date without removing the transitional prosthesis. The dentist evaluates the transitional restoration for harmonious occlusal contact in central occlusion. An implant-protective occlusal philosophy is advantageous in this restoration.[66] The dentist cements the immediate-load relined transitional prosthesis with a definitive cement such as zinc phosphate or glass ionomer. If the restoration becomes uncemented during the early loading process, then the risk of overload and failure or crestal bone loss increases.

Option 2

The second option for the immediate occlusal load process is to split the surgical appointment from the prosthesis delivery appointment. The first step of this option is similar to option 1. The preoperative appointments and implant position during surgery are the same. However, in option 2 the dentist makes an

WWW.HIGHDENT.IR

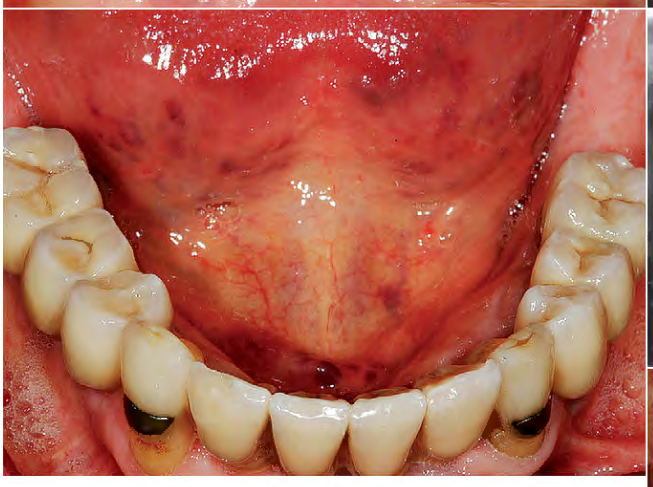


Figure 35-22, cont'd **M,** An occlusal view of the final porcelain-metal fixed restoration. **N,** An intraoral view of the maxillary fixed prosthesis. **O,** The maxillary fixed restoration in centric relation occlusion. **P,** A panoramic radiograph of the 10 implants and fixed prosthesis in the maxillary arch. **Q,** The mandibular arch was then implanted (immediate load) and finally restored. **R,** A panoramic radiograph of the immediate-loaded maxillary and mandibular fixed prosthesis. **S,** A high smile line of the restored maxillary and mandibular arches.

WWW.HIGHDENT.IR

impression of the implant body position with additional silicone. In addition, the dentist records a vertical occlusal dimension and centric bite registration. The bite registration may be made with the shell of the transitional restoration on the abutment or with a baseplate and wax rim (made before surgery and relined as necessary).

After the impression the dentist removes the abutments from the implant bodies and replaces them with permucosal extensions (PMEs). The dentist approximates the soft tissues similar to a one-stage surgical procedure.

The laboratory inserts the implant body analogs connected to the abutments into the impression, pours the impression with die stone, and mounts the cast to the opposing arch. The laboratory selects and prepares the abutments for the restoration and fabricates a transitional prosthesis.

The patient returns to the office 7 to 14 days after surgery. After removing the sutures, the dentist replaces the PMEs with the abutments that the laboratory selected and prepared. The dentist also delivers the transitional prosthesis and cements it with a definitive cement.

Diet

If the immediately loaded prosthesis becomes partially uncemented or fractures, the remaining implants holding the restoration are at increased risk of overload failure. Therefore the diet of the patient should be limited to only soft foods during the immediate-loading process. Pasta and fish are acceptable, whereas hard crust of bread, meat, and raw vegetables or fruits are contraindicated. In other words, the prosthesis and diet are similar to that for the first transitional restoration delivered in a progressive bone-loading approach.

Final Prosthesis

After healing 4 to 8 months (dependent on the bone density) the transitional restoration may be cut off and the final prosthesis fabricated (Figure 35-22, *I* to *S*).

Guidelines for Immediate Loading

Treatment plan guidelines for completely edentulous patients reflect methods to reduce stress and reduce microstrain at the developing interface.

Surface Area Factors

The dentist should consider the following surface area factors:

1. Implant number: Eight splinted implants or more are suggested for the completely edentulous maxillary arch and six splinted implants or more for the mandible—more implants if very soft bone (D4) is present or if force factors are greater (e.g., crown height or parafunction).
2. Implant size: Larger-diameter implants are required in the posterior regions of the mouth. If larger diameter is not possible, then bone grafting or greater implant number is suggested (e.g., two implants for each molar).
3. Implant design
   - High surface area implants (more threads, deeper threads)
   - Compressive versus shear loads (square- or plateau-shaped threads)
4. Implant surface condition:
   - HA-coated implants in poor bone density types (e.g., D4)
   - Rough versus smooth or machine surface condition implants in good bone density situations (e.g., D2 and D3)

Force Factors

The dentist should consider the following force factors:

1. Patient conditions: Parafunction, crown height, and muscular dynamics require more implant surface area. Severe parafunction may be a contraindication for the completely edentulous patient.
2. Implant position: In the completely edentulous maxilla, anterior implants should be at least in the bilateral canine position and posterior implants in the first- to second-molar position for the largest anteroposterior (A-P) dimension. When forces are greater, the dentist should insert an additional implant between the canines. In the mandible the largest A-P dimension possible should be used. At least three implants, one in the anterior and one in each posterior region, are necessary.
3. Occlusal load direction:
   - Narrow occlusal tables and no posterior offset loads on the transitional prosthesis
   - Long-axis loads to the implant bodies whenever possible

No posterior cantilevers should exist on transitional restorations in either arch. The cemented transitional restoration uses a definitive cement (e.g., zinc phosphate or glass ionomer cement) rather than a more temporary, weaker cement.

Implant Overdentures

The immediate-load concept for mandibular overdentures has been used in the literature for more than 50 years. The subperiosteal implant and the mandibular staple implant were loaded immediately after insertion.

WWW.HIGHDENT.IR

Babbush et al.[14] reported on immediately loaded overdentures in the early 1980s with threaded root form implants. More recently Chiapasco et al.[69] documented implant success rates of 88% to 97% over 5 to 13 years. The risk of joining implants together with a bar for an implant overdenture is less than for a fixed prosthesis, because the patient may remove the restoration at night to eliminate the risk of nocturnal parafunction. In addition, the overdenture may have some movement and load the soft tissue, which adds a stress relief system for the rigid implants.

The treatment plan for implant number and position for implant overdentures that are completely implant supported should be similar to a fixed restoration. If the prosthesis has no movement while in place, then it cannot gain support from the soft tissue. Although the restoration may be removed, it is completely implant supported during function or parafunction.

Implant overdentures with hard and soft tissue support may be at increased risk for immediate loading because the torque to the implants may be increased compared with completely implant-supported restorations. One should exercise care relative to the amount and direction of prosthesis movement during the initial loading period.

The immediately loaded overdenture procedure is similar to the second option with a fixed restoration. In other words, the dentist makes an implant body impression at the initial surgery. The position of the denture and teeth and the contours of the overdentures are important to know before fabrication of the bar and attachments that will connect the implants. One method to ascertain this information is to use a custom impression tray for the impression. The dentist makes a vacuum or press form with clear acrylic over the patient's denture before placing the implants. The dentist inserts the denture and press-form template into the mouth and makes a bite registration of the opposing arch. The dentist then removes the denture from the template, which then may act as a customized impression tray (Figure 35-23, *A* to *I*).

After inserting the implants, the dentist may place an indirect transfer coping into the implant bodies. The dentist places the customized impression tray over the implants, and the patient occludes into the attached bite registration, which helps seat the tray in the correct position (see Figure 35-23, *J*). The dentist then makes an impression, which because of the nature of fabrication also provides the vertical occlusal dimension, occlusal plane, position of the denture teeth, and contours of the overdenture (Figure 35-23, *K* and *L*). After making the impression, the dentist removes the indirect impression transfer copings and inserts the PMEs into the implant bodies. The dentist then may approximate the tissue with sutures in the usual fashion for a one-stage surgery (see Figure 35-23, *M* and *P*).

The bar and attachments are designed to be away from the denture teeth and contours of the restoration (see Figure 35-23, *Q* and *R*). When the crown height space is limited, the dentist fabricates a screw-retained bar directly to the implant bodies. When the crown height space is greater than 15 mm, the dentist most often designs a cemented bar on abutments greater than 6 mm in height. The dentist then delivers the bar for the implant overdentures at the suture removal appointment. The overdenture is hollowed out, and a soft conditioner is placed over the bar (see Figure 35-23, *S* to *U*).

The dentist asks the patient to leave the overdenture out of the mouth as much as possible. During sleeping hours, the patient does not wear the restoration to prevent nocturnal bruxism from overloading the developing bone interface. The diet is restricted to softer foods.

After 3 months or more, the final restoration may be fabricated (once the soft and hard tissues are mature). The previously constructed baseplate and wax rim make this portion of the overdenture fabrication simpler and more accurate (see Figure 35-23, *V* and *W*).

PARTIALLY EDENTULOUS PATIENTS

The immediate-load concept also may be used in the partially edentulous patient, including single-tooth applications. This concept has fewer clinical studies than full-arch cases, either fixed or removable. In 1998, Misch[70,71] published the first article during the "reinvention" of immediate "load" for partially edentulous patients. Most articles are case reports, especially for single-tooth applications. Rather than immediate loading of the implant, most reports suggest immediate restorations rather than full occlusal loading. Because the patient most often has enough remaining teeth in contact to function, the transitional restoration is primarily for esthetics, and the implant prosthesis is completely out of occlusion. Therefore a nonfunctional immediate teeth (N-FIT) concept is suggested (Box 35-1).

Box 35-1 Nonfunctional Immediate Teeth

Indications

- Partially edentulous patients with centric occlusal contacts and excursions on natural teeth (or healed implants)
- D1, D2, and D3 bone in regions of implants
- Screw-shaped implant bodies, 4 mm or more in diameter, with increased surface area designs to decrease crestal stresses (e.g., BioHorizons Maestro dental implant)

Contraindication

- Patients with parafunctional oral habits (i.e., anterior and lateral tongue thrust or biting on a pipe while smoking)

WWW.HIGHDENT.IR

The N-FIT concept presents a similar approach to the immediate-loading technique for the completely edentulous patient, with two major exceptions. Rather than submerge more than half the implants or place extra implants in case of failure, most often the ideal number of implants is positioned in the ideal locations for the final prosthesis. The second major difference is that the implant-supported transitional prosthesis is placed out of all direct opposing occlusal contacts during the bone-healing period. As a result, the dentist may fabricate an esthetic tooth replacement immediately for the patient but with no occlusal contact. The dentist also may develop the soft tissue contours with the transitional prosthesis during the bone-healing process and may evaluate them before making the final restoration.

Two clinical approaches to the N-FIT technique are similar to the fixed prosthesis for the edentulous patient. The first option is to use a surgical-prosthetic protocol similar to immediate loading with a diagnostic wax-up to fabricate the provisional restoration. Once the implants are inserted, the dentist recontours and relines the acrylic provisional prosthesis to the abutments (Figure 35-24). This alternative often is selected when the implant surgeon and restoring dentist are the same person or work in the same office.

A second alternative is to make an implant body impression with abutments or transfer copings that engage the antirotational hexagon (Figure 35-25, *A* to *C*). The dentist also makes a centric bite registration and opposing arch impression. The dentist then removes the abutments, places them into the impression with

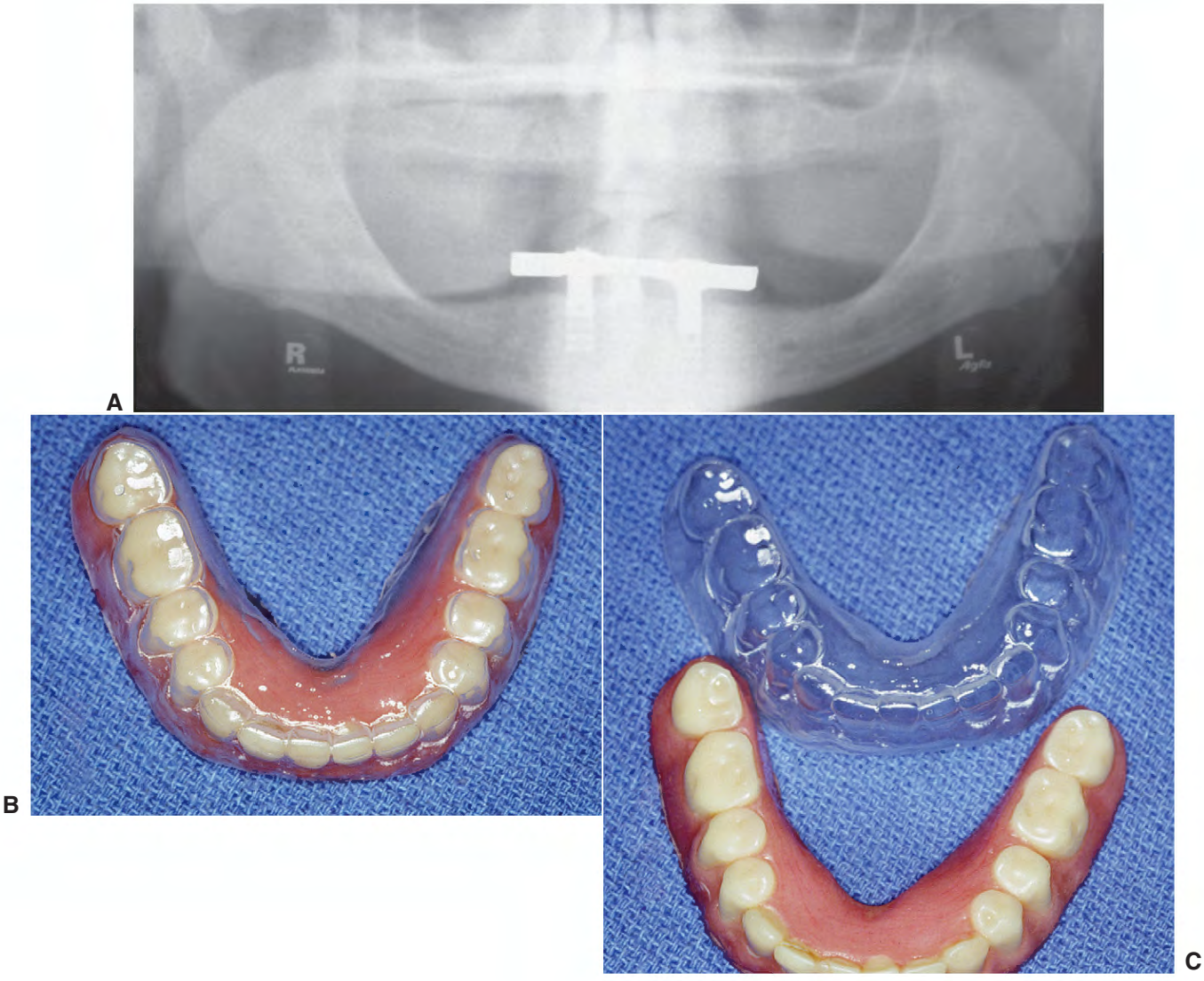


Figure 35-23 **A,** A panoramic radiograph of a failing mandibular implant overdenture. The patient's leftmost posterior implant has lost more than 50% of the surrounding bone. The patient also desires more support and stability of the overdenture. **B,** A press-form acrylic template is made over the patient's existing denture. **C,** The press-form acrylic template replicates the teeth and contours of the restoration. *Continued*

WWW.HIGHDENT.IR

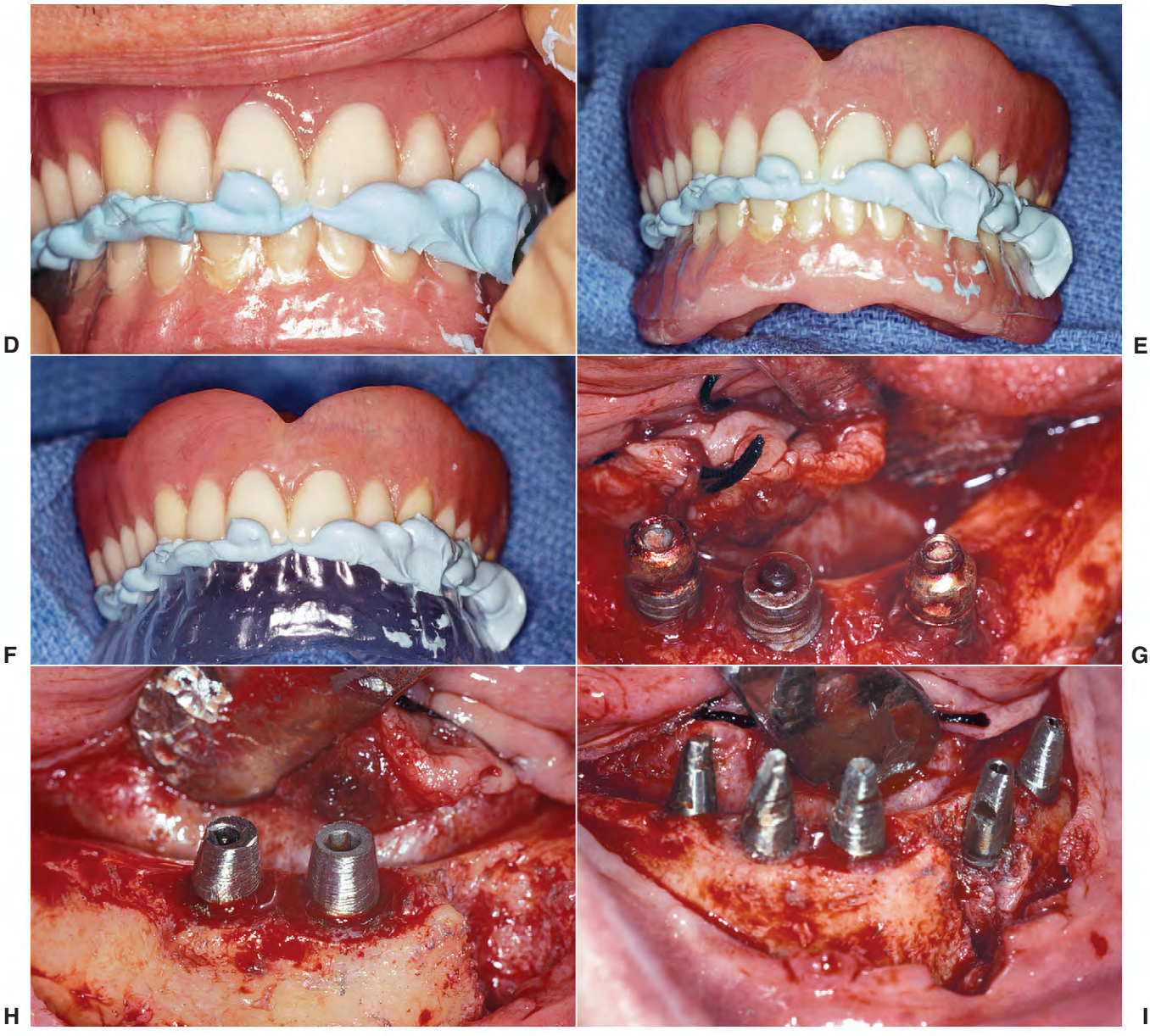


Figure 35-23, cont'd **D,** The press form is positioned over the denture, and a bite registration is made with the opposing arch at the ideal vertical occlusal dimension. **E,** The maxillary denture, mandibular overdenture, and press-form acrylic template are removed from the mouth. **F,** The press-form template is now a customized impression tray that one may use as a surgical guide for the implants and an impression tray for the overdenture bar. **G,** The tissue is reflected to allow evaluation of the implant on the far right. **H,** The implant on the far right was removed, along with the fibrous tissue. The remaining two implants are prepared for a cemented bar. **I,** Three additional implants are positioned around the original two implants. The abutments are prepared for a cemented bar.

Continued

implant body analogs, and places the permucosal healing abutments into the implant bodies before suturing (Figure 35-25, *D* and *E*; Box 35-2). This appointment is similar to a nonsubmerged implant technique, with the addition of impressions for the fabrication of a transitional prosthesis.

The laboratory fabricates working models from the impressions and mounts them with the centric registration (Figure 35-25, *E* and *F*). The laboratory may prepare the two-piece abutments for cement retention and fabricate two transitional prostheses. The first transitional prosthesis is completely out of occlusion 1 to 2 mm in centric occlusion and all excursions. The laboratory also may fabricate a second transitional restoration in occlusion.

At the suture removal appointment, the dentist removes the permucosal abutments and replaces them with the appropriate abutments for cement retention (Figure 35-25, *G*). The dentist delivers the transitional prosthesis with no occlusal contact and modifies the esthetics as required. This option is easier to use in the team approach because the restoring dentist does

WWW.HIGHDENT.IR

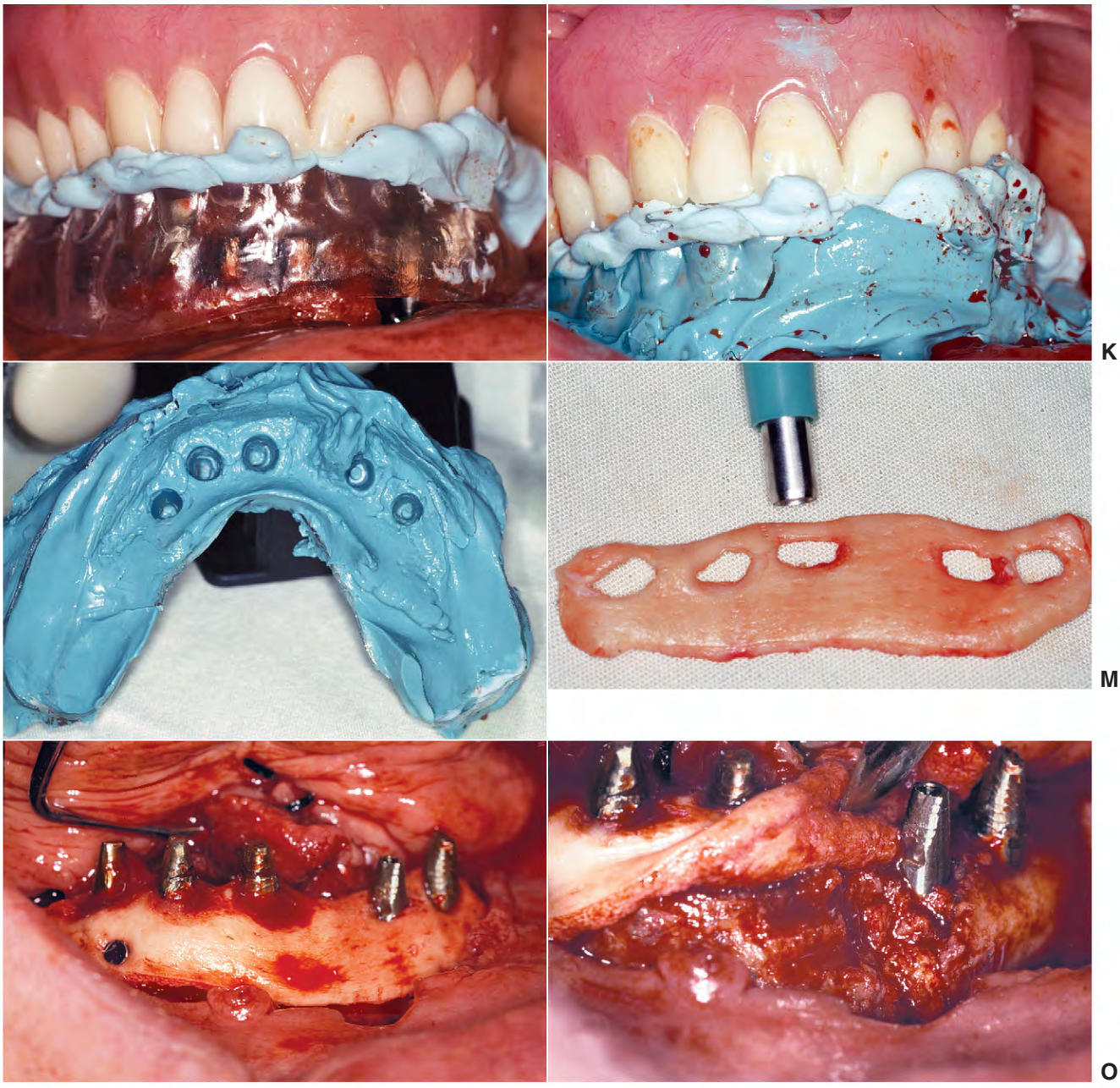


Figure 35-23, cont'd **J,** The customized impression tray is positioned with the maxillary denture. The tray is used as a surgical guide and an impression tray. **K,** A final impression is made of the implant abutments at the approximate vertical dimension of occlusion. **L,** The customized impression tray records the implant positions relative to the final contours of the overdenture prosthesis. **M,** An acellular dermal graft is prepared with a tissue punch. **N,** The membrane is positioned over the implants and tacked into position. **O,** The membrane is lifted over the failed implant site, and autologous bone is positioned over the implant. The AlloDerm may be used as a barrier membrane for bone regeneration. *Continued*

not need to be present at the surgery. In addition, the laboratory can prepare the abutments or select an angled or custom abutment to correct nonparallel implant placement.

After the appropriate bone-remodeling period (3 to 8 months, depending on the bone density), the dentist removes the first transitional restoration, evaluates the implants, and inserts the second transitional acrylic restoration in light occlusion with a heavy bite force occlusal adjustment. This procedure allows progressive loading of the bone-implant interface and increases the bone density next to the implant[66] (Figure 35-25, *H* and *I*).

The advantages of the N-FIT concept include all those of the nonsubmerged approach with these added advantages (Box 35-3):

1. An esthetic restoration replaces the missing teeth, rather than exposed titanium.
2. The implants are splinted together, rather than healing as independent units. This is a considerable

WWW.HIGHDENT.IR

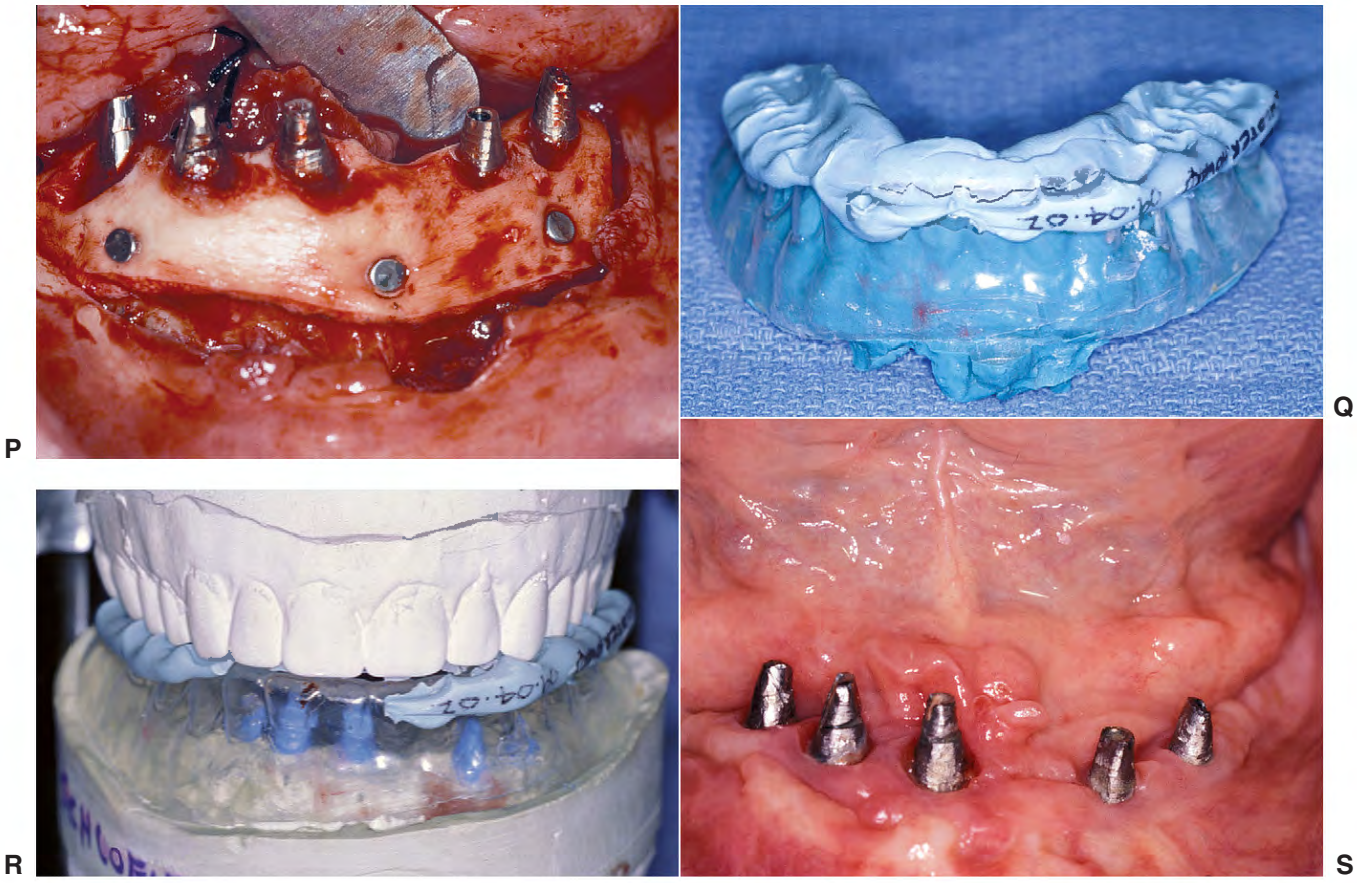


Figure 35-23, cont'd **P,** The membrane is tacked into position around the implants. The nonmobile membrane will bind to the overlying tissues and form nonmobile tissue around the anterior implants. **Q,** The laboratory pours the final impression within the customized impression tray. **R,** The laboratory mounts the cast to the opposing arch. The impression material is removed from the customized impression tray and indicates the teeth and contours of the final restoration. The overdenture bar is waxed within these contours. **S,** The patient is seen within 2 weeks of the initial implant insertion surgery.

Continued

biomechanical advantage to distribute stress and decrease crestal loads.

3. The emergence profile of the final restoration may be created with the transitional prosthesis. Interdental papillae are developed and maintained throughout the healing period.
4. The soft tissue is mature when the final restoration is fabricated because the tissue has been healing for many months. If surgical correction is required, then the dentist still may perform the correction before the final bone maturation.

The primary advantage of the N-FIT concept compared with immediate loading is the decreased risk of biomechanical overload from parafunction. The dentist encourages the patient to maintain a soft-food diet (e.g., pasta, fish) during the healing period and to avoid the implant sites. However, the bite forces during eating are less than 30 psi and are limited to less than 30 minutes per day.[72] A major concern for the immediate implant–loading concept is parafunction, which can generate 900 psi several hours a day and night. The N-FIT approach dramatically reduces the risk of parafunction. This concept even can be used for single-tooth replacement. However, if the region is out of the esthetic zone, then a more conventional approach is suggested (one stage or two stage).

The primary disadvantage of the N-FIT concept is the increased risk of biomechanical overload compared with the submerged, countersunk approach (Box 35-4). Because the surgeon does not reflect the tissue at a second-stage surgery, a direct observation of the bone is not possible to evaluate facial or lingual bone loss. In addition, impression material or acrylic may be trapped under the soft tissue. However, the decreased crestal bone loss from an implant that is not countersunk below the bone and the immediate esthetic advantages often outweigh the risk.

SINGLE-TOOTH NONFUCTIONAL IMMEDIATE-RESTORATION PROCEDURES

Worhle[73] and Misch[70,71] presented the single-tooth immediate restoration in 1998. The single-tooth implant

WWW.HIGHDENT.IR

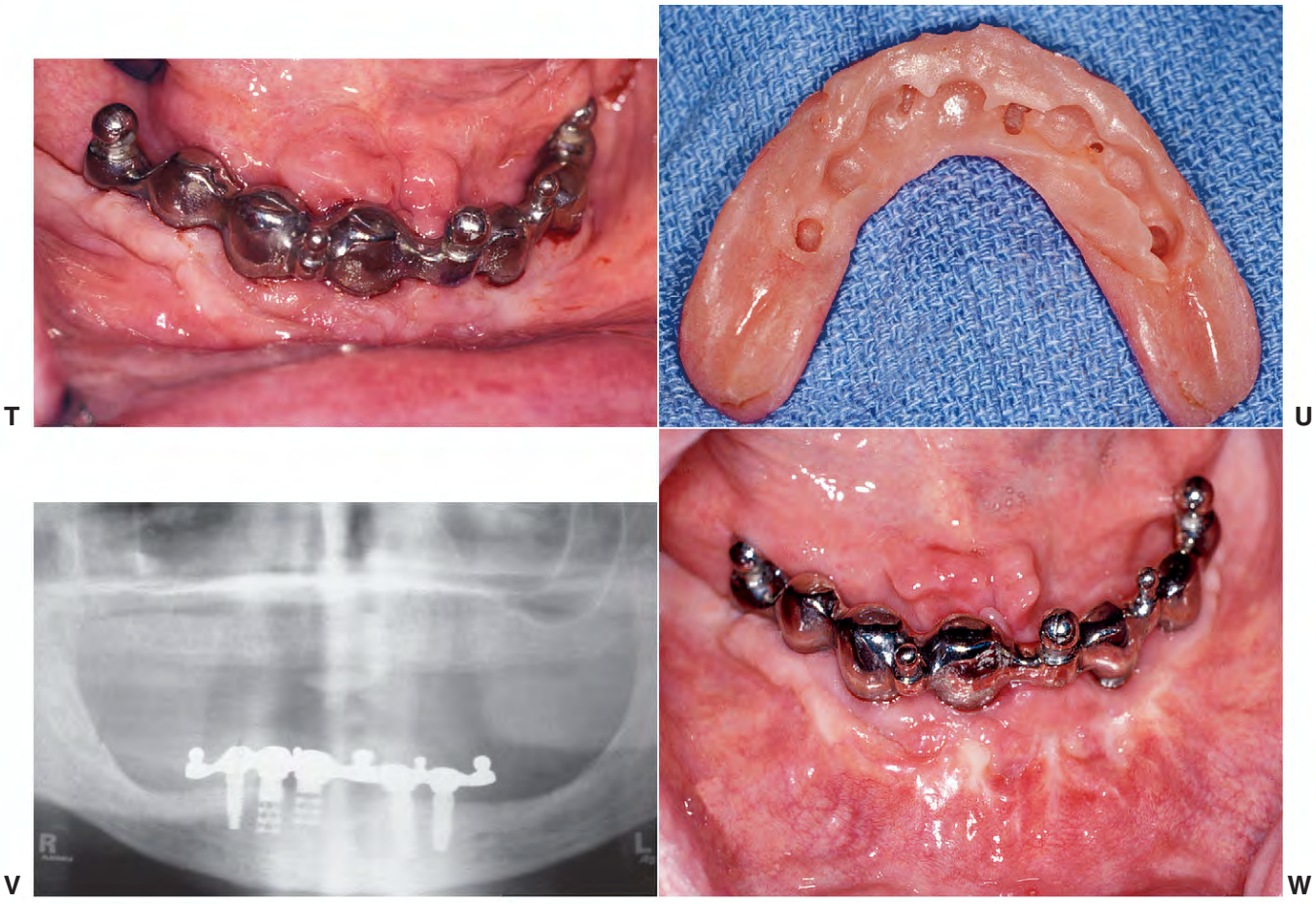


Figure 35-23, cont'd **T,** The bar is delivered to the patient within 2 weeks of surgery. **U,** The existing denture is hollowed out and relined over the overdenture bar. The patient is instructed to leave the prosthesis out of the mouth at night and to wear the prosthesis as little as possible. **V,** A panoramic radiograph of the immediately loaded overdenture bar and implants. **W,** The overdenture bar and implants 4 months after immediate

is considered for immediate restoration most often when the following occur:

1. The natural tooth requires extraction and is still present in the mouth.
2. The natural tooth is in the esthetic zone.
3. The soft tissue drape in its current form is ideal.
4. The bony housing around the natural tooth is intact, including the facial plate.

In Chapter 33, the concept of natural-tooth extraction and implant insertion is presented. This chapter presents the concept with the addition of immediate restoration.

The natural tooth is atraumatically extracted. The implant osteotomy is prepared to the opposing landmark, because additional implant length is a benefit for N-FIT. The bone filings are harvested from the drill. A bone tap is used so that the implant will not be pushed toward the facial during insertion. The bone filings are placed on the coronal third of the socket, especially on the facial aspect. The implant is threaded into position under the incisal edge of the future crown and 3 mm below the future facial free gingival margin of the implant crown.

An immediate-load restoration implant design may be one piece with no separate abutment screw. The advantage of this design is increased strength (for smaller-diameter implants) and elimination of the risk of abutment screw loosening. When a two-piece implant design is used for immediate-load restoration, tighten the abutment screw with 30 N-cm of torque and follow the prosthetic protocol (usually used several months later).

Transitional Restoration

Two options exist for the transitional restoration for a single-tooth immediate implant restoration. The first option is an acrylic crown. However, this crown has no occlusal load for 3 to 4 months. In addition, the transitional crown may be splinted to a natural tooth that has no clinical mobility (i.e., a canine). The diet should be restricted to only soft food, and the patient is told to avoid this restoration as much as possible during the initial healing period. After the initial healing, the

WWW.HIGHDENT.IR

Box 35-2 Protocol for Stage I Nonfunctional Immediate Teeth

Appointment One

1. Make impression of opposing arch and take tooth shade and centric bite registration.
2. Perform stage I implant surgery (use wider implants when possible).
3. Remove fixation screw and insert ball impression pin.
4. Make impression with additional silicone material.
5. Make sure no impression material is left under flap or around implants.
6. Remove ball impression pin and abutment for cement.
7. Place 3- to 6-mm-wide emergence permucosal healing cap.
8. Suture (tissue thickness should be less than 4 mm).

Laboratory Procedure

1. Thread ball impression pin and abutment for cement into implant body analog and reinsert into impression.
2. Pour impressions of both arches and mount on articulator.
3. Prepare abutments, if needed. (NOTE: Hexagon transfer locations are made with ball and abutment for cement transfer.)
4. Fabricate transitional prosthesis:
   a. Soft tissue emergence similar to final restoration
   b. Narrow occlusal table
   c. Transitional prosthesis 1 to 3 mm out of all occlusal contacts (centric relation and excursions)

10 to 14 Days after Stage I Surgery

1. Remove sutures.
2. Remove permucosal healing caps.
3. Insert two-piece abutment and abutment screw.
4. Use countertorque (hemostat) and tighten to 20 to 30 N-cm (which is less than final preload).
5. Insert transitional prosthesis and evaluate contour and occlusion (no occlusal contacts).
6. Cement transitional prosthesis with temporary definitive cement.
7. Instruct patient to eat soft foods (e.g., pasta, fish, cooked meat). No raw vegetables or hard bread are allowed until final prosthesis delivery. No oral habits, such as gum chewing, are permitted. When possible, the patient should avoid chewing food in implant regions.

Box 35-3 Advantages of Nonfunctional Immediate Teeth

- Patient has a fixed esthetic tooth replacement soon after stage I surgery.
- No stage II surgery is necessary (eliminates discomfort for the patient and decreases overhead for the doctor).
- Implants are splinted during initial healing for biomechanical advantage.
- The greatest bite force is only during eating and is less than 30 psi. No parafunctional forces from occlusion are possible.
- Countersinking the implant below the crestal bone is eliminated, which reduces early crestal bone loss.
- The soft tissue emergence may be developed with the transitional prosthesis and the tissue allowed to mature during the bone-healing process.
- The soft tissue hemidesmosome attachment on the implant body below the microgap connection may heal with an improved interface.

Box 35-4 Disadvantages of Nonfunctional Immediate Teeth

- Micromovement of implant that can cause crestal bone loss or implant failure is greater than with two-stage approach.
- The dentist is less likely to reflect the tissue at stage II and can evaluate implant crestal bone directly.
- Parafunction from tongue or foreign habits (pen biting) may cause trauma and crestal bone loss or implant failure.
- Impression material or acrylic may become trapped under tissue or between the implant and crestal bone. This problem is reduced greatly if the crest module of the implant is larger in diameter than the implant body.
- Bone that is too soft, small implant diameters, or implant designs with less surface area may cause too great crestal stress contours and cause bone loss or implant failure.

final restoration may be fabricated and the diet returned to normal.

The second option also uses a premade crown. The crown is modified to fit over the abutment and places the gingival margin in close approximation to the tissue. The premade crown is not relined with acrylic. A hole is then made in the mesial and distal interproximal surfaces of the crown. The adjacent teeth are acid

WWW.HIGHDENT.IR

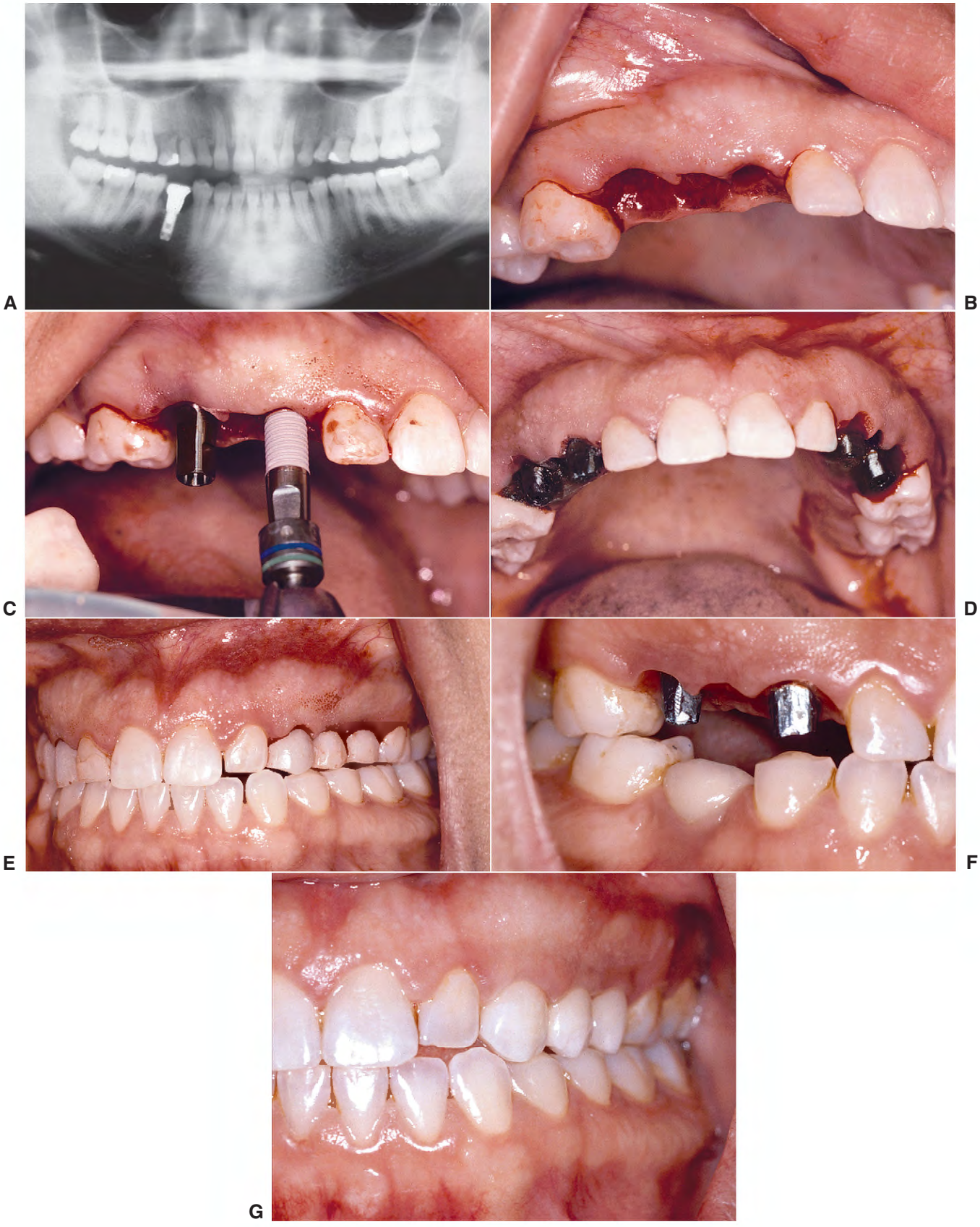


Figure 35-24 **A,** A panoramic radiograph of a patient with partial anodontia missing the bilateral permanent canines, first premolar, and second premolars. **B,** The deciduous teeth have been extracted. **C,** Two implants are used to support the prosthesis on each side. The mesiodistal space is inadequate for three implants. **D,** The four implants are prepared for a cemented transitional prosthesis. **E,** The transitional N-FIT restorations are primarily for esthetics and are completely out of occlusion in centric relation and all excursions. **F,** The final restoration is made after 4 to 6 months. At this point, the soft and hard tissues are mature. **G,** The final restoration of the three-unit fixed partial denture supported by two immediately loaded implants.

WWW.HIGHDENT.IR

etched, and composite resin is placed in the interproximal regions of the crown to lute it to the adjacent teeth. The occlusion is modified to eliminate occlusal contact. This approach provides an esthetic fixed replacement for the missing tooth, without the excess force on the implant. This approach will offer slightly less risk to the implant during initial healing, because the crown is not actually attached to the implant, and tooth contact will not overload the implant restoration. The diet is also restricted with this technique to soft foods for the initial bone-implant healing period. After 3 to 4 months the transitional crown is removed and a final restoration fabricated. Occlusal equilibration is performed to reduce the occlusal load. A periapical radiograph is taken to confirm the position of the crown and implant.

The most common congenital missing maxillary anterior tooth is a lateral incisor.[74] The restoration of one maxillary anterior crown is one of the more difficult prosthetic treatments in a general practice. A single-tooth implant is often the treatment of choice to replace

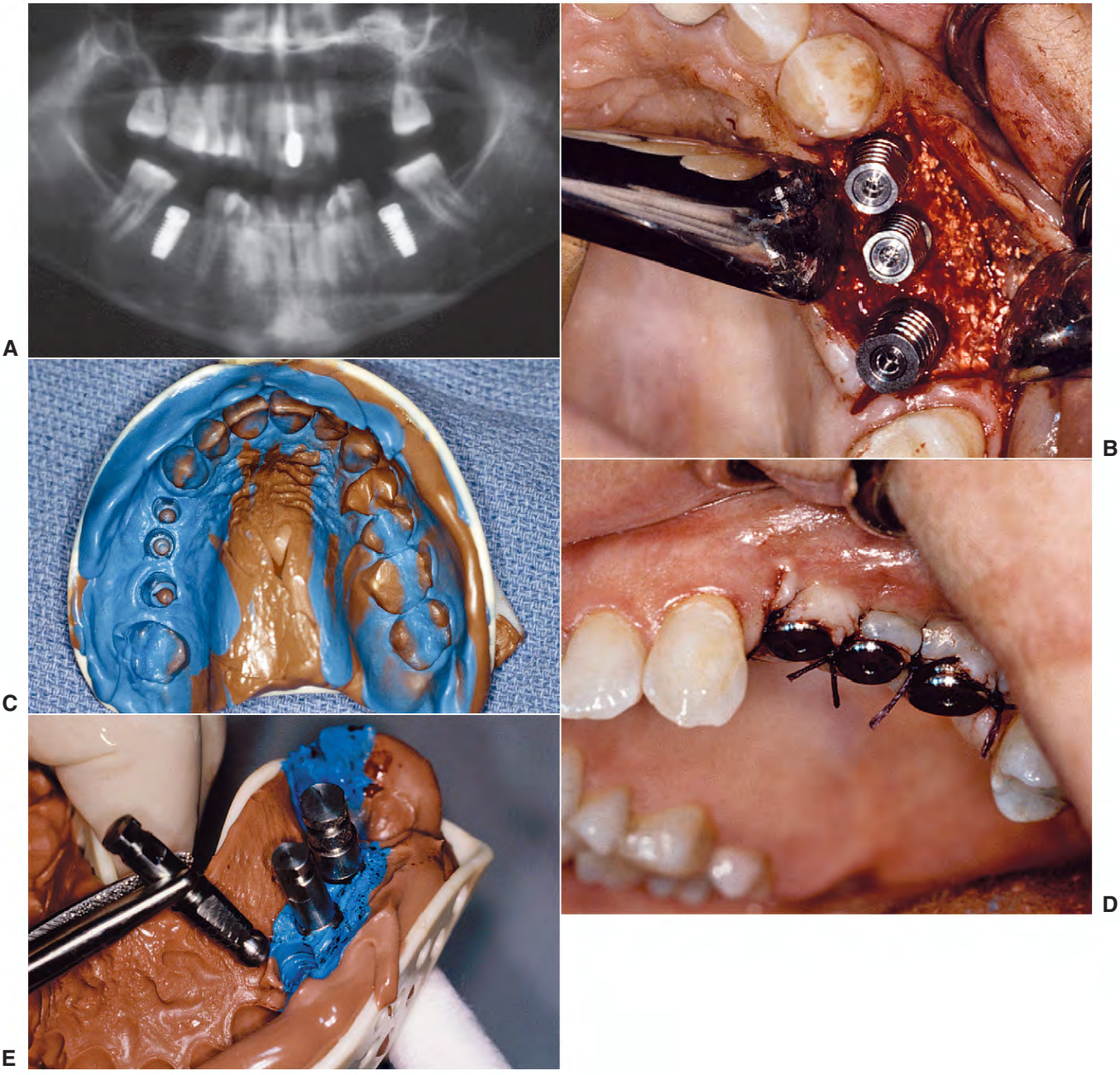


Figure 35-25 **A,** A panoramic radiograph of a partially edentulous maxilla and mandible. **B,** Three implants are inserted. The premolar implants are 4-mm-diameter BioHorizons Maestro D3 implants, and the molar is a 5-mm diameter BioHorizons Maestro D4 implant. **C,** An impression is made at surgery with an indirect impression transfer that engages the hexagon position of the implant body. **D,** Permucosal extensions (PMEs) are inserted into the implants, and the tissues are approximated, similarly to a one-stage surgical approach. **E,** The indirect impression transfers are connected to the implant body analogs and reinserted into the impression.

Continued

WWW.HIGHDENT.IR

Figure 35-25, cont'd **F,** The laboratory pours and mounts the impression made at surgery. The laboratory may prepare the abutments and fabricate a transitional prosthesis out of occlusion. **G,** At the suture removal appointment, the PMEs are removed. The abutments prepared by the laboratory are inserted, and the transitional prosthesis is delivered. The N-FIT is completely out of occlusion in centric relation and all excursions. **H,** The final restoration is made after 4 to 6 months. **I,** The panoramic radiograph demonstrates the final restorations in place. The maxillary restorations used an N-FIT approach. The mandibular restorations, which were out of the esthetic zone, used a traditional two-stage approach.

a congenitally missing lateral incisor. The implant and crown have the highest success rate of any treatment option, the adjacent teeth are usually unaffected, and the esthetic result is often ideal.[75] The following discussion presents the indications for a small-diameter implant and presents a case report of a patient missing one maxillary lateral incisor.

The average lateral incisor is 6.5 mm in width, 1 mm less than the canine, and 2 mm less than the central incisor.[76] When a permanent lateral incisor is missing, the deciduous tooth is retained longer than ideal, and the collapse of the permanent incisor mesiodistal space occurs as the canine positions itself in a more mesial position. In addition, when a lateral incisor is congenitally missing, the contralateral incisor is often peg-shaped or deficient in mesiodistal size.[74] This condition further reduces the space dimension. As a consequence, the missing lateral incisor space is often less than average and approaches 5 mm. Mandibular central and lateral incisors are missing less often but have a smaller mesiodistal dimension and average 5.4 mm.[76] Therefore single-tooth replacement may on occasion require implants of small diameter to restore the missing tooth situation.

Most manufacturers fabricate their smallest two-piece implants to a dimension of 3.5 mm or larger at the crest module, although the implant body may be smaller (e.g., 3.2 mm). A two-piece implant should have the microgap of the abutment at least 1.5 mm from the adjacent tooth.[77] As a result, the ideal minimum intertooth space for a dental implant with this dimension should be 6.5 mm (1.5 mm from each tooth and 3.5 mm for the top of the implant). This dimension is sufficient for the average dimension of a maxillary lateral incisor. Mandibular incisors are usually 5.2 to 5.4 mm in width, and congenitally missing maxillary lateral incisors are often less than 6.0 mm. As a result, most current implant designs are too large for smaller intratooth spaces.

A one-piece implant with a 3-mm-diameter was developed to overcome the challenge of small edentulous spaces in the anterior region of the mouth

WWW.HIGHDENT.IR

Figure 35-26 A one-piece implant may be positioned within 1 mm of an adjacent tooth with little risk of marginal bone loss.

(BioHorizons Maximus dental implants) (Figure 35-26). A one-piece implant does not have a microgap between the implant body and abutment connection, and therefore initial crestal bone loss over time may be reduced.[78] When the implant is not expected to lose proximal bone when positioned at the height of the crestal bone, the implant may be placed as close as 1 mm to the adjacent tooth root. Therefore mesiodistal spaces for a 3-mm-diameter implant used here may be as little as 5 mm. The primary indications for an implant of this dimension is for single maxillary lateral incisors and mandibular anterior lateral and/or central incisors.

Surgical Procedure

The surgical procedure for the 3-mm-diameter implant follows a similar protocol as other implants. A mucoperiosteal flap may be reflected, and direct observation of the bone can be made when the available bone is in question. However, when abundant keratinized tissue and bone are present, a tissue punch and implant osteotomy without tissue reflection is often the surgical protocol of choice. This was the surgical method used in this case report. An esthetic crown-lengthening procedure was also performed on the maxillary canines.

A 3-mm-diameter trephine bur was used to penetrate the soft tissue in the missing lateral incisor region (Figure 35-27, *A* and *B*). The soft tissue emergence was then contoured with a high-speed hand piece and coarse diamond so that the soft tissue profile was similar to the contralateral tooth.

An alignment drill was then used to initially prepare the implant site and begin to develop the path of insertion for the implant drills (Figure 35-27, *C*). This drill was also designed to level the crest of the ridge 3 mm below the free gingival margin of the implant crown and allow the abutment head of the implant to be level with the bone. A radiograph was taken with the alignment drill in place to evaluate the path of insertion (Figure 35-27, *D*).

A trial implant was then placed into the initial osteotomy site created by the alignment drill. The top of this device is the same size as the final implant abutment. The esthetic position and interocclusal clearance may be determined with this trial abutment. A periapical radiograph may also be taken of the trial implant to confirm the mesiodistal position and angulation. The position and/or angulation of the initial site may be corrected with the side-cutting feature of the alignment drill.

The depth of the osteotomy was established using a 2.0-mm-diameter depth drill of 12, 15, or 18 mm. A 15-mm drill was used in this patient (Figure 35-27, *E*). The longer the implant, the greater the initial stability. In addition, if the opposing dense cortical plate can be engaged, then a further benefit of rigid fixation occurs (Figure 35-27, *F*). A radiograph may be used to confirm the proper drill length and position (Figure 35-27, *G*). The osteotomy can be widened to 2.5 mm using the finishing drill when the bone is of a dense quality. Use of the final drill is not necessary in softer bone types, because the implant will condense the bone during insertion and provide greater fixation. A bone tap may be used when the bone is very dense (i.e., as is occasionally found in the anterior mandible).

The one-piece implant body-abutment was then inserted with a hand piece mount at 30 rpm (a hand wrench insertion with a ratchet adapter may also be used) (Figure 35-27, *H*). The implant was positioned so that the threads were 1 to 2 mm below the crest of the bone (Figure 35-27, *I*). A periapical radiograph confirmed the position.

A No. 702L preparation bur was used to modify the abutment as required, with consideration of the opposing teeth in occlusion. A transitional crown, without any occlusal contact, was then fabricated for the implant. The one-piece, small-diameter implant may use the same two options as previously presented for the single-tooth immediate-restoration implant (Figure 35-27, *J* to *L*). After a 4- to 6-month period, the transitional crown may be replaced with a final crown and regular implant-protective occlusion (Figure 35-27, *M*).

Limitations

Implants with smaller diameters have several limitations including less surface area, lower fatigue strength, and higher risk of screw loosening. Smaller-diameter implants have a smaller surface area for bone-implant contact, which could reduce the long-term survival of the fixture. The surface area of an implant is related to the amount of force the implant is able to resist when serving as a prosthetic abutment. The roots of posterior natural teeth have greater surface area than anterior teeth, and forces are greater on posterior teeth. Likewise, an implant with greater surface area is less likely to be overloaded during function.[78] A 1-mm decrease in width of an implant may decrease the surface area of

WWW.HIGHDENT.IR

an implant by more than 40%.[79] Therefore a 3-mm-diameter implant may have almost one-third less surface area of contact with bone as compared with a 4-mm-diameter implant.

The fatigue strength of an implant is affected by the diameter, the implant material, and the amount of force applied to the system.[80] The formula for the fracture strength of a circular implant is $\pi/4(R4)$. This means that a unit decrease in width decreases the strength of the implant by a factor of 4. For example, a 2-mm-diameter implant is 16 times weaker than a 4-mm-diameter implant. For this reason, clinicians may use a 2-mm-diameter transitional implant, but regular occlusal loads over an extended period of time would result in an unstable situation.

The fracture of the implant is also related to the metal of the implant. Regarding the materials from which implants are fabricated, the most common implant body is fabricated from titanium, because a direct bone-implant interface has been shown to develop.[81] Five grades of titanium are used for implants.[82] Grade 1 to 4 is 99% titanium, and grade 5 is titanium alloy (90% titanium, 6% vanadium, and 4% aluminum). The strength of each of these materials is different. Grade 1 titanium is four times weaker than grade 5 titanium. Although a few manufacturers have used this grade for 4-mm-diameter implants, it is inappropriate for use in a permanent small-diameter implant. Some manufacturers of transitional implants select a lower-grade titanium so that the clinician can bend the abutment post for parallelism. Several implant companies use softer grade 3 titanium for their implants, yet grade 3 is two times weaker than grade 5. Further, grade 4 titanium is 1.6 times weaker than grade 5. Thus when small-diameter implants are used to permanently replace a natural tooth, a grade 5 implant material should be used. The BIC is similar for all grades of titanium, because a similar oxide layer is formed regardless of the titanium grade.[82]

The fracture of an implant is also directly related to the amount of force placed on the implant component or body. Greater force is more likely to fracture an implant than lesser force. The maximum bite forces in the mouth are less in the anterior regions (25 to

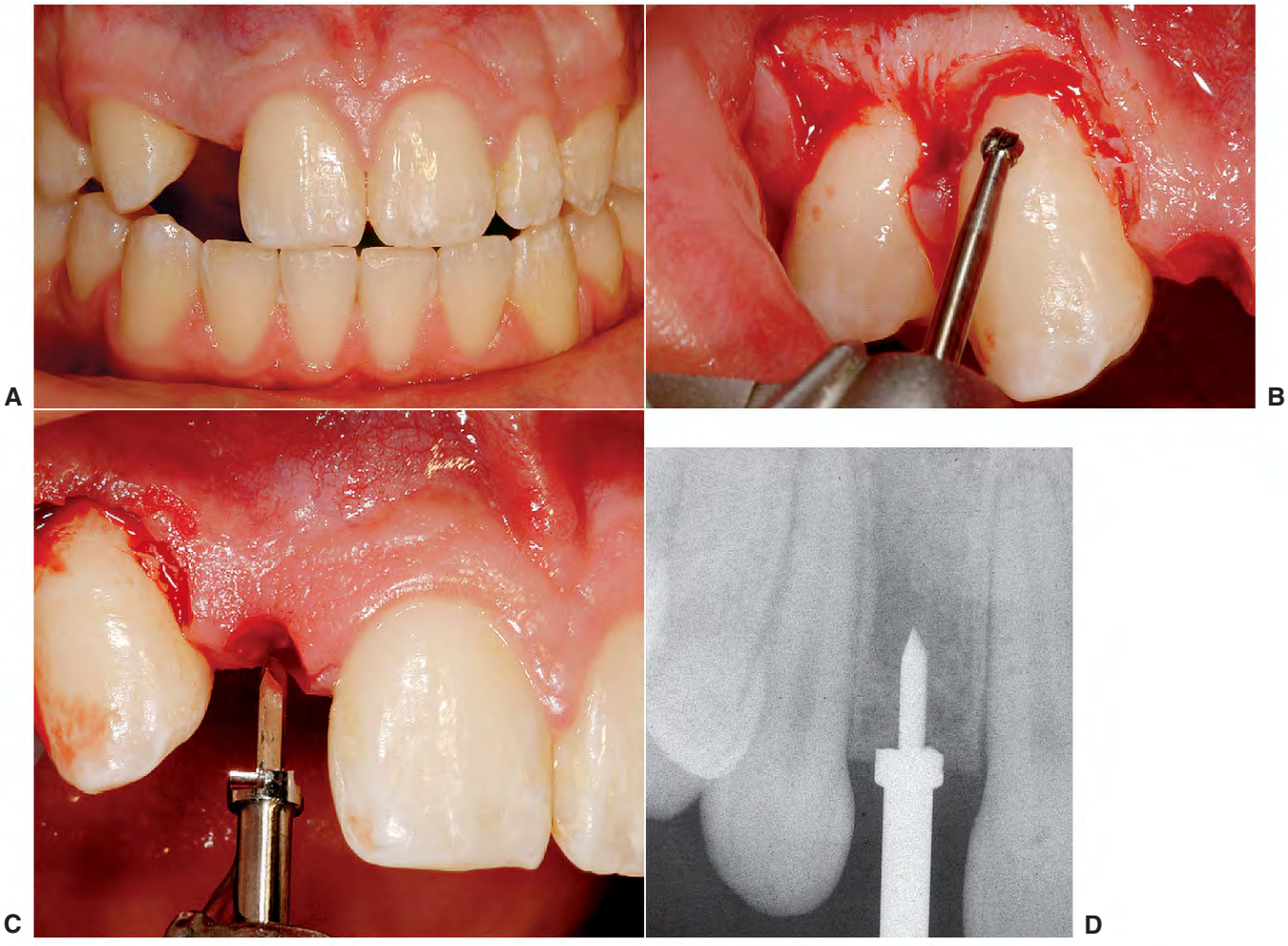


Figure 35-27 **A,** A patient with a congenitally missing right lateral incisor. The right canine would benefit from esthetic crown lengthening. **B,** Esthetic crown lengthening is performed on the canine. **C,** A pilot drill prepares the initial site of the lateral incisor. **D,** A radiograph confirms the position of the pilot drill and the adjacent roots.

Continued

WWW.HIGHDENT.IR

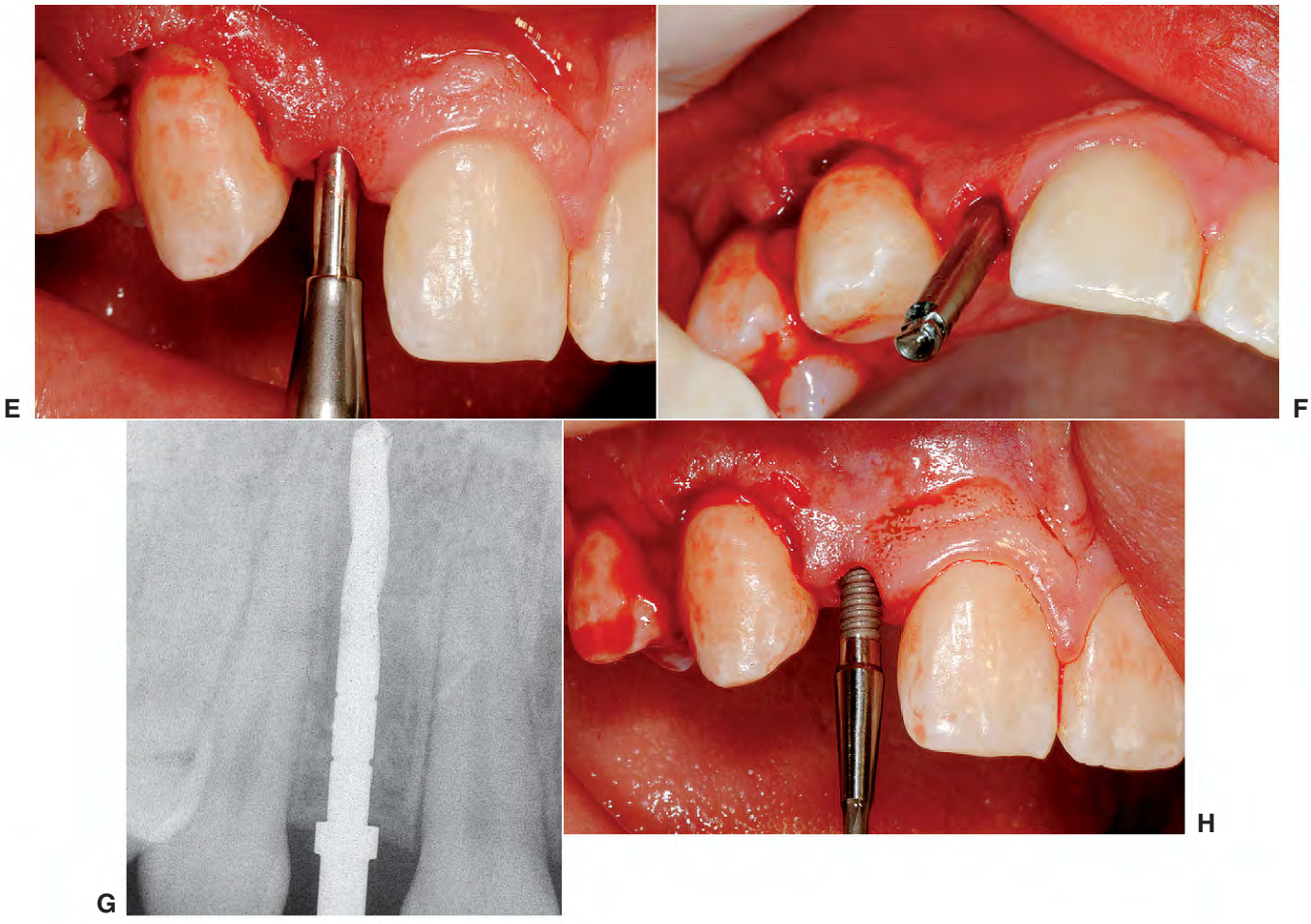


Figure 35-27, cont'd **E,** A 2-mm twist drill is used to prepare the implant osteotomy. **F,** The drill position is under the incisal edge of the future implant crown and proceeds to the opposing cortical plate (when possible). **G,** A radiograph evaluates the position of the drill. **H,** A small-diameter implant is threaded into position (BioHorizons Maximus, Birmingham, Ala.). *Continued*

50 lb/in^2) compared with the molar regions (200 to 250 lb/in^2).[83] Consequently, smaller-diameter implants should be limited to the anterior regions of the mouth to reduce the occurrence of fracture.

The prosthetic platform of a two-stage small-diameter implant is more likely to have screw loosening.[84] The narrower the abutment to implant attachment diameter, the more force applied to the abutment screw during occlusal loading. When the abutment screw becomes loose under a cemented crown, the crown may need to be cut off to gain access to the abutment screw. Abutment screw loosening is the most common prosthetic complication of single-tooth implants and has been reported to occur in 7% to 40% of cases (depending on patient factors and the implant system used).[85] A one-piece implant body and abutment has a distinct advantage because abutment screw loosening does not occur.

The one-piece small-diameter implant has several advantages when used to replace maxillary lateral incisors and mandibular incisors. The one-piece design eliminates the risk of abutment screw loosening. Because no microgap exists between the abutment and implant, the amount of crestal bone loss may also be reduced.[78] The abutment-implant connection of two-piece implants is often at or below the crestal bone. With traditional implant designs, bone loss of as much as 3 mm from the microgap has been reported.[77]

The primary disadvantage for a one-piece small-diameter implant is the requirement of immediate restoration. Because the implant abutment is intraoral at the time of surgical placement (the implant body and abutment are a single component), an increased risk of overload is present during initial bone healing. Oral habits or activities such as gum chewing, tongue thrusting and playing some musical instruments (i.e., woodwinds) may overload the developing interface. The "open" transitional crown concept, whereby the premade crown is not relined with acrylic, reduces this risk.

Traditional implant sizes of 3.5 mm and greater at the crest module are often too large to replace a missing tooth in the anterior regions of the jaws. On the other hand, temporary implants of less than 3.0-mm diameter

WWW.HIGHDENT.IR

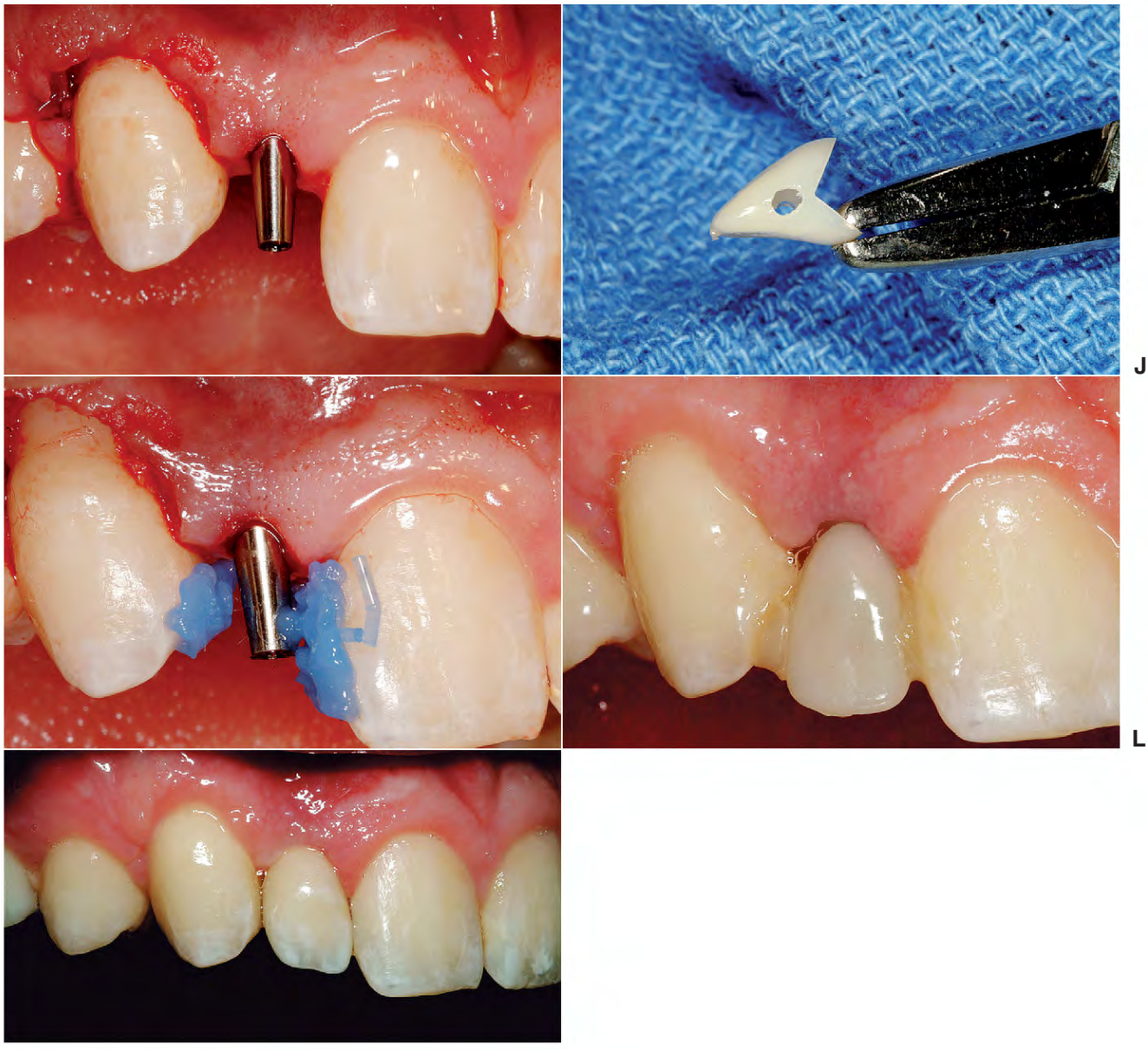


Figure 35-27, cont'd **I,** The implant in position. **J,** A hollow temporary crown with the interproximal contacts open is selected. **K,** The adjacent teeth are acid etched and bonded to the temporary crown. The hollow temporary is luted to the adjacent teeth with resin cement. The temporary crown is not attached to the implant. **L,** Soft tissue healing is allowed to take place over several months. **M,** A porcelain-metal crown is fabricated and cemented to the implant 4 or more months after surgery. Esthetic crown lengthening on the canine increased the crown height to be comparable with the central incisor.

risk fatigue fracture. A fracture of an implant will also place the adjacent teeth at risk during implant removal. An integrated implant that is fractured must be removed from the bone using a bur, at the expense of the bone and the adjacent teeth.

The indications for small-diameter implants are primarily related to limited mesiodistal spaces in the anterior region of the mouth. In this way the occlusal forces are reduced to decrease the risk of fracture. The single-tooth replacement for maxillary lateral incisors, mandibular central incisors, and mandibular lateral incisors often require smaller implant dimensions. In addition, when adjacent mandibular incisors are missing, splinting two smaller-diameter implants together is a better option than cantilevers from one implant. Two small-diameter implants have greater surface area than one traditional implant, and the moment force is reduced when the cantilever is eliminated.

WWW.HIGHDENT.IR

RISKS OF IMMEDIATE OCCLUSAL LOADING

The concept of immediate occlusal loading with dental implants offers several advantages. The implant abutments are placed at the time of surgery or at the suture removal appointment. As such, a second-stage surgery is eliminated, along with a suture removal appointment. This saves the patient pain and suffering and saves the dentist overhead time and material. The patient does not need to wear a removable prosthesis during the healing time, which is often a significant advantage when patients are having problems with these devices. The soft tissue–borne restoration also may load the implant inadvertently through the soft tissue and cause bone loss or failure. When the clinician uses a one-stage surgical procedure, the implants are independent during healing. When immediately loaded, the implants are splinted together during healing, which is biomechanically superior. The soft tissue is allowed to mature for several months before the fabrication of the final restorations. Tissue maturation is most important in esthetic zones, where tissue shrinkage after second-stage surgery may compromise the soft tissue directly related to margins and papilla contour around the final restorations.

In the immediate-loading technique for the completely edentulous patient, more implants usually are inserted, which increases the fee and makes patient acceptance less likely.

The litigious risk is low in dentistry but increases as the fees increase. As such, because immediate loading of fixed prostheses is expensive, a failure may increase a malpractice case against the dentist, especially because the patient may need to wear a removable prosthesis and may be subjected to several additional surgeries and appointments.

The primary disadvantage of immediate loading is the risk of implant failure or greater crestal bone loss around the healing implants. When an implant failure occurs, several side effects follow. Implant overload failure most often is associated with bone loss around the implant. If the bone loss includes a lateral cortical plate, then the bone width does not regenerate on its own after the implant is removed. As a result, a bone graft often is required. The bone graft often is separated from the implant extraction procedure to improve the incision line healing and decrease the risk of infection. As a result, two or three appointments usually pass before diagnosis of the implant failure, two appointments are required to remove the implant (one surgery and one suture removal), and two appointments are required for the bone graft. The bone graft often is evaluated every 2 months over a 6-month period for three more appointments. The patient then has the implant or implants replaced, for two more appointments (one surgery and one suture removal). Therefore a total of 12 appointments are associated with the implant failure over 6 to 8 months. During this time the patient most often is wearing a removable prosthesis, the very thing that often is described as a disadvantage for traditional two-stage procedures. In addition, the second implantation procedure usually does not follow an immediate-load format because the doctor and patient are more aware of the risks of the procedure. The patient wears the removable device an additional 3 to 6 months after the bone graft, during a more traditional two-stage healing period of the implant reinsertion. After this time, another two appointments are used to uncover and remove the sutures. As a result, as many as 14 additional appointments may occur for each implant failure.

Usually the dentist does not charge a fee for the implant extraction, bone graft, implant reinsertion, suture removal, and tissue checks for the treatment of the complication because the doctor-patient relationship already may be strained after the failure, and patients do not feel the additional procedures are their responsibility. Therefore loss of production, time, and income of several thousand dollars results.

The overhead in a typical implant practice is greater than 60%. The implantation procedure is repeated at no fee, but the overhead remains. In addition, the doctor loses the income of the procedures redone. When the additional appointments and procedures are added to the implant failure, the doctor most often loses the profit of five or more successful cases for each implant failure. Therefore if immediate loading is 90% successful, then one of 10 cases may fail. The one of 10 cases that fails causes the doctor to lose the profits of five cases. Therefore 90% success means the doctor is paid for 50% of the cases performed. This is a considerable economic loss in an implant practice.

In addition to lost income, the implant practice is also at increased risk. In a referral patient, the referring doctor may lose confidence in the surgeon when failure occurs. As a result, the failure also may cause future referral loss from the restoring dentist.

Early crestal bone loss after implant loading may have a relationship to occlusal overload. Whether immediate loading increases the risk of early crestal bone loss is unclear but is strongly suspected. The early loading was speculated to interfere with the ability of necrotic bone (created by the surgical trauma) to be replaced by newly formed bone. Successful implants with greater than 5-mm soft tissue pockets may be more often a result of immediate loading.

SUMMARY

The delivery of care for patients missing one or all of their teeth very often requires implants to restore function, esthetics, bone and soft tissue contours,

WWW.HIGHDENT.IR

speech, and intraoral health. The delayed occlusal-loading protocol, either one- or two-stage approach, has been evaluated for more than 30 years by a number of clinical settings and situations. However, in some patient conditions, the delayed healing process can cause psychological, social, speech, and/or function problems. A full range of treatment options relative to the initial hard and soft tissue healing is available. Immediate restoration of a patient after implant surgery is one of these alternatives.

A benefit/risk ratio may be assessed for each patient condition to ascertain whether immediate occlusal loading is a worthwhile alternative. The greater the benefit and/or the lower the risk, the more likely that immediate loading is considered. A complete edentulous mandible restored with an overdenture supported by four or more implants is a very low-risk condition. If the patient cannot tolerate a mandibular denture and does not wear the device, then an immediate-load protocol would be a high benefit. The highest risk for immediate loading would be a posterior single-tooth implant. Implant number cannot be increased, and implant length cannot engage cortical bone. When the single-tooth replacement is out of the esthetic zone, very low benefit is obtained with the immediate-restoration approach.

Additional clinical studies to evaluate the associated risks, especially in the maxillary arch, are expected over the next several years. Until the profession has longer-term evidence and more multicenter studies, immediate occlusal loading will be a secondary treatment option, restricted on a case-by-case basis.

A biomechanical rational for immediate loading may decrease the risk of occlusal overload during initial healing. The stresses applied to the implant support system result in strain to the bone interface. The greater the stress, the higher the strain. Increasing implant area and/or reducing the forces applied to the prosthesis may reduce stress. The implant size, design, and surface condition all affect the area over which the occlusal forces are dissipated. The forces may be reduced by patient factors, implant position, reducing force magnifiers such as crown height or cantilever length, reducing the occlusal contacts, decreasing angled forces to the prostheses, and altering the diet. The mechanical properties of bone also affect the risk of overload, because the bone density is directly related to the strength of bone, its elastic modulus, and the amount of BIC. All these factors are important in the traditional two-stage approach. They are especially noteworthy for immediate loading, because the surgical trauma of placing the implant also modifies the mechanical properties of bone during initial healing.

The majority of clinical reports reveal similar survival rates between immediately loaded and two-stage unloaded healing approaches in the completely edentulous patient. Nonetheless, these findings do not imply that a submerged surgical approach is no longer necessary or prudent in many cases. Future studies may find indications based on surgical, host, implant, and occlusal conditions more beneficial for one versus the other. For example, the strength of bone and the modulus of elasticity are related directly to bone density. The softest bone type may be 10 times weaker than for the densest types. The microstrain mismatch of titanium and the softest bone is much greater than the densest bone. As a result, higher implant failure and greater crestal bone loss seem likely but as yet are not reported in the literature.

The biomechanical treatment approach to increase surface area and decrease forces applied to the immediate restorations is most likely the major reason for the high implant survival.

References

1. Brånemark PI, Hansson BO, Adell R et al: Osseointegrated implants in the treatment of edentulous jaw: experience from a 10 year period, *Scand J Plast Reconstr Surg* 2 (suppl 10):1-132, 1977.
2. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 10:387-416, 1981.
3. Van Steenberghe D, Lekholm N, Bolender C et al: The applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study of 558 fixtures, *Int J Oral Maxillofac Implants* 5:272-281, 1990.
4. Gotfredsen K, Hjorting-Hansen E: Histologic and histomorphometric evaluation of submerged and nonsubmerged titanium implants. In Laney WR, Tolman DE, editors: *Tissue integration in oral, orthopedic, and maxillofacial reconstruction*, Chicago, 1990, Quintessence.
5. Schroeder A, Mawglen B, Sutter F: Hohlzylinderimplantat: Typ-F zur Prothesen-retention bei zahnlosen Kafer, *SSO Schweiz Monatsschr Zahnheilkd* 93:720-733, 1983.
6. Buser D, Weber HP, Bragge U et al: Tissue integration of one stage ITI implants: 3 year results of a longitudinal study with hollow cylinder and hollow screw implants, *Int J Oral Maxillofac Implants* 6:405-412, 1991.
7. Strock AE, Strock M: Experimental work on a method for the replacement of missing teeth by direct implantation of a metal support into the alveolus, *Am J Orthod* 25:467-472, 1939.
8. Linkow LI: The blade-vent: a new dimension in endosseous implants, *Dent Concepts* 11:3, 1968.
9. Cranin AN, Rookin MF, Garfinkel L: A statistical evaluation of 952 endosteal implants in humans, *J Am Dent Assoc* 94:315, 1977.
10. Smithloff M, Fritz ME: The use of blade implants in a selected population of partially edentulous adults: a five-year report, *J Periodontol* 47:19, 1976.
11. Kapur KK: Veterans Administration co-operative dental implant study comparison between fixed partial dentures supported by blade-vent implants and partial dentures, *J Prosthet Dent* 59:499-512, 1987.
12. Linkow LI, Donath K, Lemons JE: Retrieval analysis of a blade implant after 231 months of clinical function, *Implant Dent* 1:37-43, 1992.
13. Misch CE, Wang HL, Misch CM et al: Rationale for the application of immediate load in implant dentistry. I. *Implant Dent* 13:207-215, 2004.

WWW.HIGHDENT.IR

14. Babbush CA, Kent JN, Misiek DJ: Titanium plasma spray (TPS) screw implants for the reconstruction of the edentulous mandible, *J Oral Maxillofac Surg* 44:274-282, 1986.
15. Schnitman DA, Wohrle PS, Rubenstein JE: Immediate fixed interim prostheses supported by two-stage threaded implants: methodology and results, *J Oral Implantol* 16: 96-105, 1990.
16. Schnitman DA, Wohrle PS, Rubenstein JE et al: Brånemark implants immediately loaded with fixed prostheses at implant placement: ten year results, *Int J Oral Maxillofacial Implants* 12:495-503, 1997.
17. Tarnow DP, Emtiag S, Classi A: Immediate loading of threaded implants at stage one surgery in edentulous arches: ten consecutive case reports with 1 to 5 year data, *Int J Oral Maxillofac Implants* 12:319-324, 1997.
18. Scortecci G: Anchored disk-design implants without bone augmentation in moderately to severely resorbed completely edentulous maxillae, *J Oral Implantol* 25:37-79, 1999.
19. Horiuchi K, Uchida H, Yamamoto K et al: Immediate loading of Brånemark system implants following placement in edentulous patients: a clinical report, *Int J Oral Maxillofac Implants* 15:824-830, 2000.
20. Ganeles J, Rosenberg MM, Holt RL et al: Immediate loading of implants with fixed restorations in the completely edentulous mandible: report of 27 patients from a private practice, *Int J Oral Maxillofac Implants* 16:418-426, 2001.
21. Jaffin RA, Kumar A, Berman CL: Immediate loading of implants in partially and fully edentulous jaws: a series of 27 case reports, *J Periodontol* 71:833-838, 2000.
22. Misch CE, Degidi M: Immediate loading implants with fixed prostheses in the completely edentulous patient, *Clin Implant Dent Relat Res* 5:100-203, 2003.
23. Strid KG: Radiographic results. In Brånemark PI, Zarb GA, Albrektsson T, editors: *Tissue integrated prostheses,* Chicago, 1985, Quintessence.
24. Frost HM: The regional acceleratory phenomenon: a review, *Henry Ford Hosp Med Bull* 31:3-9, 1983.
25. Enlow DH: *Principles of bone remodeling,* Springfield, Ill, 1963, Charles C Thomas.
26. Buchs AU, Levine L, Moy P: Preliminary report of immediately loaded Altiva natural tooth replacement dental implants, *Clin Implant Dent Relat Res* 3:97-105, 2001.
27. Roberts WE, Turkey PK, Brezniak N et al: Implants: bone physiology and metabolism, *CDA J* 15:54-61, 1987.
28. Roberts WE, Smith RK, Zilerman Y et al: Osseous adaptation to continuous loading of rigid endosseous implants, *Am J Orthod* 86:95-111, 1984.
29. Eriksson A, Albrektsson T, Grane B et al: Thermal injury to bone: a vital-microscopic description of heat effects, *Int J Oral Surg* 11:115-121, 1982.
30. Eriksson AR, Albrektsson J: Temperature threshold levels for heat-induced bone tissue injury: a vital microscopic study in the rabbit, *J Prosthet Dent* 50:101-107, 1983.
31. Sharawy M, Misch CE, Wellner N et al: Heat generation during implant drilling: significance of motor speed, *J Oral Maxillofac Surg* 60:1160-1169, 2002.
32. Matthews LS, Hirsh C: Temperatures measured in human cortical bone when drilling, *J Bone Joint Surg Am* 54:297-308, 1972.
33. Haider R, Watzek G, Plenk H: Effects of drill cooling and bone structure on IMZ implant fixation, *Int J Oral Maxillofac Implants* 8:83-91, 1993.
34. Satomi K, Akagawa Y, Nikai H et al: Bone implant interface structures after non tapping and tapping insertion of screw-type titanium alloy endosseous implants, *J Prosthet Dent* 59:339-342, 1988.
35. Parfitt AM: The physiological and clinical significance of bone histomorphometric data. In Reck RR, editor: *Bone histomorphometry, techniques and interpretation,* Boca Raton, Fla, 1983, CRC Press.
36. Romanos G, Tok CG, Sias CH et al: Peri-implant bone reactions to immediately loaded implants: an experimental study in monkeys, *J Periodontol* 72:506-511, 2001.
37. Sharawy M: *Immediate vs delayed loading in a canine model: a histometric and volume fraction analysis,* unpublished data, 2000.
38. Suzuki JB, Misch CE, Sharawy M et al: Clinical and histological evaluation of immediate loaded posterior implants in non-human primates, *Implant Dent* 16:176-186, 2007.
39. Testori T, Szmukler-Moncler S, Francetti L et al: The immediate loading of Osseotite implants: a case report and histologic analysis after 4 months of occlusal loading, *Int J Perio Restorative Dent* 21:451-459, 2001.
40. Piatelli A, Corigliano M, Scarano A et al: Bone reactions to early occlusal loading of two-stage titanium plasma-sprayed implants: a pilot study in monkeys, *Int J Periodontics Restorative Dent* 17:162-169, 1997.
41. Piatelli A, Corigliano M, Scarano A et al: Immediate loading of titanium plasma-sprayed implants: an histologic analysis in monkeys, *J Periodontol* 69:321-327, 1998.
42. Piatelli A, Ruggeri A, Franchi M et al: An histologic and histomorphic-metric study of bone reactions to unloaded and loaded non-submerged single implants in monkeys: a pilot study, *J Oral Implantol* 19:314-320,1993.
43. Randow R, Ericsson I, Nilner K et al: Immediate functional loading of Brånemark dental implants: an 18-month clinical follow-up study, *Clin Oral Implants Res* 10:8-15, 1999.
44. Ledermann PD: Long-lasting osseointegration of immediately loaded, bar-connected TPS screws after 12 years of function: a histologic case report of a 95-year-old patient, *Int J Periodontics Restorative Dent* 18:562-563, 1998.
45. Frost HM: Mechanical adaption of Frost's mechanostat theory. In Martin DB, Burr DB, editors: *Structure, function and adaption of compact bone,* New York, 1989, Raven Press.
46. Tricker ND, Garetto LP: Cortical bone turnover and mineral apposition in dentate dog mandible, *J Dent Res* 76(special issue):201, 1977.
47. Misch CE, Bidez MW, Sharawy M: A bioengineered implant for an ideal bone cellular response to loading forces: a literature review and case report, *J Periodontol* 72:1276-1286, 2001.
48. Brunski JB: Biomechanical factors affecting the bone-dental implant interface: review paper, *Clin Mater* 10:153-201, 1992.
49. Balshi TJ, Wolfinger GJ: Immediate loading of Brånemark implants in edentulous mandible: a preliminary report, *Implant Dent* 6:83-88, 1997.
50. Brånemark PI, Engstrand P, Ohrnell LO et al: Brånemark Novum: a new treatment concept for rehabilitation of the edentulous mandible—preliminary results from a prospective clinical follow-up study, *Clin Implant Dent Relat Res* 1:2-16, 1999.
51. Misch CE: Divisions of available bone. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.
52. Strong JT, Misch CE, Bidez MW et al: Functional surface area: thread form parameter optimization for implant body design, *Compend Contin Educ Dent* 19:4-9, 1998.

WWW.HIGHDENT.IR

53. Steigenga J: *Thread geometry and its effect on initial osteointegration using reverse torque testing and histometric analysis* [master's thesis], Ann Arbor, Mich, 2003, University of Michigan.
54. Reilly DT, Burstein AH: The elastic and ultimate properties of compact bone tissue, *J Biomech* 80:393-405, 1975.
55. Lum LB, Beirne OR, Curtis DA: Histologic evaluation of hydroxylapatite-coated versus uncoated titanium blade implants in delayed and immediately loaded applications, *Int J Oral Maxillofac Implants* 6:456-462, 1991.
56. Block MS, Kent JN, Kay SF: Evaluation of hydroxylapatite coated titanium dental implants in dogs, *J Oral Maxillofac Surg* 45:601-607, 1987.
57. Sullivan DO, Sennerby L, Meredith N: Measurements comparing the initial stability of five designs of dental implants: a human cadaver study, *Clin Implant Dent Relat Res* 2:85-92, 2000.
58. Sirota C, Fiorellini J, Corso M et al: Immediate loading of implants with various coating in beagle dogs, *J Dent Res* 75:400, 1996.
59. Evans GH, Mendez AJ, Caudill RF: Loaded and non-loaded titanium versus hydroxyapatite-coated threaded implants in the canine mandible, *Int J Oral Maxillofac Implants* 11:360-371, 1996.
60. Misch CE: Patient force factors. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.
61. Grunder U: Immediate functional loading of immediate implants in edentulous arches: two-year results, *Int J Periodontics Restorative Dent* 21:545-551, 2002.
62. Barbier L, Schepers E: Adaptive bone remodeling around oral implants under axial and non axial loading conditions in the dog mandible, *Int J Oral Maxillofac Implants* 12:215-223, 1997.
63. Albrektsson T: On long-term maintenance of the osseointegrated response, *Aust Prosthodont J* 7:15-24, 1993.
64. Arvidson K, Bystedt H, Frykholm A et al: A 3-year clinical study of Astra dental implants in the treatment of edentulous mandibles, *Int J Oral Maxillofac Implants* 7:321-329, 1992.
65. Misch CE: Partial and complete edentulous maxilla implant treatment plans: fixed and overdenture prostheses. In Misch CE, editor: *Dental implant prosthetics,* St Louis, 2005, Mosby.
66. Misch CE: Mandibular full-arch implant fixed prosthetic options. In Misch CE, editor: *Dental implant prosthetics,* St Louis, 2005, Mosby.
67. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing and progressive bone loading, *Int J Oral Implantol* 6:23-31, 1990.
68. Misch CE, Qu Z, Bidez MW: Mechanical properties of trabecular bone in the human mandible: implications for dental implant treatment planning and surgical placement, *J Oral Maxillofac Surg* 57:700-706, 1999.
69. Chiapasco M, Gatti C, Rossi F et al: Implant-retained mandibular overdentures with immediate loading: a retrospective multicenter study on 226 consecutive cases, *Clin Oral Implants Res* 8:48-54, 1997.
70. Misch CE: Non-functional immediate teeth in partially edentulous patients: a pilot study of 10 consecutive cases using the Maestro dental implant system, *Compend Contin Educ Dent* 19(special issue):25-36, 1998.
71. Misch CE: Non-functional immediate teeth, *Dent Today* 17:88-91, 1998.
72. Graf H: Occlusal forces during function. In Rowe NH, editor: *Proceedings of symposium on occlusal research in form and function,* Ann Arbor, Mich, 1975, University of Michigan.
73. Wohrle P: Single tooth replacement in the aesthetic zone with immediate provisionalization: fourteen consecutive case reports, *Prac Periodont Aesthet Dent* 10:1107-1114, 1998.
74. Graber JM: Anomalies in number of teeth. In Graber TM, editor: *Orthodontics principles and practice,* ed 2, Philadelphia, 1966, WB Saunders.
75. Misch CE: Single tooth implants. In Misch CE, editor: *Contemporary implant dentistry,* ed 2, St Louis, 1999, Mosby.
76. Wheeler RC: *A textbook of dental anatomy and physiology,* ed 4, Philadelphia, 1965, Lea & Febiger.
77. Tarnow DP, Eskow RM: Preservation of implant esthetics, soft tissue and restorative consideration, *J Esthet Dent* 8:12-19, 1996.
78. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, *Postgrad Dent* 2:3-17, 1995.
79. Misch CE: Divisions of available bone in implant dentistry, *Int J Oral Implantol* 7:9-17, 1990.
80. Bidez MW, Misch CE: Clinical biomechanics in implant dentistry. In Misch CE, editor: *Contemporary implant dentistry,* ed 2, St Louis, 1999, Mosby.
81. Brown SA, Lemons JE: Medical applications of titanium and its alloys, *ASTM Stand News* 1272:103, 1996.
82. Lemons JE, Dietsh-Misch F: Biomaterials for dental implants. In Misch CE, editor: *Contemporary implant dentistry,* ed 2, St Louis, 1999, Mosby.
83. Helkimo E, Carlsson GE, Helkimo M: Bite force and state of dentition, *Acta Odontol Scand* 35:297-303, 1977.
84. Boggan S, Strong JT, Misch CE et al: Influence of hex geometry and prosthetic table width on static and fatigue strength of dental implants, *J Prosthet Dent* 82:436-440, 1999.
85. Jorneus L, Jemt T, Carlsson L: Loads and designs of screw joints for single crowns supported by osseointegrated implants, *Int J Oral Maxillofac Implants* 7:353-359, 1992.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 36

Keys to Bone Grafting and Bone Grafting Materials

Carl E. Misch, Francine Misch-Dietsh

To satisfy the ideal goals of implant dentistry, the hard and soft tissues need to present ideal volumes and quality. The alveolar process is affected so often after tooth loss that augmentation is usually indicated to achieve optimum results, especially in the esthetic zones. Augmentation is also required relative to functional conditions of the implant treatment plan, because a reduction of stress at the crestal bone region and a greater resistance to screw loosening and fatigue fracture occurs with larger-diameter implants. Therefore an improved understanding of biomechanical requirements for long-term prosthesis survival and the increasing use of implants in esthetic restorations often require ridge reconstruction before implant placement for complete or partially edentulous patients. This is especially true in the surgical placement of maxillary anterior implants, which is usually critical for ideal esthetics, phonetics, and function. As a result, treatment methods to improve the recipient bone dimensions to optimize success should be considered, especially in the premaxilla.

ALVEOLAR RESORPTION

In the anterior regions of the mouth, the labial cortical plate over the natural teeth is much thinner than its lingual counterpart. Periodontal disease creates infrabony defects on the lingual aspect of the supporting bone but often causes complete loss of the labial alveolar process. Dehiscence of the labial plate may also occur as a consequence of tooth eruption, orthodontic therapy, parafunction, trauma, vertical root fracture, apicoectomy, ill-fitting crown margins, subgingival tooth preparation, and extractions. The facial plate of bone is also first to remodel or resorb after tooth extraction, disease, or trauma, and it does so to a greater extent than the lingual cortical bone.

In the anterior maxilla, the alveolar bone is rapidly recontoured after the loss of the natural teeth, even in the presence of an intact alveolus after extraction. There is a 25% decrease in volume during the first year and a 40% to 60% decrease in width within the first 3 years after tooth loss.[1,2] This resorption is primarily at the expense of the facial dimension, as the lingual contour undergoes resorption only when more advanced atrophy occurs. As a result, an 8-mm-wide anterior ridge may remodel to less than 3 mm within 5 years after extraction.[3,4] In the posterior regions, the rate of initial bone loss is often greater than in the anterior regions. However, because the initial posterior ridge dimension is twice the width, even a 50% bone loss often leaves adequate volume to place 4-mm-diameter implants. As a consequence of these dimensional changes, the remodeled labial cortex is more medial than its original position.

SURGICAL KEYS TO BONE GRAFTING

Consistent bone grafting results for volume have been difficult to achieve, often because similar techniques are used, regardless of the patient's existing conditions, the volume of bone, and the region of the augmentation. Specific elements (keys) need to be present for successful bone grafting.[5] The doctor should evaluate the existing condition and alter the grafting technique and materials in function of each treatment performed. In other words, bone grafting is very much like opening a combination safe that requires at least seven of 11 different numbers. If only five or six numbers are correct, the safe will not open. The more numbers used to open the "safe" of successful bone grafting, the more predictable the growth of sufficient bone volume for implant placement.

The keys to bone grafting are local factors that affect the prognosis of the procedure and include: absence of infection, soft tissue closure, space maintenance, graft immobilization, regional acceleratory phenomenon (RAP), host bone vascularization, growth factors, bone morphogenetic proteins (BMPs), healing time, defect size and topography, and transitional prostheses. Several

WWW.HIGHDENT.IR

همیار دندانسازان

of these keys are interrelated; often, one key affects another and may form a cascade toward failure or success. The surgeon should attempt to provide all these elements, but especially those factors missing in the bone augmentation site.

Graft materials such as collagen, autogenous bone, demineralized freeze-dried allograft (DFDB), and calcium phosphate materials also are necessary elements for bone grafting procedures. The purpose of this chapter is to present these keys and materials as a basis for the methods presented within this book for predictable intraoral bone grafting for implant dentistry.

Surgical Asepsis/Absence of Infection

Bone and graft materials resorb at different rates under normal pH conditions, based upon porosity, size, and crystallization. However, all graft materials rapidly resorb through solution-mediated resorption in conditions of low pH (Figure 36-1). The hydroxylapatite crystal of bone (or enamel) is dissolved into calcium and phosphate components at a pH of 5.5 or less. For example, the enamel of a tooth is composed of 95% dense, crystalline hydroxylapatite.

Lactobacillus acidophilus bacteria produce a pH of 5.5, and when bacterial plaque and bacteria remain in enamel for more than 5 days, a solution-mediated resorption occurs, which is seen as a radiolucent zone on the radiograph. Infections within the bone often create a pH of less than 2. As a result, when a tooth becomes nonvital, a solution-mediated resorption occurs at the apex and a radiolucency at the apex of the tooth with an endodontic lesion may occur within a few days. Therefore bone grafting in the presence of infection, or infected bone grafts after surgery, increase the risk of insufficient volumes of bone formation and may even cause recipient bone loss (Figure 36-2). Before bone grafting, all evidence or potential causes of infection should be eliminated.

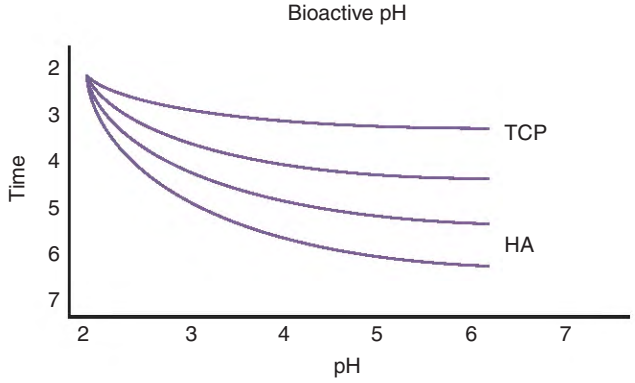


Figure 36-1 Bone graft materials resorb at different rates under normal pH conditions. However, when the pH is low, all graft materials (including bone) resorb rapidly through a solution-mediated resorption. *TCP,* Tricalcium phosphate; *HA,* hydroxyapatite.

Contamination of the bone graft may occur from endogenous bacteria, lack of aseptic surgical technique, or failure of primary soft tissue closure. Graft materials that fall into the oral cavity may be contaminated by saliva and should be thoroughly irrigated before use or discarded. The lack of primary soft tissue closure or incision line opening places the graft at significant risk. Barrier membranes or fixation screws that become exposed often become contaminated by bacteria. The bacteria invade the graft site and cause local inflammation with resultant decrease in bone formation.[6-10]

A blood supply within the graft is required for the normal distribution of an antibiotic to the site. Because no blood supply is present early on in the graft material, when bacterial contamination is a greater risk (i.e., sinus grafting), antibiotics may be added to the alloplastic material and autograft. Although tetracycline is often used in periodontal bone grafting to improve collagen formation, it chelates calcium and arrests the bone formation process.[11-13] Instead, parenteral penicillin, cephalosporin, or clindamycin may be mixed into the graft material, as these antibiotics do not affect the bone regeneration process (Figure 36-3). Tablets or capsules for oral administration of antibiotics are not used in

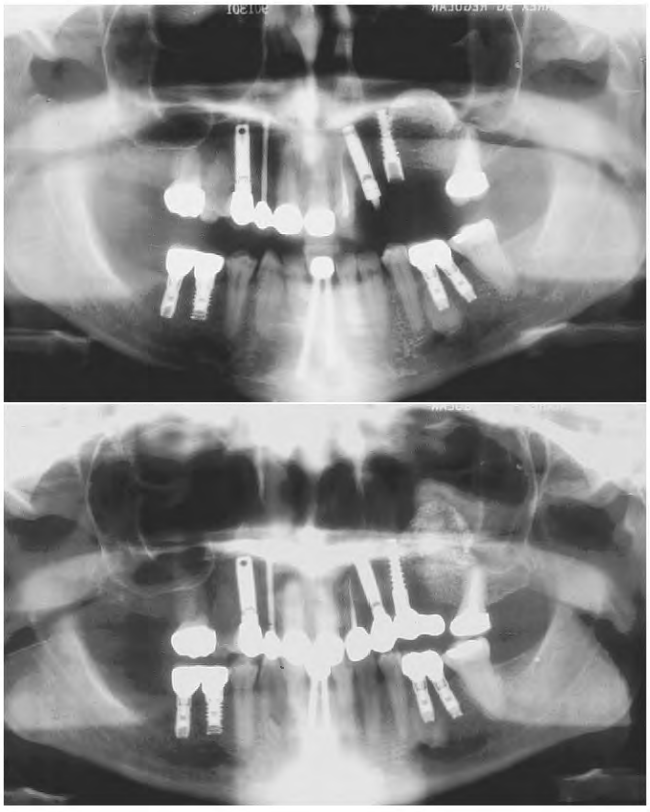


Figure 36-2 **A,** A panoramic radiograph of a sinus graft immediately post surgery. The radiopaque portion of the right sinus is from the mineralized, ceramic graft material which was inserted onto the maxillary floor. **B,** A postoperative panoramic radiograph of the same patient after a maxillary sinus infection, 2 weeks after the sinus surgery. The graft shows evidence of solution-mediated resorption of the graft material as a result of the infection.

WWW.HIGHDENT.IR

Figure 36-3 A parenteral form of cefazolin sodium (Ancef) may be added to the graft material when the site has been contaminated or become infected in the early healing process. This provides increased levels of the antibiotic at the site, even though vascularization is incomplete.

the graft site, as these often contain fillers of no benefit to the graft site.

Soft Tissue Coverage (Submucosal Space Technique) and Flap Design

Primary soft tissue closure is a mandatory condition for the success of grafting procedures other than socket grafting. It ensures healing by primary intention and requires minimal soft tissue collagen formation and soft tissue remodeling. It also minimizes postoperative discomfort. It is a necessary step for predictable bone regeneration. Even when socket grafting is performed without primary closure after extraction of a tooth, the epithelium covers the healing socket before bone forming in the crestal region.

Incision line opening during initial healing is the most common postoperative complication in intraoral bone grafting.[14-16] As a result, the graft is contaminated or lost, vascularization is delayed or eliminated, and bone growth is impaired.[8-10,17-20] The reason incision line opening is more common during bone grafting, compared with implant surgery, is that the overlying tissue must be advanced over a larger volume of bone and the tension on the incision line may pull the soft tissues apart. In addition, the soft tissues are poor in local growth factors under the reflected flaps that lie over a graft material or barrier membrane, rather than the host bone.

There are general guidelines to reduce the incidence of incision line opening.[21] The primary incision should be in keratinized tissue whenever possible. Not only does this reduce the initial intraoral bleeding, but it severs smaller blood vessels and reduces postoperative edema, which may add tension to the incision line. The crestal incision is designed more lingual, especially in the maxilla, as this places a greater amount of keratinized tissue onto the thinner facial flap and minimizes tearing of the tissue during suturing. Vertical relief incisions are designed away from the graft site directly on the host bone and create a broad-based flap[21,22] (Figure 36-4, *A* to *C*).

The blood supply to the reflected flap should be maintained whenever possible. The primary blood supply to the facial flap, which is most often the flap reflected for a bone graft, is from the unkeratinized mobile mucosa. This is especially true where muscles of facial expression or functional muscles attach to the periosteum. Therefore these vertical release incisions are made to the height of the mucogingival junction, and the facial flap is reflected only 5 mm above the height of the mucogingival junction. Both these procedures maintain more blood supply to the facial flap. In addition, incisions and reflection in the mobile alveolar mucosa increase flap retraction during initial healing, which may contribute to incision line opening and may increase risk of scar formation and delayed healing of the incision line as a consequence of reduced blood supply (see Figure 36-4, *D*, *E*).

The soft tissue flap design should also have the margins of the wound over host bone, rather than on the bone graft or barrier membrane. The host bone provides growth factors to the margins and allows the periosteum to regenerate faster to the site. The margins distal to the elevated flap should have minimal reflection. The palatal flap and the facial tissues distal to the reflected flap should not be elevated from the palatal bone (unless augmentation is required), because the blood supply to the incision line will be delayed. In addition, the unreflected flap does not retract during initial healing, which could place additional tension on the incision line. The soft tissue reflection distal to the graft site is split thickness to maintain some of the periosteum on the bone around the incision line. This improves the early vascularization to the incision line and adhesion of the margins to reduce retraction during initial healing (see Figure 36-4, *F, G*).

Primary wound closure should be without tension. Past techniques to expand tissue primarily used a more apical tissue reflection and horizontal scoring of the periosteum parallel to the primary incision. This is usually effective for primary closure when less than 5-mm advancement of the flap is necessary.

A submucosal space technique, developed by Misch in the early 1980s, is an effective method to expand tissue over larger grafts (greater than 15 × 10 mm in height and width).[5] The full-thickness facial flap first is elevated off the facial bone for only 5 mm above the height of the vestibule. One incision with a scalpel, 1 to 2 mm deep, is made through the periosteum parallel to the crestal incision and 3 to 5 mm above the vestibular height of the mucoperiosteum. This shallow incision is made the full length of the facial flap and may even extend above and beyond the vertical release incisions (see Figure 36-4, *H*). Care is taken to make this incision

WWW.HIGHDENT.IR

همیار دندانسازان

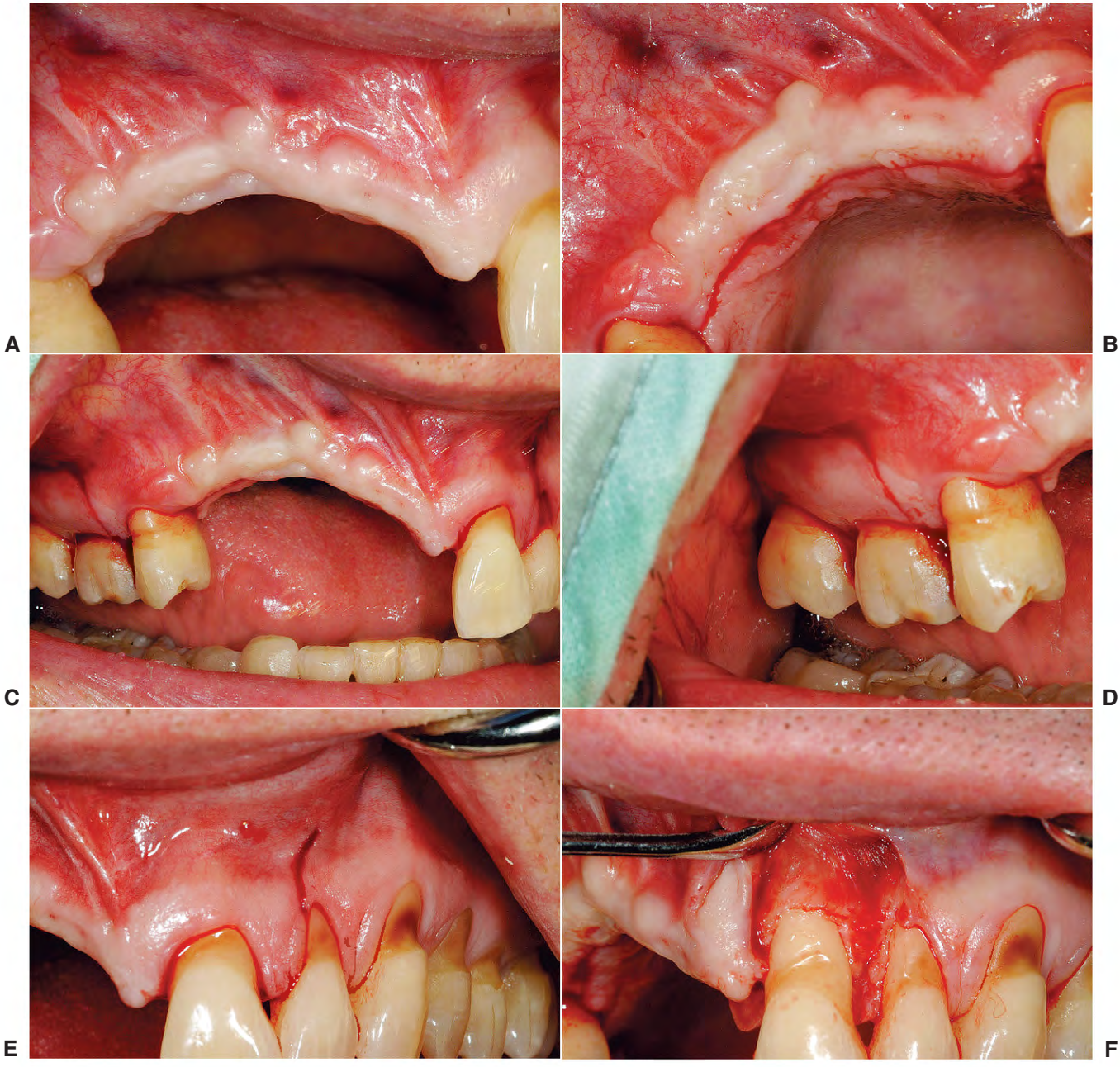


Figure 36-4 **A,** The site of the bone augmentation should ideally have adequate zones of attached keratinized tissue. **B,** The crestal incision is made in the attached keratinized tissue and slightly toward the palate. Incisions in attached keratinized mucosa minimize bleeding during surgery and edema post surgery, as smaller blood vessels are present compared with those in mobile, unkeratinized mucosa. A greater surface of keratinized tissue on the facial aspect provides a band of more tenacious tissue on the more mobile facial tissue flap. The risk of sutures tearing through the soft tissue during postoperative swelling or flap movement during function is therefore minimized. **C,** The larger the bone augmentation site, the farther distal the vertical release incisions. This increases blood supply to the reflected flap and minimizes tension on the incision line postoperatively. It also places the vertical release incisions on the split-thickness reflected sites away from the graft site. **D,** The vertical release incisions are made more distal from the augmentation site, as the size of the graft site increases. This gives more tissue to the reflected flap and improves the blood supply. The more distal incisions also ensure the wound margins will be on host bone, not over the graft site. **E,** The vertical release incision does not extend beyond the mucogingival junction and into the mobile alveolar mucosa, as larger blood vessels would be severed, increasing bleeding, flap retraction during initial healing and scar formation at the incision line. **F,** The reflection of the flap is split thickness away from the graft augmentation site, to improve early vascularization and reduce retraction of the flap.

Continued

above the microgingival junction; otherwise, the flap may be perforated and delay soft tissue healing. Soft tissue scissors (i.e., Metzenbaum) are used in a blunt dissection technique to create a tunnel apical to the vestibule and above the unreflected periosteum. The scissors are closed and pushed through the initial scalpel incision approximately 10 mm deep, then opened. This submucosal space is parallel to the surface mucosa (not deep toward the overlying bone) and above the unreflected periosteum. The thickness of the facial flap should be 3 to 5 mm, because the scissors are parallel to the surface (see Figure 36-4, *I*, *J*). This tunnel is

WWW.HIGHDENT.IR

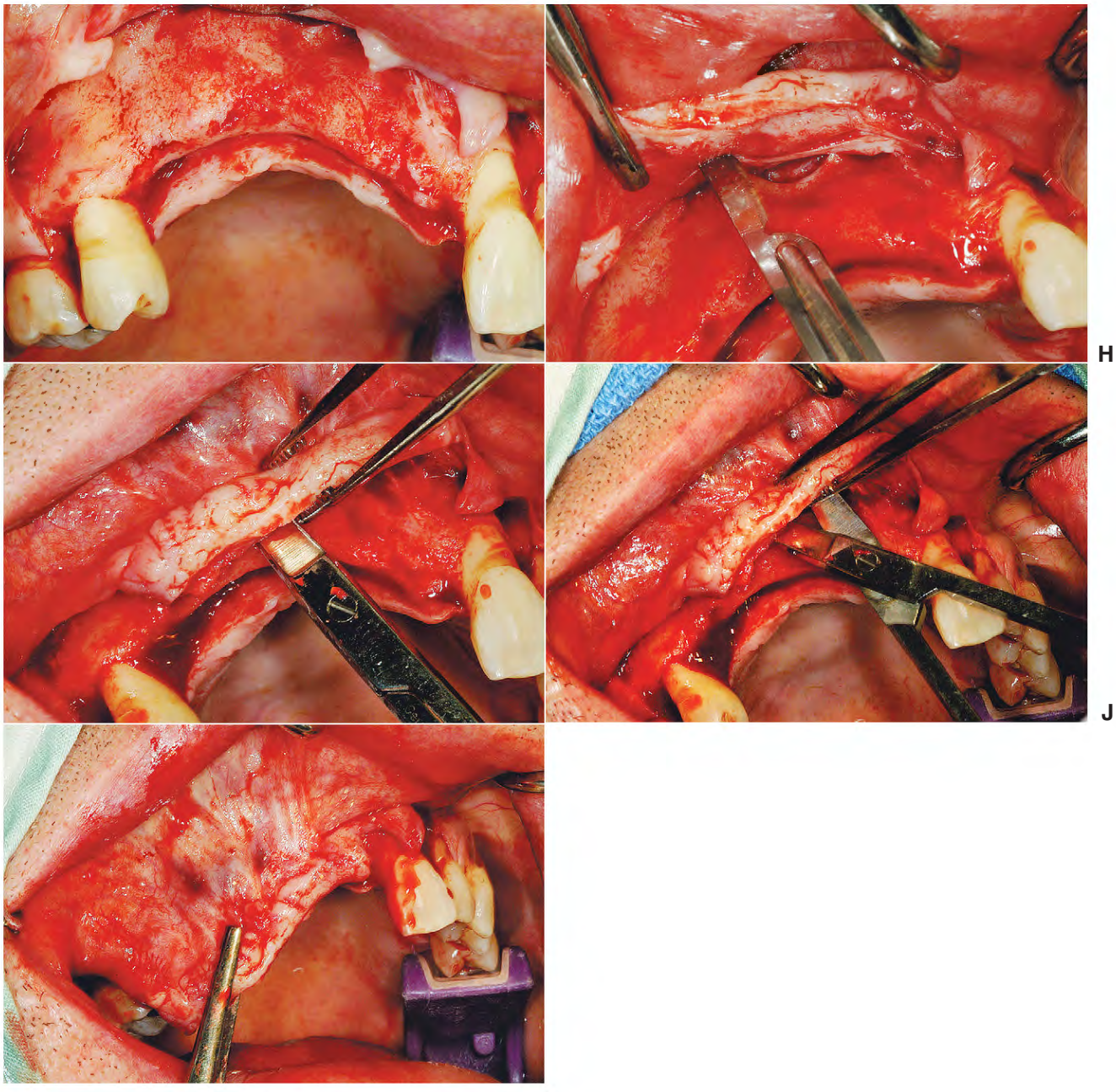


Figure 36-4, cont'd **G,** The full-thickness facial flap is reflected from the host bone 5 mm above the height of the mucogingival junction. **H,** A scalpel makes one incision parallel to the crestal incision, 1 to 2 mm deep, and extends the full length of the flap, 3 to 5 mm above the MGJ. **I,** A tissue pick-up may be positioned on the facial flap, with the end of the beak at the height of the mucogingival junction. When the flap is reflected, the lingual aspect of the tissue pick-up transfers the height dimension to the internal aspect of the flap. A soft tissue scissors (i.e., Metzenbaum) is pushed into the facial flap for approximately 10 mm with the blades closed, parallel to the surface mucosa. The thickness of the facial flap is approximately 3 to 5 mm. **J,** The tissue scissors are opened, once at the proper depth. This blunt dissection does not sever any blood vessels or nerves to the facial flap but does create a submucosal space or tunnel. **K,** Once the submucosal space or tunnel is created over and beyond the vertical release incisions, the facial flap may be advanced over the graft for primary closure, without tension.

expanded with the tissue scissors several millimeters above and distal to the vertical relief incisions.

Once the submucosal space is developed, the flap may now advance the distance of the "tunnel" and drape over the graft, to approximate the tissue for primary closure without tension (see Figure 36-4, *K*). In fact, the facial flap should be able to advance over the graft and past the lingual flap margin by more than 5 mm. Then the facial flap may be returned to the lingual flap margin and sutured. This soft tissue procedure is performed before preparing the host region and harvesting the donor site. Access to the facial tissues is easier before graft placement, and particulate graft and membranes barrier are not dislodged during the soft tissue manipulation when it is performed before bone grafting.

WWW.HIGHDENT.IR

همیار دندانسازان

The submucosal space technique is very effective to achieve tension-free closure over large graft sites. However, a side effect of the procedure is the loss of vestibular depth, especially when grafting for residual ridge height. In addition, a lack of keratinized tissue also may exist on the facial region of the grafted site, because the original facial tissue is now part of the crestal region after bone augmentation. As a consequence, soft tissue grafts or vestibuloplasties may be required after bone grafting when within the esthetic zone. It is suggested that these procedures be delayed for at least 4 months to allow regeneration of the blood supply to the soft tissue and the underlying bone. When necessary, these soft tissue procedures can be associated with implant placement or implant uncovery procedures, or even prior to the bone graft. To have mature tissue for the soft tissue advancement procedures, the soft tissue graft should be done 3 months or longer before the bone graft .

Platelet-rich plasma (PRP) may be placed over the bone graft to provide an additional source of transforming growth factor beta (TGF-β) and vascular endothelial growth factor (VEGF), which promote collagen formation and blood vessel growth (Figure 36-5).

Suture material selection should be made in function of the type and size of the bone grafting.[23-30] Silk has been shown to release less tension during early retraction of the flap from healing and to elicit greater inflammation and may contribute to incision line opening more often than synthetic materials.[26,27] Therefore it is not recommended for bone augmentation procedures. Chromic gut causes inflammation, loses tension, and resorbs too quickly to maintain soft tissue approximation over an augmented site. Therefore it is not recommended when the tissues are advanced for a bone augmentation.[29] Polyglycolic acid (PGA, Vicryl) shows the mildest tissue reaction and maintains sufficient tension over the first 2 weeks to be used for most bone graft procedures.[28] In larger-size bone grafts, the soft tissue is often approximated for primary closure with nonresorbable sutures (e.g., Prolene, Gore-Tex). Resorbable sutures usually lose 50% of their tensile strength after 14 days and may be associated with a delayed incision line opening (Figure 36-6). Each penetration of the tissue by the suture causes an approximate 1-mm devital zone and the need for repair. When the sutures are too close to the margin, the devital zone may include the incision line. The nonresorbable sutures remain in place for 2 or more weeks to enhance soft tissue maturation.[30]

The selection of a specific suture design also follows basic principles.[23,24] Interrupted sutures should be used 3 to 5 mm from each side of the tissue margin and 3 to 5 mm apart, and are appropriate in short spans of edentulous spaces (Figure 36-7). Too many sutures or too much tension impair the blood supply to the incision line and increase the risk of incision line opening.

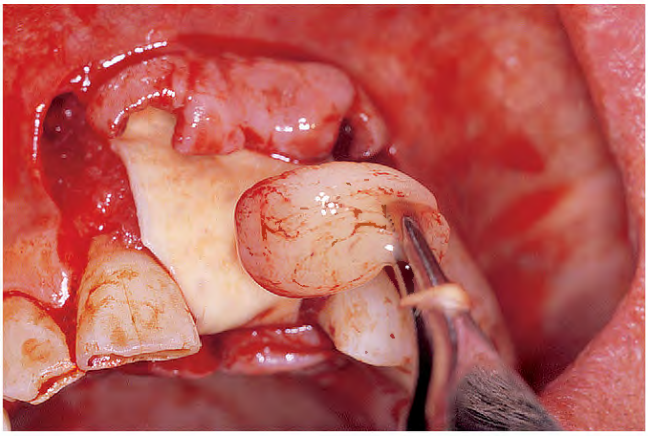


Figure 36-5 Platelet-rich plasma (PRP) may be applied to the graft site prior to soft tissue closure. TGF-β and VEGF are released from the PRP and may contribute to vascularization and early soft tissue healing.

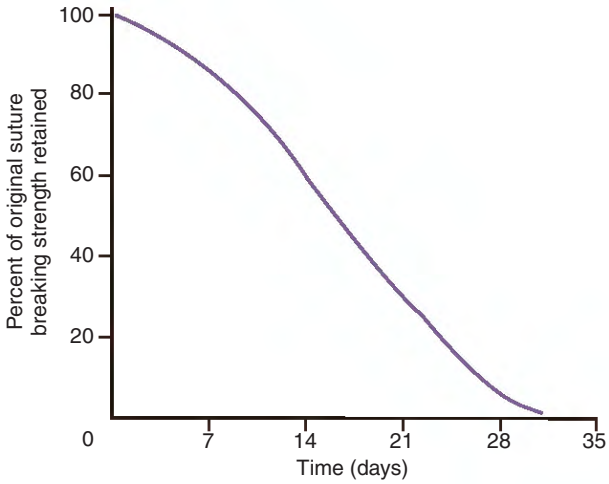


Figure 36-6 Vicryl sutures lose 50% of their strength 2 weeks after the initial surgery. As a result, a delayed incision line opening may occur if tension on the incision line from flap retraction during initial healing or from muscle pull during function/parafunction is present.

Because the tissues are passive while in place, sutures are not required to obtain the soft tissue closure. Soft tissue spans necessitating four or more interrupted sutures are best approximated with continuous nonlocking sutures. This suture design places less tension on the sutures and soft tissue and allows faster vascularization of the reflected soft tissue flaps.

Horizontal (or vertical) mattress sutures allow greater tension to be applied on the soft tissue closure without risk of tearing the soft tissue flap. It should be emphasized that they are not used to obtain primary closure when tension on the soft tissue flaps is present at surgery. The tissues should rest passively together before suturing. However, during function/parafunction movement of the tissues, the tension on the incision line may be reduced with a horizontal mattress suture. They are often used in the mandible when the floor of the mouth is in proximity to the lingual flap and the tissue is thin. They may also be used on a facial flap with a strong

WWW.HIGHDENT.IR

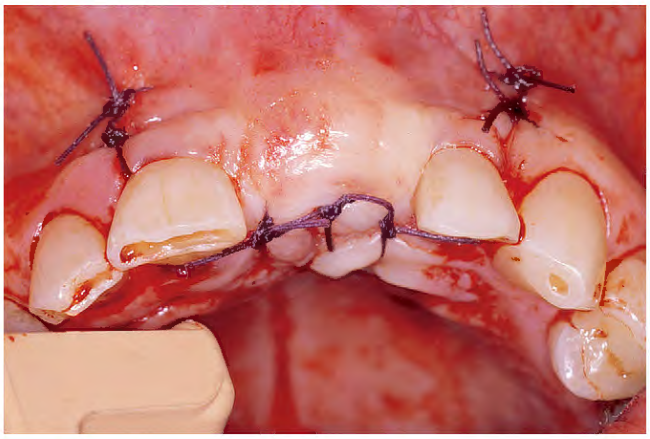


Figure 36-7 Interrupted (or continuous) sutures should be 3 to 5 mm from the incision line margin and 3 to 5 mm apart. They are used to passively approximate the tissues. Too many sutures delay vascularization to the flap and slow the healing process.

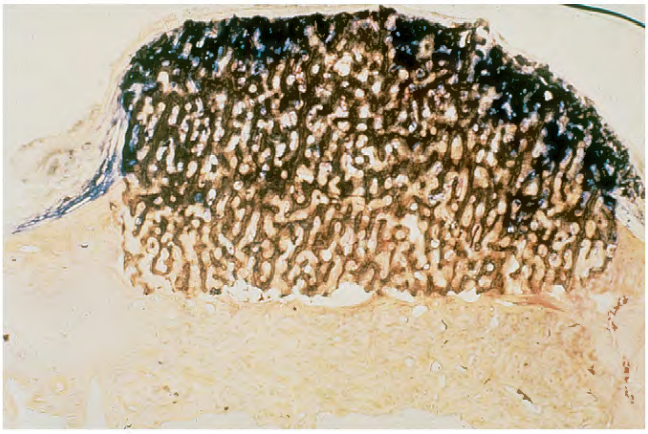


Figure 36-8 A "barrier by bulk" technique uses a graft material to maintain the space for a bone augmentation. In this picture, an alloplast (hydroxylapatite) block was placed on top of the bone and only soft tissue covered the site. After 4 months, the block biopsy was made and stained. The soft tissues stained blue and bone stained brown. Note the soft tissue invaded the block several millimeters, and bone grew into the porous block many millimeters. Removing the top layer of the graft material would remove the soft tissue and leave the rest of the site augmented with bone and the slowly resorbing graft material.

muscle pull on the soft tissue. In addition, horizontal mattress sutures avert the soft tissue margin and ensure primary closure without epithelium entrapment. A combination of a few horizontal mattress sutures with a continuous suture may be indicated to close large soft tissue spans.

No particulate graft material should be present in the incision line during initial primary closure, as this will delay soft tissue healing. Therefore, once the tissues are sutured, the incision line is inspected for any bone graft particles between the soft tissue margins.

Gentle pressure is then applied to the reflected soft tissue flaps for 3 to 5 minutes. This pressure may reduce postoperative bleeding under the flap, which may cause "dead spaces" and delayed healing. Any stagnant blood under the flap is "milked" from under the soft tissue by gentle pressure. This also allows the fibrin formation from the platelets to help "glue" the flap to the graft site.

Systemic corticosteroids may be administered before and after surgery to decrease soft tissue edema, as edema has been shown to contribute to incision line opening. Incision line opening has been associated with postoperative smoking[16,31-33]; therefore patients are instructed not to smoke until the incision line has healed. If a removable soft tissue–borne interim prosthesis is used, it should not contact the grafted area. When possible, a fixed transitional restoration is designed to reduce complications of incision line opening and graft micromovement during healing.

Space Maintenance

Space maintenance in the area of the bone graft site is paramount to the bone formation process. The *space* of the graft site refers to the anatomical size and contour of the desired augmentation, and *maintenance* refers to the fact the space must exist long enough for bone to fill the desired region. A barrier membrane is a sheet of material that covers the potential bone graft site and prevents the overlying soft tissue from growing into the graft site. A barrier membrane technique without graft material underneath has been suggested for ridge augmentation.[18,34-38] However, collapse of the space under the membrane may impair the desired size and contour. A fixation screw, elevated above the host bone level to the height/width of the desired bone volume, may support a barrier membrane and is called a *tent screw.* When the space under the barrier membrane is only air or blood, the initial quality of bone is poor for many months. Tent screws, titanium-reinforced membranes, and graft material beneath the membrane have been advocated to maintain the desired space during the augmentation process.[39-47] Space maintenance may be provided in a socket or sinus cavity by resorbable graft material. However, if the material resorbs too rapidly compared with the time required for bone formation, the site may fill with connective tissue rather than bone. Therefore the space or contour and size of the augmentation should remain until the bone graft has formed enough new bone to maintain the space itself.

The space for bone regeneration may also be provided by a graft material, as an autograft or alloplast "in excess."[48] The "barrier by bulk" concept by Misch applies to situations in which the graft site is over-contoured by several millimeters with a resorbable alloplast.[5] As bone grows below the alloplast, the invading fibrous tissue only invades the superficial alloplast layer. When the soft tissue is reflected to insert the implants, the top layer of fibrous tissue and alloplast is removed, and the new regenerated bone underneath remains (Figure 36-8). This technique works best when larger graft volumes still allow primary

WWW.HIGHDENT.IR

همیار دندانسازان

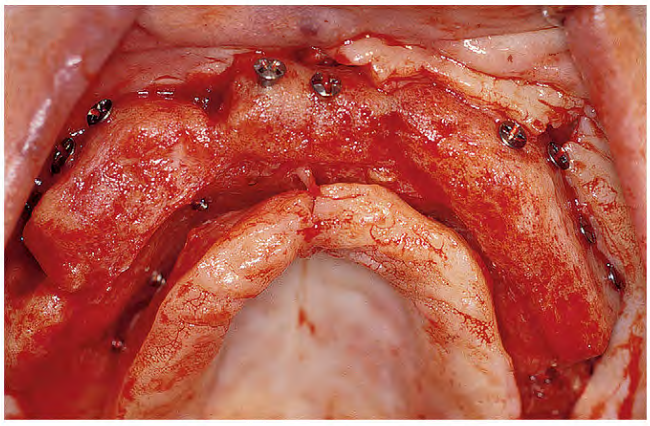


Figure 36-9 Block bone grafts from the iliac crest are fixated to the resorbed maxilla with fixation screws. These devices immobilize the graft during the bone modeling process.

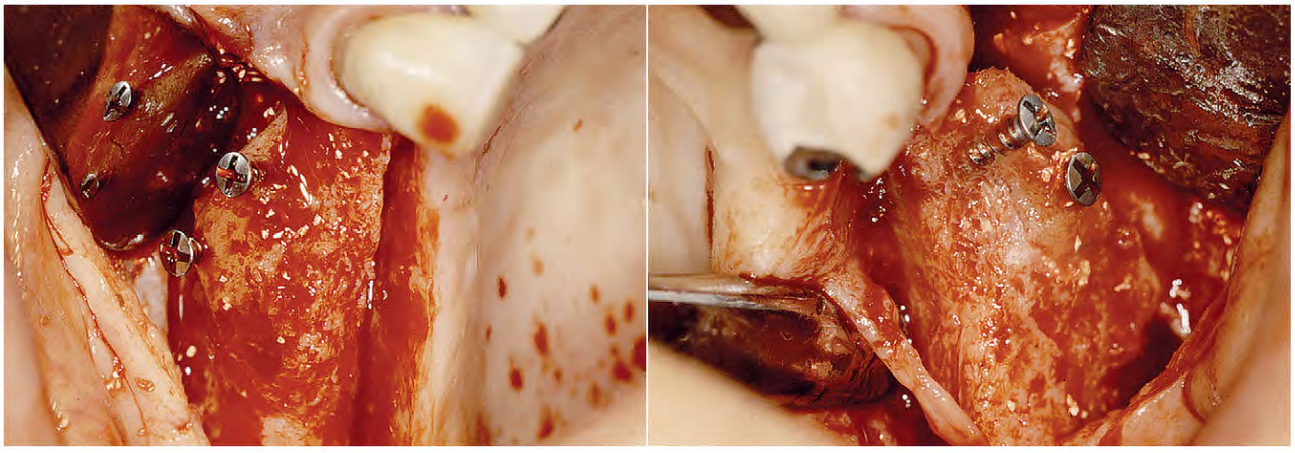
soft tissue closure and in the absence of pressure of the soft tissue. The use of soft tissue–borne provisional prostheses is discouraged for all augmentation procedures because the graft size may be modified and the graft may become mobile.

Graft Immobilization/Stability/Fixation

An implant with movement greater than 150 µm during healing does not integrate to the bone. A loaded implant may move as much as 100 µm during initial healing and develop a direct bone implant interface. However, this process occurs through bone remodeling and is less demanding on mobility. Even bone remodeling has its limits relative to micromovement. Changing the volume of bone is bone modeling and requires a more rigid interface during the bone formation process. Micromovement as low as 20 µm may be too much for bone modeling and may result in a nonfixated graft or fibrous encapsulation.

Graft stabilization is paramount to obtain a predictable bone augmentation. This ensures initial blood clot adhesion with its associated growth factors[49-56] (cytokines such as interleukin-1, interleukin-8, tumor necrosis factor, and growth factors such as platelet-derived growth factor [PDGF], insulin-like growth factor, and fibroblast growth factor [FGF]).[53-56] The granulation tissue that develops after blood clot stabilization is the initial mechanism for bone modeling and remodeling.[53,54] If pieces of a particulate graft material or block bone grafts are mobile, they cannot develop a blood supply for new bone formation. Instead, the graft becomes encapsulated in fibrous tissue and often sequestrates. Likewise, when barrier membranes or fixation screws become loose or mobile, fibrous tissue will encapsulate them. Therefore, for barrier membranes or particulate graft materials to work most effectively, no loads should be placed on the soft tissue over the graft, which may cause movement of the graft.

Barrier membranes may be immobilized to the graft site by bone tacks. As a result, when the soft tissue over the barrier membrane moves, the barrier membrane remains fixated. A tent screw placed under the barrier membrane can prevent movement to the graft site when the overlaying soft tissue is loaded. A block bone graft fixation with bone screws maintains a rigid interface more effectively than particulate grafts (Figure 36-9). A fixed transitional prosthesis, which protects the graft site, is the better option to limit graft movement during the augmentation process, especially with a "barrier by bulk" technique of particulate graft materials. When possible, soft tissue–bone removable restorations should have rest seats and clasps to prevent loading the soft tissue (Figure 36-10).



A B

Figure 36-10 **A,** Upon reentry, the particulate bone graft resorbed and provided inadequate volume for implant insertion. **B,** The other side of the patient underwent height resorption that included part of the block bone graft. Bone width was maintained because the fixation screw protected the inferior portion of the graft.

WWW.HIGHDENT.IR

Regional Acceleratory Phenomenon

The RAP is the local response to a noxious stimulus and describes a process by which tissue forms faster than the normal regional regeneration process.[57-61] By enhancing the various healing stages, this phenomenon makes the healing process occur two to 10 times faster than normal physiologic healing. The RAP begins within a few days of injury, typically peaks at 1 to 2 months, usually lasts 4 months in bone, and may take 6 to more than 24 months to subside.[55-57] The duration and intensity of the RAP are directly proportional to the type and amount of stimulus and the site where it was produced. For bone injuries, the degree of remodeling activity varies depending on the extent of bone injury, the quantity of soft tissue involved in the injury, and the configuration of the bone fracture or trauma.

Noxious stimuli of sufficient magnitude, such as fractures, mechanical abuses, and noninfectious inflammatory injuries (including dental implant procedures), can evoke an RAP. Bone grafting surgery and internal fixation procedures also produce an RAP.[57] In animals, an RAP has been shown to exist after mucoperiosteal surgery on mandibular bone, with a clear correlation between quantity and quality of the noxious stimuli and degree of the RAP response.[62-64] When bone graft augmentation is required on both the buccal and lingual aspects, the RAP is created on both sides of the ridge. The host site during a bone graft procedure should be decorticated by drilling holes in the cortical bone. These holes provide access for trabecular bone blood vessels to the graft site, expedite revascularization, and bring growth factors to the graft site.[65,66] The marrow provides differentiated cells that evolve into osteoclasts and osteoblasts. The surgical trauma increases the RAP process, which, among other factors, includes the platelets released from the damaged vessels, which release PDGF and TGF, and increase the availability of osteogenic cells in the graft site. The penetrating holes in the cortical plate also improve graft union to the host bone, which is important when implants are placed within the seam of the donor and host site region.

The host bone decortication process may use a 20:1 low-speed hand piece at 2500 rpm with a bone drill designed for fixation screws to perforate the host bone with holes 3 to 5 mm apart and should be performed under copious amounts of saline irrigation to prevent surgical heat trauma, which delays healing (Figure 36-11). Excess heat transfers several millimeters within the bone and causes thermal necrosis, which could

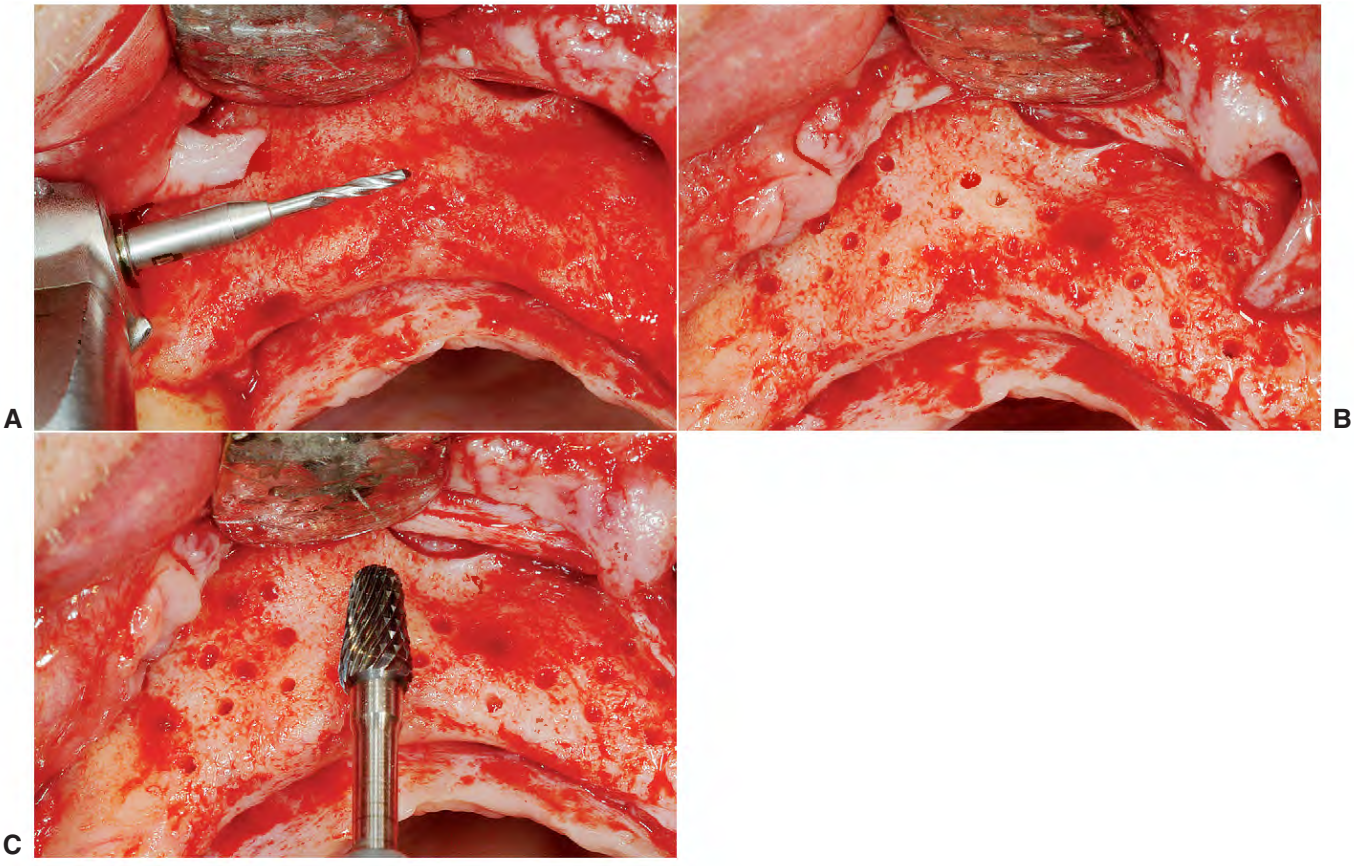


Figure 36-11 **A,** A low-speed hand piece (2500 rpm), similar to a root form osteotomy, may use a bone drill designed for fixation screws, with copious amount of saline irrigation. **B,** The holes perforate the labial plate every 3 to 5 mm to set up a regional acceleratory phenomenon (RAP) for bone repair at the graft–host bone interface. **C,** After the holes prepare the surface, a large pear-shaped drill may be used to recontour the host site to receive the graft and ensure no fibrous tissue remains on the host bone interface to the graft. This also increases the RAP.

WWW.HIGHDENT.IR

همیار دندانسازان

damage bone and blood vessels needed for repair. When injury to the bone is due to a pathologic process (e.g., arthrofibrosis, neuropathic soft tissue problems, rheumatoid phenomena, secondary osteoporosis, excessive heat), the RAP is either delayed or not initiated, and a complete healing process may not occur. When the RAP is inadequate, the result is a slow callus formation that is replaced by lamellar bone. This process contributes to the formation of biologically delayed unions and nonunion.

The increased rate of new formation of bone caused by the RAP does not result in a change in bone volume. In other words, the RAP in and of itself is usually restricted to bone remodeling.[67] In addition, RAP is more evident in cortical bone because the normal turnover of bone cells is 2% compared with 18% for trabecular bone. Biochemical agents, such as prostaglandin E_1 and bisphosphonate, also appear to facilitate the RAP.[62,68]

The RAP is usually accompanied by a systemic response, defined as the systemic acceleratory phenomenon (SAP), that demonstrates a metabolic response similar to the local response.[69] Inadequate RAP is also associated with several systemic medical conditions, including diabetes mellitus, peripheral neuropathies, regional sensory denervation, severe radiation damage, and severe malnutrition.

Host Bone Blood Vessels

Nutrient blood vessels must provide nourishment to an autologous bone graft to keep the transplanted cells alive. These vessels may arise from two primary sources. The host cortical bone contains very few arterioles, whereas cancellous bone has an intensely vascular network. For the blood vessels to penetrate into the autologous bone graft site, the cortical bone should be perforated or removed. This is especially important in the mandible, where the cortical plate is thicker compared with the maxilla.

The host bone blood vessels that grow into a bone graft are of primary importance for predictable bone augmentation.[70] These arteries can grow rather rapidly, compared with other tissues. Fibrous tissue may grow 1 mm each day, whereas woven bone grows at a rate of 60 µm each day. It would appear then that fibrous tissue would always win the race to fill a bony void. Yet, bone forms in an extraction socket when surrounded by walls of bone. One primary reason is that the blood vessels from the surrounding walls grow rapidly into the void and determine what type of tissue will form in the extraction site. The maxillary sinus region forms bone predictably, almost regardless of the type of alloplast or allograft material. This is in part because the antrum is surrounded by bone, and the primary source of revascularization of the graft comes from the adjacent bony walls.

Blood vessels from bone that enter the graft site provide pluripotential perivascular cells that have the capability to become osteoblasts. Monocysts in the blood form osteoclasts, which precede the blood vessel into the bone graft site by forming cutting cones, which resorb devital bone and graft material. As the osteoclasts resorb the graft material, the blood vessel can grow into the site. As importantly, the sides of the blood vessel carry osteoblasts, but only when the vessel comes from the host bone (Figure 36-12). Not only is the blood vessel needed to help the autograft maintain vitality, it is also needed to repopulate the area with osteoblasts to grow new bone. It has been postulated that the surface fibroblasts, if left to migrate within the graft, not only invade the space, but also may inhibit osteogenesis by contact inhibition.[56]

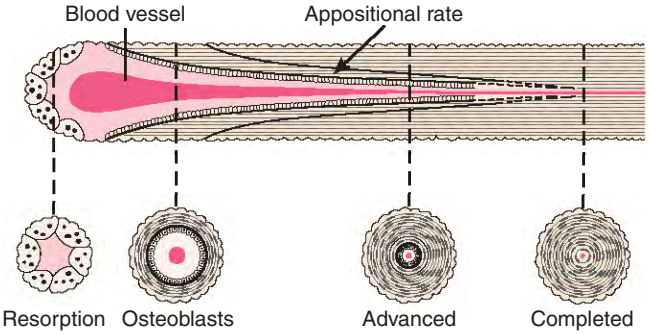


Figure 36-12 When a blood vessel grows into the graft site and comes from bone, several favorable factors occur. The osteoclasts precede the blood vessel, resorb the graft, and allow the blood vessel to grow into the space. The blood vessels from bone bring osteoblasts along the sides to repopulate the graft site with bone-forming cells. Blood vessels from the soft tissue do not populate the graft site with osteoblasts.

A tooth extraction socket fills with bone because the blood vessels from bone form granulation tissue in the site and prevent the epithelial cells from migrating into the site. Four to 6 months are then needed for the socket to replace the area filled with the blood clot (initially) and granulation tissue (later) with bone.[71]

One effective method to increase the amount of host blood vessels in a graft site is to decorticate the host site with a rotary drill.[72] The blood vessels from the trabecular bone are then able to invade the graft site from below and bring pluripotential cells. To permit blood vessels from the bone to enter the graft site, there should be spaces available between the graft particles. When autogenous bone is used as a graft material, the trabecular bone graft provides these open spaces, whereas cortical bone, which presents a denser surface, takes longer to revascularize. Spaces are also necessary when alloplasts are included in the bone graft site.

When an alloplast is placed upon a decorticated ridge and covered with soft tissue (without a barrier membrane), the bone from below the graft grows four times faster into the graft voids than the fibrous tissue grows down into the graft (Figure 36-13). This is because the blood vessels from bone grow rapidly into the region, and once they invade the site, bone follows.

WWW.HIGHDENT.IR

Figure 36-13 A bone graft site is decorticated and a "barrier by bulk" technique is used with an alloplast (in this case particulate hydroxyapatite) and covered with fibrous tissue. After 4 months, a block section was obtained and stained. The soft tissue is stained blue and the bone red. Note the bone grew up into the graft material four times farther than the soft tissue grew down into the graft. This is because the blood vessels grow faster than the fibrous tissue, and when the vessels come from bone, bone cells repopulate the graft and form bone. (NOTE: The same mechanism occurred in Figure 36-8.)

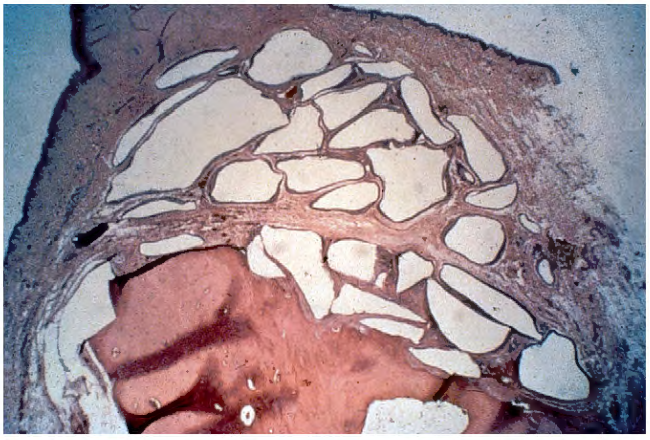


Figure 36-14 An alloplast (hydroxyapatite) placed on top of the bone that is not decorticated was covered with soft tissue and allowed to heal 4 months. A block section reveals the particles are surrounded with fibrous tissue. Because the bone blood vessels do not grow into the graft site, no bone around the particles is observed.

Box 36-1 Growth Factors

1. Platelet-derived growth factor (PDGF)
2. Fibroblast growth factor (FGF)
3. Transforming growth factor (TGF)
4. Insulinlike growth factor (IGF)
5. Platelet-rich plasma (PRP)
6. Bone morphogenetic proteins (BMP)

An alloplast placed upon host cortical bone forms fibrous tissue around the graft, as no host bone blood vessels are able to grow into the graft material (Figure 36-14). The key to whether bone or fibrous tissue forms in the bone graft site is the host blood vessels.

Growth Factors

Bone growth factors can enhance formation and mineralization of bone, induce undifferentiated mesenchymal cells to differentiate into bone cells, and trigger a cascade of intracellular reactions and the release of a number of additional bone growth factors and cell-enhancing factors. These growth factors bind to specific receptors on the surface of target cells. More than 50 known growth factors have been identified and categorized, with some specific to the functions in bone healing (Box 36-1). These constitute a separate group of proteins because of the way they can be produced and their mode of action. They are, however, part of the large superfamily of TGF-β.[73] Bone growth factors are primarily present in bone matrix and released during remodeling or after trauma. They act on the local osteoprogenitor differentiated cells and therefore have limited areas of action.[74] In contrast, BMPs, although also found in extracellular bone matrix, are osteoinductive and can trigger the differentiation of mesenchymal cells into osteoblasts.[75]

Platelet-Derived Growth Factors

Platelet-derived growth factors are produced by activated macrophages and stored in platelets and bone matrix.[76] Platelets are the greatest source of this growth factor and may be further divided into different types, AA, BB (homodimers), and AB (heterodimers). These factors have the characteristics of a wound hormone, acting as a chemoattractant and recruiting mesenchymal cells into the wound.[77,78] The activated platelets also enhance hemostasis by attracting additional platelets to the site, which release thrombin, thromboxane A_2, and adenosine diphosphate. Howes et al. used demineralized bone powder with PDGF in older rats to generate an increased production of mRNA for collagen II, alkaline phosphatase activity, and calcium content of the demineralized bone compared with controls without PDGF.[79] PDGFs increased cartilage and bone formation in the graft site. They have also been shown to activate collagenase within the latter stages of wound healing, which remodels collagen to promote soft tissue wound healing.

The primary roles of PDGFs in bone modeling and remodeling are to (1) increase the number of cells necessary for bone formation (including osteoblasts) at the repair site; (2) trigger capillary formation through its potent mitogenic activity; (3) enhance site debridement;

WWW.HIGHDENT.IR

همیار دندانسازان

and (4) provide a continued source of growth factors for bone repairs.[80] PDGF mixed with autologous bone grafts can accelerate mineralization by as much as 40% during the first year.[79,81-89] PDGF combined with other growth factors (such as IGF)[90] have shown promising results in periodontal regeneration with guided bone regeneration (GBR).[91] Further research targets the use of PDGF combined with IGF to enhance the implant-bone interface.[92]

Fibroblast Growth Factors

Fibroblast growth factors are stored in the extracellular matrix of bone and have many of the same functions as PDGFs. FGFs can stimulate the proliferation of osteoblasts, resulting in the net formation of bone. In addition, they are a potent angiogenic factor.[93] However, FGF-β needs to be in the presence of bone to be effective and is more effective when used in combination with PDGF. Research on the potential benefits of FGF is ongoing but has yielded varied results, with some studies showing bone abnormalities[94] or no bone growth at all.[95]

Transforming Growth Factor Beta

The super family of growth factors is TGF-β, with more than 47 known varieties. TFG-β includes cytokines that contribute to connective tissue repair and bone regeneration. Bone is the body's most abundant storage site for TGF-β, which acts as a weak mitogen for human osteoblastic cells.[93] TGF-β also induces chemotaxis and stimulates extracellular matrix formation in osteoblastic cells and may inhibit osteoclast formation.[93]

TGF-β_1 activates fibroblasts to form procollagen, which deposits type 1 collagen within the wound[77,90] and is therefore credited with the enhancement of soft and hard tissue repair. PDGF and TGF-β_1 assist in soft tissue healing and bone mineralization; therefore they can be mixed with grafting materials into the bone graft site and applied to the top layer of the graft.

Insulin-Like Growth Factors I and II

Insulin-like growth factors I and II (IGFI, II) mimic insulin in several ways. They are fabricated in the liver and travel through the blood stream. IGF has been shown to act as a chemotactic factor for mesenchymal progenitor cells derived from bone marrow.[96]

PRP is a volume of autogenous plasma that has a platelet concentration above baseline[97] (1 million platelets/μL versus normal average of 200,000 platelets/μL). It is a concentrated autogenous source of seven growth factors that are "native growth factors in their biologically determined ratios"[88] (contrary to recombinant growth factors, which are high concentrations of synthesized growth factors in the laboratory) (Box 36-2). It is not osteoinductive by itself, cannot induce bone formation alone, and therefore needs to be in the presence of bone or DFDB. However, it is a benefit to soft tissue healing also, and because soft tissue closure and blood supply are important keys to bone grafting, it is also a secondary benefit even when autologous or DFDB bone is not present.

It has been observed that 90% of the growth factors of PRP are released within 10 minutes of clot activation and the remainder within $1^1/_2$ hours. This is a very short time in the scheme of bone augmentation, which is a 4- to 9-month process. It is to be considered primarily a "spark" that ignites a process of cell enhancement features, which are then able to continue the process.

On the other hand, soft tissue healing is a process that is 90% complete within several weeks. The PRP influence is more noteworthy in this shorter time frame. As a result, the use of PRP should not influence the time of implant insertion into a graft if applied to an implant, nor should not be a major factor is when the device is prosthetically loaded. A study by Gerard et al. in dogs found PRP increased the amount of bone formation in a graft site at the 1- and 2-month time in load.[98] However, at 6 months, the control and PRP bone grafts had similar vital bone percentages (Figure 36-15). However, PRP may decrease incision line opening and enhance soft tissue maturation at a clinical rate that is advantageous to the general scope of the implant procedures for a bone graft.

Box 36-2 Growth Factors in Platelet-Rich Plasma

1. PDGF aa
2. PDGF bb
3. PDGF Fab
4. TGF-β1
5. TGF-β2
6. VEGF
7. Epithelial growth factor

PDGF, Platelet-derived growth factor; *TGF,* transforming growth factor; *VEGF,* vascular endothelial growth factor.

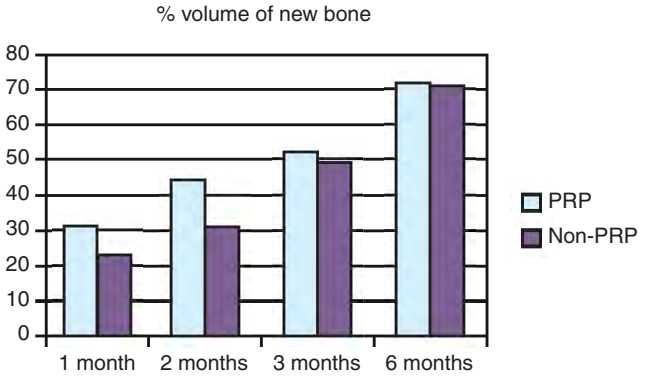


Figure 36-15 A bone graft with platelet-rich plasma (PRP) has greater vital bone cell percent for the first few months, but after 6 months, the control and PRP grafts are similar.

WWW.HIGHDENT.IR

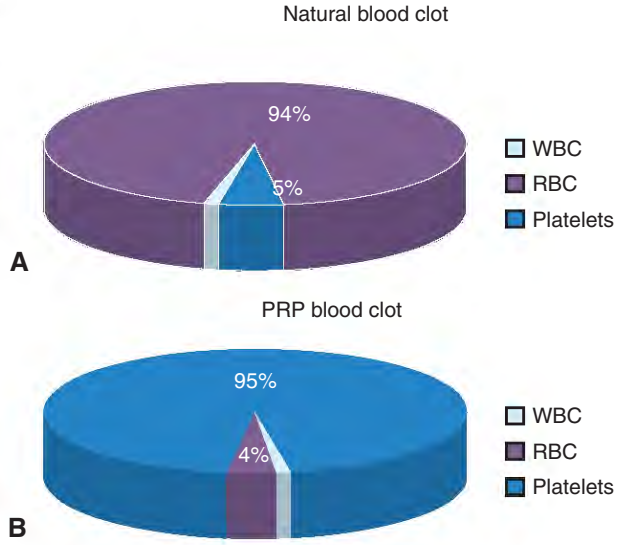


Figure 36-16 **A,** A regular blood clot that forms in the region of surgery is 94% RBC and 6% platelets. **B,** When a PRP concentrate forms a blood clot, 94% are platelets and only 5% are RBCs. *WBC,* White blood cell; *RBC,* red blood cell; *PRP,* platelet-rich plasma.

Preparation of Platelet-Rich Plasma

Specific steps and materials are to be used to produce PRP of a concentration of at least 1 million platelets/μL in a 5-mL volume.[86] Whole blood is primarily composed of red blood cells (RBCs) (almost 50%), white blood cells (WBCs), platelets, and serum. Red blood cells are needed for oxygen and nutrients to the cells. However, stagnant RBCs lyse and the pH is reduced, with the risk to kill vital bone cells.

In a study by Marx, iliac crest osteoblasts were stored in whole blood and in saline 9%. After 3 hours, 97% of the bone cells stored in saline 9% were still alive, whereas only 85% of cells in whole blood were vital. The stagnant RBCs in whole blood withdrawn from a vein or a normal blood clot are a deterrent to bone cell survival.[99] When a blood clot forms, 94% of the clot consists of RBCs, 6% platelets, and less than 1% WBC. In comparison, a PRP clot is 94% platelets, 5% RBCs, and 1% WBCs (Figure 36-16).

The first spin of the double centrifugation technique separates red blood cells from plasma containing platelets, WBCs, and clotting factors. The RBCs are separated from this first process and respun in the centrifuge. The second spin separates platelets and WBCs from the plasma and produces PRP. When the plasma is physically separated from the PRP, the plasma represents the platelet-poor plasma (PPP). One-spin techniques are ineffective at separating PRP from the PPP and produce too low a platelet count. The higher the number of platelets collected, the higher the concentration of growth factors (Figure 36-17).

The PRP factors have a threshold prior to making a difference in the bone and soft tissue healing process. When the threshold amount is not achieved, no difference in clinical result is obtained. Unfortunately,

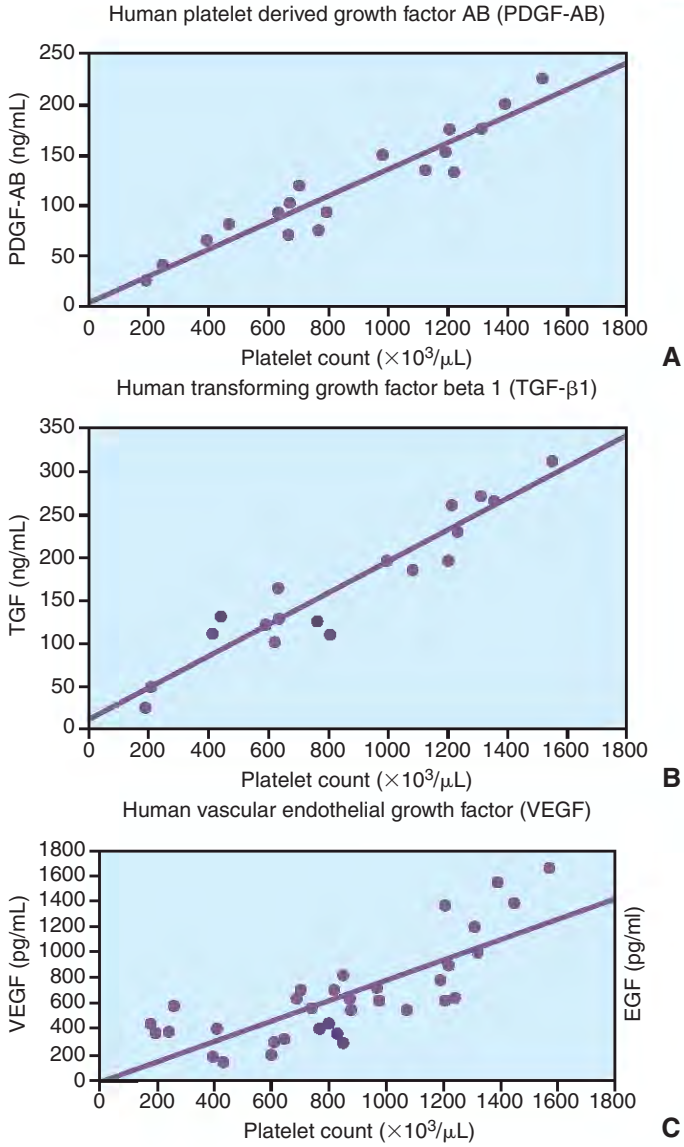


Figure 36-17 **A,** The greater the number of platelets/mL, the greater the amount of PDGF. **B,** The greater the number of platelets/mL, the greater the concentration of TGF-β. **C,** The greater the number of platelets/mL, the greater the concentration of VEGF.

the threshold of making a difference in the healing process is different from one person to the next. The higher the concentration, the more likely a difference will be observed in the rate of healing. The lower the concentration of platelets, the less likely a difference will be observed.

Typically 20 to 60 mL of blood is drawn from the patient to produce a sufficient PRP volume for dental use. Once the PRP is ready for use, it should be clotted (which activates the platelets) at the time of use, because once activated, platelets start secreting growth factors immediately (90% within the first 10 minutes, 100% in 30 minutes). Therefore, an anticoagulant (such as citrate dextrox-A [ACD-A]) is incorporated into the blood at withdrawal from the vein, which may keep the PRP uncoagulated for as long as 8 hours. However,

WWW.HIGHDENT.IR

همیار دندانسازان

PRP is best used when produced and then within 1 to 3 hours.

PRP is not a barrier membrane. Barrier membranes prevent soft tissue from invading the bone graft site for at least several weeks or months. PRP does not prevent fibroblasts from invading a bone graft site over an extended time frame. However, it may be applied to a barrier membrane. Because it also has fibrinogen, it provides added benefits to soft tissue, acts as a hemostatic agent, and has the ability to reduce postoperative pain and edema. Bone formation where PRP is added to the graft may increase in rate, quality, and volume (Figure 36-18).[80-102]

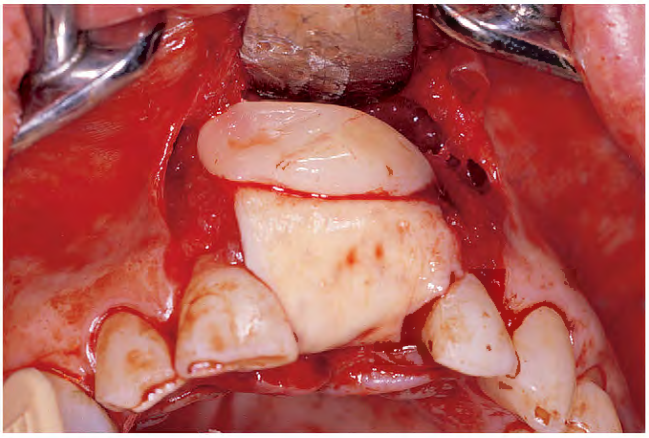


Figure 36-18 Platelet-rich plasma (PRP) is not a barrier membrane, as it does not prevent soft tissue from growing into a bone graft site for several weeks to months. However, it may be applied to a barrier membrane (e.g., Alloderm) and assist in the soft and hard tissue healing process.

Bone Morphogenetic Proteins

Bone morphogenetic proteins are distinct from growth factors in that they can be found in extracellular bone matrix and can induce mesenchymal cell differentiation into chondroblasts or osteoblasts.[75] However, they do not have mitogenic properties.[103] Urist first identified BMPs and showed their role in inducing ectopic bone formation with DFDB.[104-106]

BMP represents a collective term that now regroups more than 15 proteins (BMP-1 through BMP-15), many of which have been purified and cloned. For example, recombinant (concentrated) human BMP-2 (rhBMP-2) and others have been produced and have been shown to induce a complete sequence of endochondral ossification in decreased time, even for large defects.[107-113] Recombinant technology permits the synthesizing of BMP-2 and BMP-7 in larger quantities. A primary advantage is that although isolating BMP from cadaveric bones yields only 0.1 mg BMP per kilogram of bone, rhBMP-2 can be readily produced for widespread use. Areas of application that are being developed for the use of recombinant human BMP include large bone grafting sites, such as the maxillary sinus,[114,115] large periodontal defects,[116-118] and the bone-implant interface.[119,120] Giannobile et al. have reported on novel delivery systems for these BMPs to enhance periodontal and oral rehabilitation.[121,122]

Recombinant BMP-7 (OP-1) has been developed in the orthopedic field for very large bone defects. Most of the literature has concentrated on long bones of endochondral origins, and dental applications appear remote.[123]

In conclusion, there are four methods to increase bone and tissue growth factors during bone augmentation:

1. The PRP collected from the patient's blood may benefit the bone formation process and/or laid on top of the graft and membrane to promote soft tissue healing.
2. The use of autologous bone in the graft site can increase PDGF, FGF, TGF-β, IGF, and BMPs, as all are stored in the bone and released during the augmentation process. The BMP in an autograft primarily has an effect to provide growth factors at 2 weeks and with a peak at 6 weeks.
3. A third method to introduce growth factors at a bone graft site is to use an allograft in the graft site.[90] The DFDB from cortical bone contains a higher percent of BMP than trabecular bone, and therefore is the material of choice. However, the amount of BMP in commercial bone bank allografts is very small (0.001 mg) and is not a very significant factor.
4. A fourth method to increase the growth factors in the graft site is by the RAP process, which triggers a release of growth factors into the site.

All four of these methods are typically used in the bone augmentation process, especially when other key elements are scarce.

Healing Time

Adequate time must be provided for the graft to resorb and regenerate new bone volume. The amount of time required is variable and depends on local factors, such as the number of remaining walls of bone, the amount of autogenous bone in the graft, and the size of the defect. Larger grafts, less autogenous bone in the graft, and fewer bony walls surrounding the site increase the amount of healing time. In addition, systemic diseases such as diabetes, hyperparathyroidism, thyrotoxicosis, osteomalacia, osteoporosis, and Paget's disease may all affect the healing response. It is usually best to err on the side of safety. As a general rule, 4 to 6 months are recommended when graft volumes are less than 5 mm in dimension. Graft volumes more than 5 mm in dimension often require up to 6 to 10 months.

Often, a premature reentry gives the impression the bone graft did not work, and alloplast materials still represent the majority of the graft. Although this may

WWW.HIGHDENT.IR

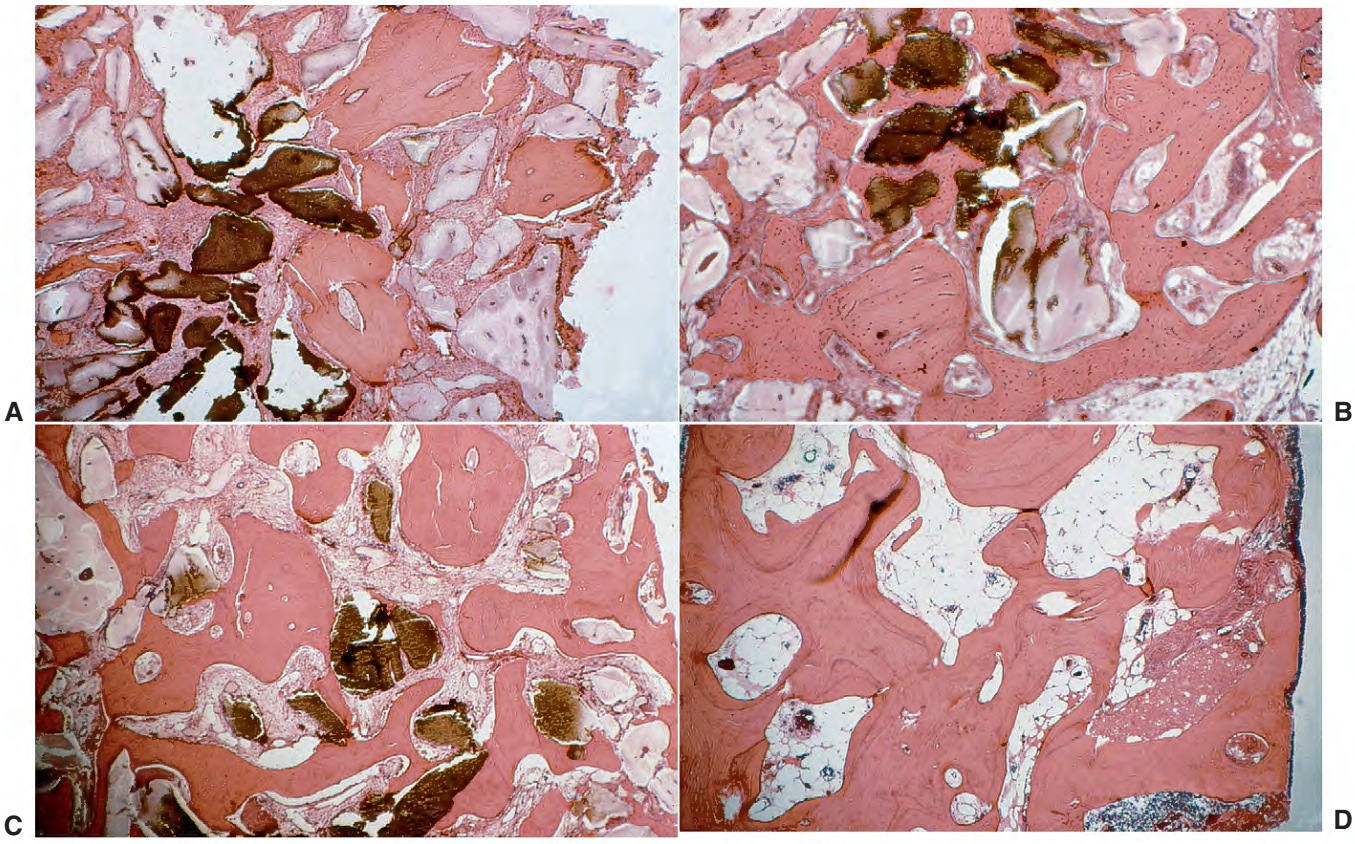


Figure 36-19 **A,** A combined autologous, demineralized freeze-dried bone (DFDB), and alloplast (bovine hydroxyapatite [HA]) graft, was placed as a sinus graft, and a biopsy was taken at 2 months. Most of the core biopsy is allograft and alloplast. **B,** Another core biopsy from the same patient after a 4-month healing period. Note most of the DFDB has been resorbed, and more vital bone is present. **C,** The same sinus graft patient had another core biopsy taken at 8 months. Almost all the DFDB and most of the bovine HA has resorbed and been replaced by vital bone. The bone is beginning to form a trabecular pattern. **D,** A core biopsy taken at 12 months in the same patient and graft site. All the sinus graft has been replaced by vital bone, and the bone structure is similar to a normal posterior maxilla. *(Courtesy S. Wallace.)*

be correct, it is not unusual that an additional healing period of several months would have allowed reentry in a successful graft site (Figure 36-19). Time frames may be shortened when additional keys to bone grafting are incorporated. As a general rule, the surgeon should provide enough keys to bone grafting to permit reentry in less than 8 months. If more than half the graft is from autogenous origin, a prolonged healing period of more than 1 year is often not beneficial and may result in bone resorption of the newly grafted site. On the other hand, some dense bovine calcium phosphate materials may require more than 2 years to be resorbed and replaced by bone.

Defect Size and Topography

The size of the defect is a factor in the healing time, the vascularization, and the transitional prosthetic options. It is also a factor for the graft material selection. The larger the bone defect in width and height, the longer the period of bone maturation before implant insertion. The larger the bone defect, the more autologous component needed in the graft. This relates to the creeping resorption and vascularization requirement for a larger graft site. The larger graft sites may require a removable restoration, as fixed transitional spans of four or more teeth are very prone to fracture and uncementation.

On the other hand, when a graft site is small, it is not unusual to insert the implant in the correct position and graft the site at the same time as implant insertion. When this is considered, the implant most often is not placed in function but is left to heal with an alloplast and primary soft tissue closure. When an implant is uncovered and a small defect is present, most often the graft site is augmented and the permucosal element or abutment is added. The implant may be restored with a transitional prosthesis out of occlusion, if it is rigidly fixated and fulfills all other requirements. The implant should not be loaded because crestal marginal stresses are greater under load. A bone graft at the crest of the ridge will not perform as predicted during the modeling/remodeling process that occurs during early bone formation.

The topography of the graft site is also a key for predictable bone augmentation, as it affects soft tissue closure, space maintenance, graft immobilization, vascularization, growth factors, BMPs, and healing time—in other words, almost every aspect of the equation. When

WWW.HIGHDENT.IR

همیار دندانسازان

the vital bone of an extraction socket is 1.5 to 2 mm thick or greater on the facial, lingual, mesial, distal, and apical regions, a 5 bony wall defect is present. This is an ideal environment for bone growth, as most all the keys are already present. The space will be maintained by the surrounding walls of bone and the graft is immobilized by the bony walls. Growth factors, BMPs, and RAP are released from the periodontal complex and walls of bone as a result of the extraction. As a result, bone grows in the site, even without initial soft tissue closure over a graft material.

On the other hand, when a bone augmentation for height and width is necessary, as for an onlay graft in a Division D bone volume host site, almost no keys are present for the augmentation. This condition represent a one-wall bony defect that needs 12 mm in height and 6 mm or greater in width and requires the surgeon to provide as many key factors as possible. In addition, the bone graft material in these unfavorable conditions must be primarily autologous cortical and trabecular bone—in other words, the number of walls of host bone and the size of the defect combine to affect almost all aspects of the bone augmentation process.

In the periodontal literature, it is well documented that a three-wall bony defect next to a tooth root can be restored more predictably than a two-wall bony defect. Likewise, a three-wall bony defect in an edentulous site can be augmented better than a two-wall defect. Most often, a three- to four-wall defect in implant dentistry corresponds with a lack of facial bone. The bone is present on the lingual, mesial, distal, and apical regions (four-wall defect), the apical region is too narrow or compromised (three-wall defect), or the bone defect is next to a tooth root (Figure 36-20). Under these conditions, soft tissue closure, space maintenance, and graft immobilization become more critical. A barrier membrane and longer healing time are often necessary. The graft material in a three- or four-wall defect does not require only an autograft as the major component, although it is of benefit. The number of bony walls remaining around the defect is a key to predictable augmentation (see Chapter 37).

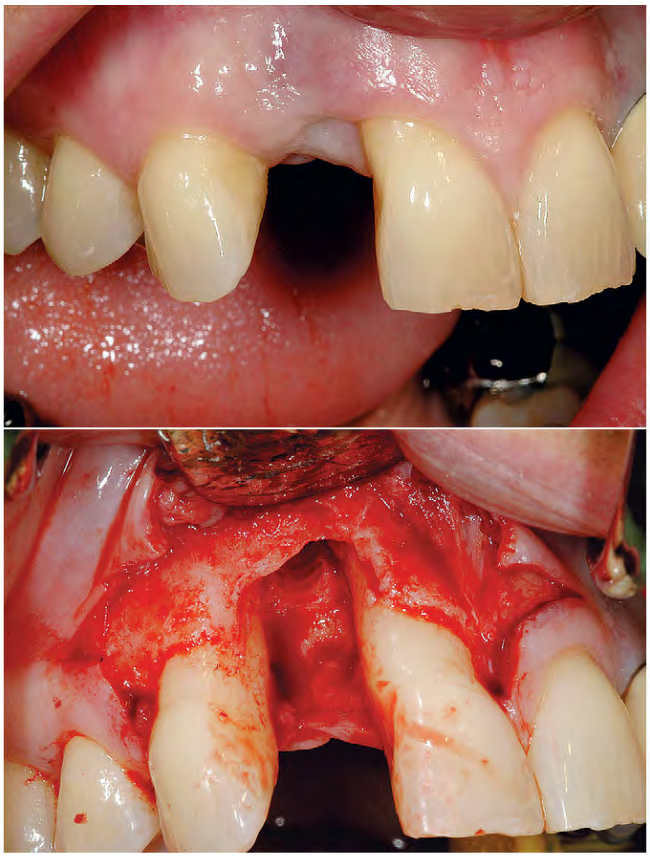


Figure 36-20 **A,** A bone defect is observed between two teeth. **B,** A bony wall is present on the palatal and apex. No bone is on the mesial, distal, or buccal. This two-wall bony defect will be difficult to cement because few keys are present.

Transitional Prostheses

The transitional prosthesis worn during the soft tissue healing and maturation of the bone is a critical component of predictable bone grafting. The transitional restoration affects the keys of soft tissue closure, the maintenance of space for the augmentation, and the immobilization of the graft during healing. In addition, the restoration often restores some esthetic and functional components for patients during the many months required for the maturation of the bone. It may also help contour the soft tissues and allow maturation before the final fabrication of the prosthesis. The value of transitional restorations should not be underestimated from either a patient management or a predictable outcome perspective.

Whenever possible, a fixed restoration that does not rely on soft tissue support in the area of the bone graft is preferred. There are several options to accomplish this goal. When natural teeth are in position around the graft site and require restoration, a transitional acrylic fixed partial denture may use the natural abutments and pontics span the site of the augmentation (Figure 36-21). Whenever the pontic span is greater than two teeth, consideration is given to metal reinforcement, as the risk of fracture exponentially increases. Other options include increasing the bulk of acrylic (twice the thickness decreases fracture risk by half) and eliminating occlusal forces over the pontics.

When natural teeth are present adjacent to the augmented sites but do not require restoration, a resin-bonded prosthesis may be fabricated. These are designed gingival to the occlusal contacts, so the teeth do not require preparation. However, this increases the risk of debonding, so a secondary removable device may be delivered and used as an emergency until the patient is able to return to the office for rebonding of the restoration. These resin-bonded prostheses should use

WWW.HIGHDENT.IR

Figure 36-21 **A,** A provisional prosthesis with a pontic was used to restore the missing tooth over the grafted site to provide an undisturbed healing environment. **B,** A fixed transitional prosthesis is the best treatment option during graft maturation.

acrylic denture teeth so that they may be modified (add or subtract) at the time of surgery and during the healing process.

When natural teeth are not present adjacent to the bone graft site, a fixed temporary may be fabricated on a small diameter and temporary implants placed in the lingual plate or in sites away from the bone graft. These implants are often inserted at the same time as the bone graft and immediately restored with an acrylic, fixed, transitional prosthesis. These restorations should be out of occlusion in partially edentulous patients and have no posterior cantilevers out of the esthetic zone in completely edentulous patients (Figure 36-22).

The transitional implants should engage as much cortical bone as possible and therefore are usually longer than implants with a delayed loading protocol. At the reentry and final implant placement, the transitional implants are often removed. At this point, the final implants may be immediately loaded to support a modified transitional restoration, or a removable prosthesis may be fabricated. The risk of micromovement on the soft tissue over the graft site is less, as the graft has already matured 4 to 8 months before the implant placement. There is a higher risk of implant failure with transitional implants, which, as a consequence, may cause loss of graft volume. Therefore other transitional methods, when possible, are suggested.

A removable restoration may be fabricated over the bone graft but should be designed to not load the soft tissue over the graft site. When teeth are present, a cast metal framework with direct and indirect retainers and rest seats may be fabricated. Before the removable partial denture framework design, a stone cast of the patient's mouth is augmented with clay or wax in the laboratory in the sites of the future bone graft. In addition, the framework does not have metal mesh over the bone graft site. Otherwise, the surgeon most often must remove the chrome cobalt mesh, as the laboratory often does not provide enough relief between the augmented site and the metal framework (Figure 36-23).

Box 36-3 Bone Grafting Materials

- Collagen
- Autologous bone (osteogenesis)
- Allografts DFDB, FDB (osteoinduction)
- Xenografts (osteoconduction)

BONE GRAFT MATERIALS

In addition to the keys needed to develop a predictable bone augmentation site, there are materials necessary to augment the location. Bone graft materials and their mechanism of action are not all the same. The materials most often used in implant dentistry to aid in bone augmentation include: (1) collagen, (2) human DFDB, (3) human freeze-dried allograft (FDB), (4) xenograft bone, and (5) autogenous bone (Box 36-3).

Collagen

Several types of collagen are found in the human body. Type I collagen is among the first products synthesized by the body when bone formation occurs.[124,125] The irregular pattern of initial bone formation (woven bone) is a result of the rapid, unorganized response of the body to lay down collagen, which is then invaded and mineralized with HA along the direction of the collagen fibers by osteoblasts. The haphazard organization of collagen results in unorganized bone formation, called *woven bone*, which forms first and at a faster rate and is less strong. The most common source of collagen in implant dentistry is bovine collagen from the "Achilles" tendons in the leg. Another source of type I collagen is DFDB, which can provide the space necessary for blood vessel ingrowth into the graft and may contribute to the bone formation process. However, DFDB alone has not proven an effective graft material and should

WWW.HIGHDENT.IR

همیار دندانسازان

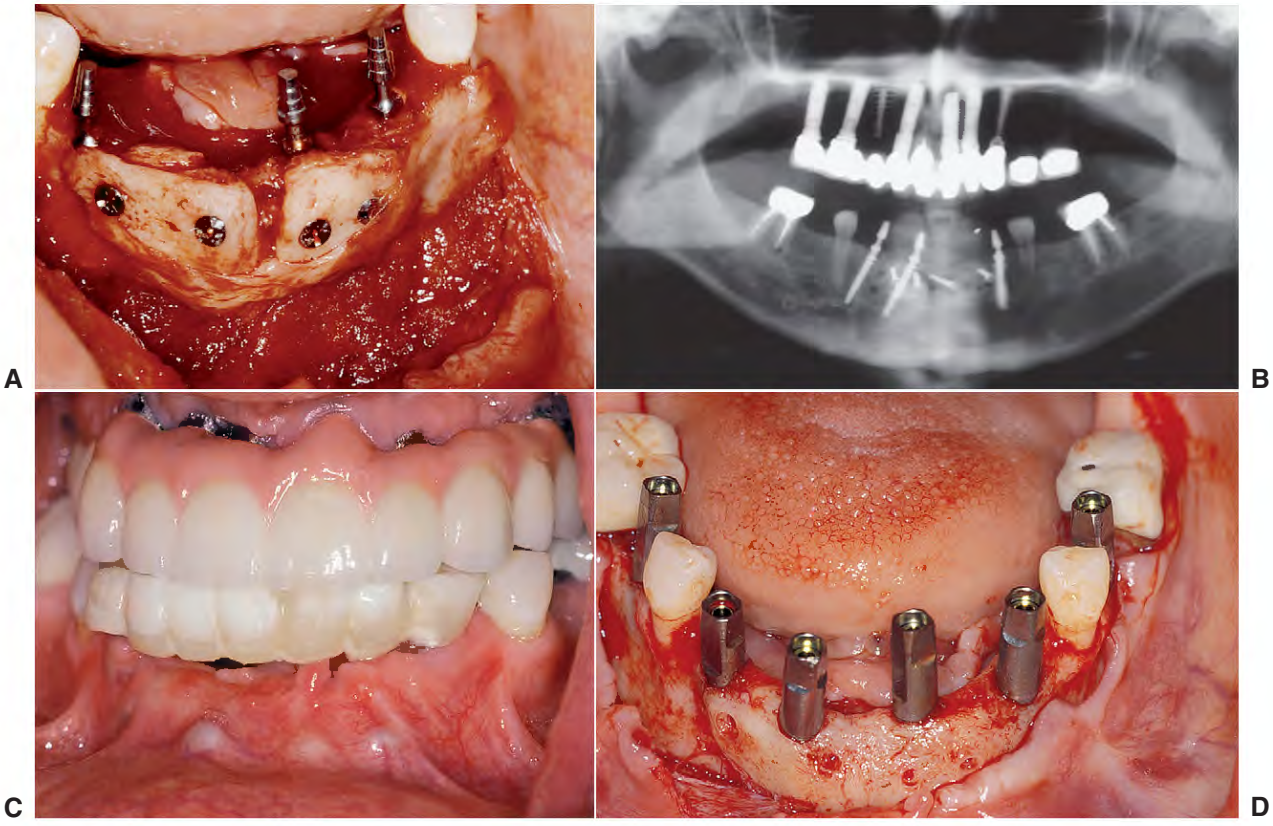


Figure 36-22 **A,** A block bone graft is placed in an anterior mandible. Small-diameter transitional implants are inserted into the lingual plate. **B,** A postoperative panoramic radiograph of the mandibular block bone graft and three transitional implants. **C,** A transitional prosthesis on the small-diameter implant is out of occlusion during the healing period. **D,** After bone graft maturity, the transitional implants are removed and the final implants are inserted.

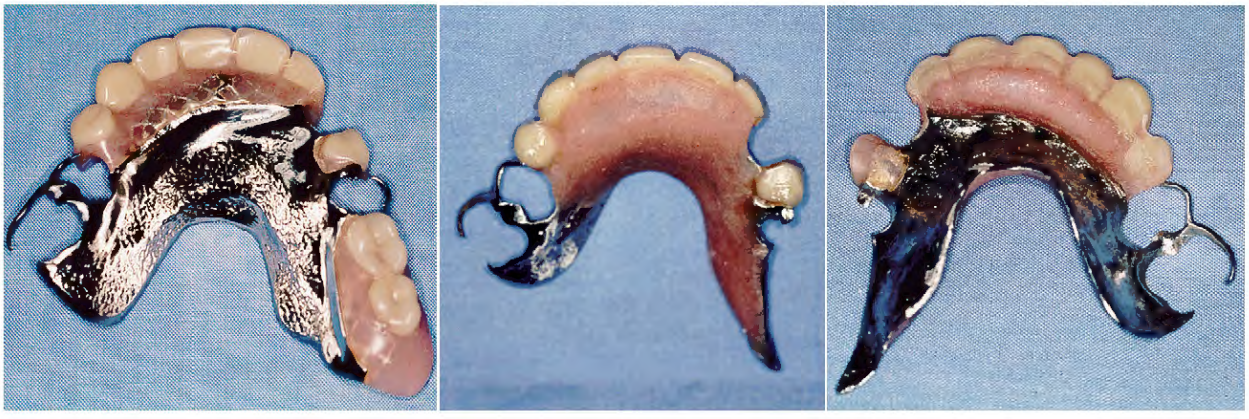


Figure 36-23 **A,** A removable partial denture with rest seats and clasps may be modified over a bone graft. **B,** Acrylic is added to the external side of the prosthesis to increase the thickness over the grafted site. **C,** The framework is then relieved to ensure a lack of pressure on the grafted site.

be combined with other categories of bone-grafting materials.

Collagen also is an integral part of the soft tissues with chemotactic and hemostatic properties. It can bond and activate platelets to form a platelet plug within the vessel. It may also act as a scaffold for migrating cells of the epithelium. In the application of bone regeneration, collagen may be used at the level of the soft tissue to accelerate healing over an extraction site or to promote coagulation of a bleeding surgical site.

Over the last decades, several collagen barrier membranes have been introduced for guided bone regeneration. Processed bovine type I collagen membranes (from tendons and dermis) have yielded favorable results to decrease the invasion rate of epithelial cells or fibroblasts into a graft site. Resorption rates of collagen

WWW.HIGHDENT.IR

barrier membranes are variable, with ranges from a few months to 1 year, and are often intended to be maintained for the whole bone regeneration cycle.

Autologous Bone and Osteogenesis

An important key for predictable bone augmentation is the presence of autogenous bone as a component of the graft. Autogenous bone is the only graft material that directly forms bone from transplanted trabecular bone cells. The autogenous graft also contributes to bone growth with several growth factors (e.g., BMPs) that are released into the environment during incorporation of the graft and form bone through induction. The autograft, which becomes nonviable bone, acts as an osteoconductive matrix of calcium phosphate for bone growth by creeping substitution (Figure 36-24). The space requirements of grafting may also be fulfilled by the autogenous bone because its physical volume maintains the contours of the desired augmentation. The growth factors also help the soft tissue healing on the outside and accelerate blood vessel growth into the graft site of the host bone on the inside of the graft.

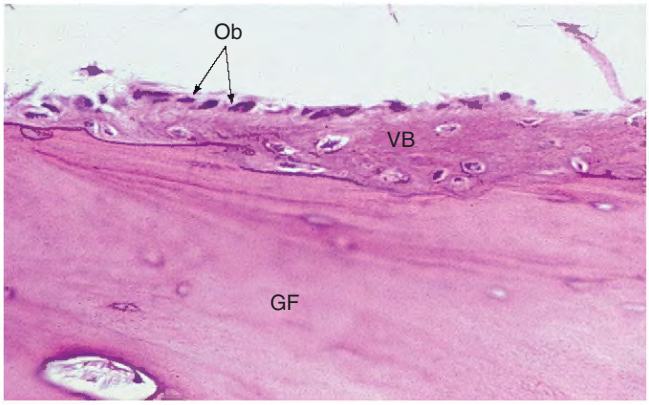


Figure 36-24 A bone biopsy of an iliac crest bone graft after 6 months of healing. Most of the autograft is devital. Bone is growing into the graft around the newly formed blood vessel. As the bone graft resorbs, it is replaced by new, vital bone (creeping substitution). *Ob,* Osteoblasts; *VB,* vital bone; *GF,* graft (autograft).

Osteogenesis

Osteogenesis refers to the growth of bone from viable cells transferred within the graft. Autogenous bone is the only graft material available with osteogenic properties. There are more osteoblasts in trabecular bone than cortical bone. New bone may be regenerated from endosteal osteoblasts and marrow stem cells transferred with the graft. This is not to say cortical bone is not an effective graft material. In fact, the author has observed that the cortical bone grafts from the mandibular ramus and symphysis and the cortical bone grafts from the iliac crest form more volume of bone than trabecular bone grafts from these regions. Apparently, cortical bone, although less rich in osteoblasts, has great osteoconductive properties and bone growth factors.

For the transplanted autogenous bone graft to produce osteoid, it must remain vital. Vitality of the graft may be improved in several ways. The recipient site is prepared first so that minimal time elapses between graft harvest and placement. It is then placed into an environment that provides for prompt angiogenesis to maintain its viability, rather than mixed with other synthetic graft materials, which impair blood vessels to reach the autogenous bone graft (Figure 36-25). As a result, the optimal place for the autologous graft is directly on the host bone after the cortical bone of the host site has been decorticated with a surgical bur (Figure 36-26).

Once harvested, the autologous graft should be used immediately (which is preferable) or stored in sterile saline, lactated Ringer's solution, or D_5W to maintain cell vitality. Distilled water is contraindicated as a storage media, as the hypotonicity results in cell lysis. Storing bone grafts in blood is also detrimental to cellular

Figure 36-25 The harvested autograft should not be mixed with other alloplasts or allografts, but should be placed on bone for early vascularization to minimize autograft cell death.

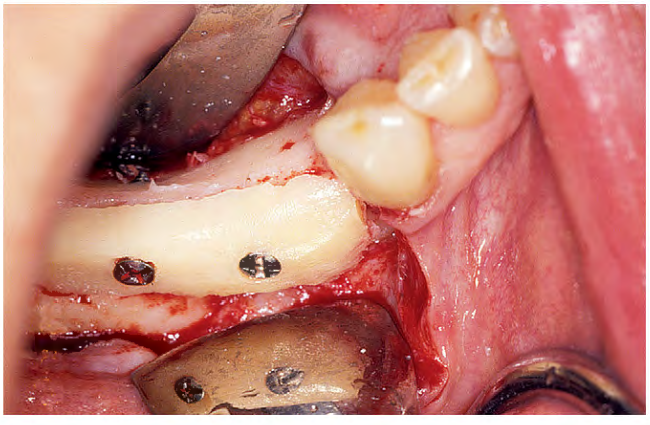


Figure 36-26 An autograft should be laid directly on top of the host bone. This releases growth factors, including those required for early vascularization, which is needed to maintain the vitality of the bone graft.

WWW.HIGHDENT.IR

همیار دندانسازان

survival, as red blood cells release several cytotoxic products once the cells lyse.[98] Because the autogenous graft material may require an additional operative site, it is usually selected when conditions for growth of bone are less ideal or in combination with other allografts or alloplasts.

The mechanism of bone growth within autogenous bone grafts includes three phases. Some transplanted osteoblastic cells survive the first 3 or 4 days through nutrition from the surrounding vascular tissue. The osteoblasts and stem cells at the surface of the bone graft from either trabecular or cortical bone that survive the transplantation process are responsible for proliferation and formation of new osteoid product.[126] This osteogenic process, referred to as *phase I bone*,[127] is related to the number of cells transplanted and initially dictates the amount of new bone that will directly form beyond the original dimension. Most of the osteocytes within the mineralized cancellous bone die because they cannot directly access nutrients (Box 36-4).

Blood vessels can grow into a graft site with almost the same speed as fibrous tissue, at approximately 1 mm per day. The graft success, therefore, depends on early vascularization.[128] In the hypoxic environment of the recipient bed and graft, macrophages secrete macrophage angiogenesis factor (MDAF) and macrophage-derived growth factors (MDGF). Platelet degranulation releases PDGF, which also initiates angiogenesis.[127] This process is directly proportional to the density of cells transplanted.[129] Therefore, to increase the transplanted cell volume when trabecular bone is harvested, the overall volume of the graft is packed into a syringe and compressed to provide as many bone cells per area as possible.[130]

The fresh autogenous trabecular bone that provides the survival of the maximum number of transplanted bone and undifferentiated cells for large grafts is usually harvested from the ilium. However, only osteoblasts within 300 microns of the blood supply within the first 1 to 2 weeks will survive, whereas all others die before adequate nutrition can reach them by diffusion.[127] Because blood vessels may grow 1 mm per day, only the autologous graft less than 7 to 10 mm thick will survive the transplantation.

Box 36-4 Autogenous Bone

Bone Blood Supply

Phase I: Osteogenesis—bone regeneration
Surviving cells 4 weeks (osteoid)
Phase II: Osteoinduction
BMP release 2 weeks to 6 months; peak at 6 weeks
Phase III: Osteoconduction
Inorganic matrix–space filler
Phase IV: Cortical plate, barrier membrane

BMP, Bone morphogenetic protein.

In the 3 to 7 days after bone grafting, stem cells and endosteal osteoblasts produce only a small amount of osteoid. Production increases as soon as oxygen and nutrients can be brought by the newly developed bloodstream. Therefore, initially, primary osteoid develops between the trabecular bone. New bone consolidation also occurs during this phase, which produces an unorganized woven bone.

Demineralized Freeze-Dried Bone and Osteoinduction

Osteoinduction involves new bone formation from osteoprogenitor cells derived from primitive mesenchymal cells under the influence of one or more inducing agents that emanate from the bone matrix.[131-133] When an osteoinductive material is placed subcutaneously in the absence of bone or within a muscle, it has been shown to induce bone formation in the ectopic site. Landmark work in this particular class of bone graft materials has been performed over the years by Urist et al. to identify the osteoinductive factors and their mode of action.[104,134-136]

The first phase of the bone formation process (osteogenesis) diminishes within 4 weeks. As the transplanted bone cells die, osteoclasts from the host tissue precede the invading blood vessels and remodel the graft by resorption. Inductive proteins and growth factors are then released from the transplanted bone and initiate the phase-II osteoinductive process. Stem cells in the graft and BMPs mostly from cortical bone also contribute to osteoblast differentiation and new bone formation and result in phase II bone, which is less cellular, more mineralized, and organized. This phase begins after approximately 6 weeks and lasts as long as 6 months.

The most commonly used osteoinductive materials for bone augmentation in implant dentistry are bone autografts and allografts. A bone allograft is an osseous, transplanted tissue from the same species as the recipient but of different genotype. Therefore, when performing grafts on humans, cadaver bone must be used. However, when using allograft for animal studies on dogs, dog bone must be processed. The main advantage of allografts is that they eliminate the need for a donor site. In addition, their unlimited availability permits their use in large quantities if necessary. However, larger grafts most often require other material to make the bone augmentation predictable. Osteoinductive materials are more contributory to bone formation during the remodeling process.[134] The tissue is processed and then stored in various shapes and sizes in bone banks for future use.[137] There are primarily three types of bone allografts: frozen, freeze-dried, and demineralized freeze-dried.

Frozen bone is often harvested, frozen, and stored for use on the same patient at a later date. It may also

WWW.HIGHDENT.IR

be irradiated to decrease the immune reaction when used for a different recipient. Allograft frozen bone is rarely used in implant dentistry because of the risks of rejection and disease transmission. It has often been used in biomechanical analyses to determine the physical properties of bone found in the jaws.

The most common allografts used in implant dentistry are DFDB and FDB. The process to form FDB and demineralized DFDB is different and specific, although some variations among laboratories exist.[136] In both allografts, cortical and trabecular bone is harvested in a sterile fashion from a disease-free donor. The bone is then washed in distilled water and ground to a particle size of 500 µm to 5 mm. It is then immersed in 100% ethanol to remove fat, frozen in nitrogen, then freeze-dried and ground to smaller particles (250 to 1500 µm), which has been shown to promote osteogenesis.[136] The desiccating step allows for long-term storage and decreases antigenicity. This bone process results with FDB. Solvent-preserved products have been developed instead of freeze drying to reduce antigenicity and still assure a minimal risk of contamination.[128-140] The inorganic and organic matrix is therefore maintained because the calcium and phosphate salts of HA remain.

The organic material in bone, which includes bone morphogenetic proteins, is found within the structure of the HA. However, osteoclasts are required to resorb the bone to release its bone growth factors. This delays the release over a longer period. Coupled with the fact that very little growth factors are present, this results in a graft material that is primarily an inorganic mineral source of HA, which serves as scaffold for bone formation. Therefore FDB acts primarily through an osteoconductive process, because inductive proteins are slowly released after resorption of the mineral and often found in only minute quantities (Table 36-1).

DFDB and FDB have a similar initial processing step, but DFDB is produced by an additional step, demineralizing the ground bone powder in 0.6-N hydrochloric or nitric acid for 6 to 16 hours. The BMP is not acid soluble, but the calcium and phosphate salts of HA are acid soluble and therefore are removed from the bone in the acid-reducing process. As a result, the demineralization of the freeze-dried bone more readily exposes the BMPs.[141,142]

Completely demineralized grafts have been shown to stimulate more bone induction than partially demineralized material.[143] Because the mineral salts are removed from the bone, the nonsoluble BMPs are available in the local environment earlier than with freeze-dried bone, which requires their release by osteoclastic activity. As a result, more undifferentiated cells may transform earlier into osteoblasts.[144,145]

Therefore FDB is primarily osteoconductive and DFDB is not osteoconductive (as it has no mineral) but is believed to be more osteoinductive.

After washing and dehydration of the DFDB after the acid bath, it is often either irradiated or sterilized in ethylene oxide (EO) to further decrease the antigenicity and to protect the host from disease transmission. The use of irradiation is controversial, and it is generally agreed that doses greater than 2.5 Mrad of irradiation are destructive to the BMPs and, therefore, to bone formation.[146] EO is also controversial, and it is recommended to limit it to a 5-hour EO sterilization process at 29° C to maintain osteoinductive properties.[147]

Several tests are performed to evaluate the safety of the allograft and to ensure that the acid demineralization process destroys all pathogens.[148-150] The probability that a particular graft of DFDB might contain human immunodeficiency virus has been calculated to be 1 in 2.8 billion,[151,152] and to the author's knowledge, no occurrence has been reported in the literature. Because some reports suggested that a dose of irradiation or EO sufficient to kill spores may render the BMP in the allograft unable to induce bone formation, it may be beneficial to use DFDB bone that is harvested from soft tissue donors with no history of disease transmission and no preparation history of radiation or EO.[148] Results of studies using DFDB for bone regeneration are conflicting. Several reports conclude that DFDB exhibits osteopromotive properties, whereas others question such benefits.[34,35,153-158] In particular, some reports raise the question of variability of BMP concentration and activity in commercially available allografts, and therefore

Table 36-1 Allografts

MATERIAL	COMMERCIAL SOURCE	COMPOSITION	BONE GROWTH METHOD	RESORPTION TIME/%
DFDB (Demineralized)	Pacific Tissue Bank Grafton MTF DynaGraft	Collagen + growth factors	Osteoinduction varies based upon processing method	+/– 6 months
Autologous bone				
FDB (mineralized)	MinerOss Puross	Minerals + collagen	Slight osteoinduction + conduction	1 yr+

WWW.HIGHDENT.IR

همیار دندانسازان

qualify them as less predictable.[139,159] Product fabrication and formulation are critical in ensuring the quality and osteoinductive properties of the product.[160-164] The osteoinductive properties of DFDB are variable from one cadaver source to another. As a general rule, the younger the cadaver, the more BMPs available in the bone.[165] The demineralized cortical bone mineral contains a higher concentration of BMPs than trabecular bone and is recommended. In addition, membranous cortical bone exhibits greater concentrations of BMP than endochondral cortical bone, so the skull represents a better source of inductive proteins than the rest of the skeleton.

Some studies that substantiate the osteoinductive capacity of DFDB have been performed with materials enriched with rhBMP-2. These studies may have as much as 500 to 1200 mg of BMP compared with 0.001 mg found in a 5-g vial of DFDB from a bone bank; therefore the results may not be reproducible in clinical practice.[108,166,167] This explains some of the wide variability of the results discussed in the literature related to DFDB.

The particle size of DFDB may also affect its efficacy. Sizes smaller than 150 µm are less effective than those of 250 µm or larger. Fibers of cortical bone (e.g., Grafton) are more effective than particles. When a bone matrix fiber forms new bone, the osteoid material creeps along the length of the fiber. When a particle forms new bone, it appears localized to the particular spot (Figure 36-27). On a histologic slide, 1 DFDB particle out of 10 to 20 appears to form new bone, whereas more fibers appear to participate more often in the inductive process and act as a wick to promote bone formation along the entire length of the cortical fiber. More recently, putty consistency products have been introduced that facilitate delivery and use of the material intraorally. The fillers used to create the putty do not participate in the bone formation process and serve only to make the product more user friendly. The bone fibers in this preparation provide a scaffold for new bone growth and have been shown to be more inductive than particle forms of the DFDB.

Calcium Phosphate Minerals and Osteoconduction

It is postulated that the inorganic matrix of HA, which forms a scaffold in the autogenous graft, contributes the osteoconductive effect of bone formation as new bone forms by creeping substitution. This may be considered a third phase of bone formation by autogenous bone[48] (see Box 36-4).

Osteoconduction, which characterizes bone growth by resorption or apposition from the surrounding bone, has been called *creeping substitution*. Therefore this process must occur in the presence of bone or differentiated mesenchymal cells. Osteoconductive materials do not grow bone when placed into subcutaneous tissues, muscles, or fibrous tissue. Instead, the material remains relatively unchanged or resorbs. Osteoconductive materials are biocompatible, and bone or soft tissue can grow adjacent to them by apposition without evidence of a toxic reaction. The most common osteoconductive bone grafting materials used in implant dentistry are allografts, alloplasts, and xenografts. Allografts such as FDB already have been addressed. Recently, allografts that maintain the inorganic portion of bone (versus the organic components, such as in DFDB) have gained acceptance as the osteoconductive portion of the graft. Alloplastic materials are exclusively synthetic, biocompatible products developed to cover a broad range of indications. They come in a great variety of textures,

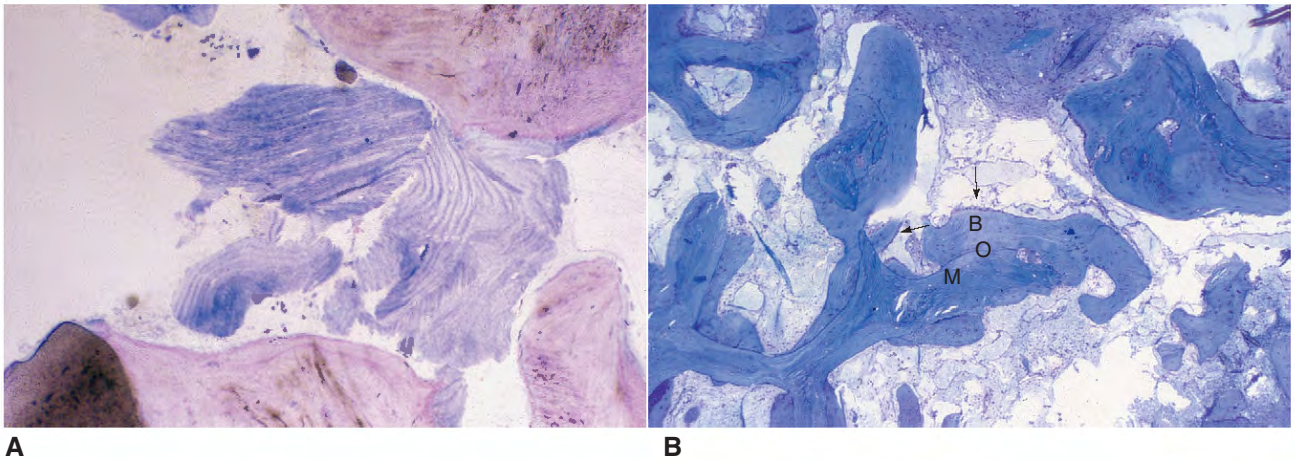


Figure 36-27 **A,** A particle of demineralized freeze-dried bone (DFDB) may be inductive and contribute to the bone graft. However, it has been observed by the author that very few particles participate in the inductive process, and marginal new bone formation next to a particle is observed. **B,** A cortical fiber of DFDB (Grafton) in a histologic section after a similar time frame elicits greater bone formation, more fibers participate in the inductive process, and greater new bone contact is observed along the length of the fiber. *B,* New bone formation; *O,* initial bone formed by induction; *M,* matrix of DFDB fiber (Grafton).

WWW.HIGHDENT.IR

Table 36-2 Alloplasts

TYPE	MATERIAL	BRAND NAMES	STRUCTURE PARTICLE SIZE	RESORBABLE	RESORPTION TIME/%
Ca phosphates	HA synthetic HA natural	Osteograf D	Crystalline HA	N	–
	Bovine bone derived	Bio-Oss	Cancellous (0.25-1.0 mm) or cortical (1-2 mm)	Y	1.5 to 2.5 yr+/100%
		Osteograf N	Micro + macroporous N-300 = 250-420 µm N-700 = 420-1000 µm	Y	1.5 to 3 yr+/100%
		PepGen P-15	Peptide + microporous HA	Y	↓ time for osteoconduction 1.5 to 3 yr+/100%
	Allogeneic bone (FDB)	Puros MinerOss	Mineral + collagen	Y	± 6 mo to 1 yr
	TCP	Cerasorb Curasan	βTCP micropores and macropores (10-65 µm) TCP + crystalline HA Round particles (50-150µm, 150-500µm, 500-1000µm, 1000-2000µm)	Y	3-24 mo/100%
Ca carbonate	Coral	Interpore 200 (coralline)	Porous HA Blocks-granules	±	Very long
		Biocoral	Natural coral aragonite 98% $CaCO_3$	Y	5-7 yr+/partial
Bioactive glass ceramics		Bioglass	Ca salts + P + Na salts + silicone	N	7 yr+
		Perioglass	Ca, P, Silicone, Na 90-710µm	N	5 yr+
		Bioglass	Ca, P, Si, Na 300-355µm	Y	
Ca sulfate		Capset	$CaSO_4$ (Plaster of Paris)		
Polymer		BioPlant HTR	Composite polymer + Ca hydroxide Open spheres 750µm	Y/N	Very long; 4-5 yr Partial

HA, Hydroxyapatite; *TCP,* tricalcium phosphate; +, more than; –, less than; ± approximately, *FDB,* freeze-dried bone.

particle sizes, and shapes (Table 36-2). They may be separated into ceramics, polymers, and composites. The most frequently used are ceramics, which may be characterized as bioinert (e.g., aluminum oxide and titanium oxide) or bioactive (e.g., calcium phosphate). Bioinert ceramics exhibit direct bonding with the host bone (at the light microscopic level) and are mechanically held in contact to the bone. The healing of bone around a bioinert osteointegrated implant is an osteoconductive process, which follows typical phases of remodeling at the bone-implant interface.

Bioactive ceramics are the largest family of alloplasts used for bone augmentation and include calcium phosphate products such as synthetic HA and bovine-derived anorganic bone matrix, tricalcium phosphates, and calcium carbonates. Calcium phosphate materials display a complete lack of toxicity. Their ability to act as a substrate for bone growth makes them a popular material for osteoconductive properties, but they are not osteogenic or osteoinductive.[168]

Xenografts are fabricated from the inorganic portion of bone from animals other than humans and are also osteoconductive. Xenograft bone is reported as a good bone bank material, provided it is completely deproteinated and placed in a bed of bleeding trabecular bone or used with autologous red marrow.[169] It may also be used to augment soft tissue. Proponents of xenografts believe that the nonorganic bone of the animal most resembles the natural human bone mineral.

Osteoconductive materials for hard tissue maintenance or augmentation may be characterized as nonresorbable or resorbable, dense or porous, or crystalline or amorphous materials. Dense HA has become a popular bone substitute when living bone is not a requirement for the augmentation. This material has been described as nonresorbable when it is a highly dense crystalline structure.[170,171] In the presence of bone, a direct bone-HA interface may be observed.[170] This finding is more common when dense HA is placed within the bone and fibrous tissue is not in direct contact during healing.

WWW.HIGHDENT.IR

همیار دندانسازان

Fibrous tissue proliferates at a rate of 0.5 to 1 mm daily, compared with bone that forms much slower (approximately 50 µm per day). Thus the soft tissue has the capacity to reach and encapsulate the HA when it is placed on top of cortical bone. The contacting layer of HA may develop a bony interface, but the majority of material is surrounded by fibrous tissue.[173] The purpose of this type of augmentation is to serve as a denture support region or tissue augmentation to improve soft tissue contours around implants or teeth. When the material is placed into a bone preparation, tooth socket, or other cavity, such as the maxillary sinus, or covered with a barrier membrane (which prevents fibrous tissue from reaching the HA), the tissue developing at the interface is more likely to be bone. However, the material does not resorb to allow creeping substitution and new bone to replace the HA.

Dense HA is inorganic and, as such, cannot grow or integrate to an implant surface. It is also three times harder than cortical bone and is more similar to dentine than bone. Therefore when placed into bone, its primary purpose is to obtund a space and maintain bone contour and volume. If an endosteal implant is planned in the bone-dense HA region, a diamond and high-speed hand piece may be required to prepare the HA. Therefore this material is not recommended for placement into sockets that may receive dental implants in the future. A more common use for dense HA is its placement on the facial or crestal aspect of a ridge around implants to improve the soft tissue contour, or as a ridge augmentation material for denture support.[174] Its use was more widespread a few years ago when fewer bone augmentation products were available.

Highly crystalline HA is more resistant to cellular breakdown than the amorphous form. The crystalline structures of available graft materials present differences based on the origin of the product. It is felt that small crystals, such as observed in normal bone, are desirable. The different treatment (chemical or heat) results in different crystal sizes. It is argued that heat treatment (in excess of 1000° C) results in crystal growth, which does not change the basic structure but may cause altered surface characteristics. In vivo implantation studies in animals suggest that the resorption rate is somewhat proportional to the tricalcium phosphate (TCP) content of the material.[175,176] Therefore all HA may resorb, depending on the surrounding pH, porosity, particle size, volume, and crystallinity.

More porous or amorphous forms of calcium phosphate ceramics are osteoconductive materials, which are resorbable when placed into bone or soft tissue, and are replaced by a process similar to "creeping substitution" found in natural bone remodeling.[177,178] As the field of oral implantology grows, this category of products has been greatly expanded to assist in preservation of the alveolar ridge anatomy after tooth loss and before or in conjunction with implant placement. These materials are usually made of HA, beta-tricalcium phosphate (βTCP), the inorganic portions of a xenograft bone, or various combinations. This category of bone graft produces also includes human cadaver bone that has been freeze dried, but not demineralized (FDB).

Resorption primarily occurs through two different mechanisms: solution and cell mediated. Solution-mediated resorption of a material is a consequence of the pH of the surrounding media. As the pH decreases, mineralized materials such as HA dissolve. It should be noted that HA resorbs at these low pH levels at a similar rapid rate whether calcium phosphate, porous HA, or formulations of dense HA.[172] Solution-mediated resorption occurs too quickly to maintain a space and participate in bone formation. Therefore this category of resorption is to be avoided in most all augmentation sites.

In cell-mediated resorption, cells surrounding the grafted material resorb the material by phagocytosis and then intracellular degradation.[179,180] Osteoclasts and osteoblasts also have been shown to participate in this activity. It is suggested that cell-mediated resorption leads to the activation of protein kinase C, endocytosis, and intracellular dissolution of calcium phosphate materials, which then increases the intracellular calcium concentration, which in turn activates a "calcium-dependent pathway" leading to a mitogenic response.[180]

The resorption rate of calcium phosphates by cellular activity is affected by the particle size, volume of material porosity, and composition of the material. Larger particles require a longer time to resorb, if all other things are equal. For example, a 250-µm particle (a common size of bone substitutes) resorbs faster than a 750-µm particle. Resorption is also related to the volume of material implanted. Larger-size defects filled with HA take longer to be replaced by bone than smaller defects, with all other factors being equal.

The porosity of the material has a primary effect on the resorption time. Dense HA particles exhibit little to no porosity. A macroporous HA exhibits a larger, usually visible porous architecture with 15% holes or more by volume.[181] This type of topography may be produced by a hydrothermal exchange reaction with $CaCO_3$, found in the natural particles of the coral reef. A microporous HA (usually obtained from the inorganic portion of bone of xenografts or freeze-dried cadaver bone) exhibits in excess of 30% holes by volume with cortical bone and up to 70% pores when trabecular bone is used, thus leaving a large intraparticle volume of grafted area available for the regeneration of bone. Microporous HA will resorb by cellular activity faster than macroporous HA, and dense HA is the slowest to resorb, if all other factors are similar.

The tissue where the graft material is inserted also affects the cellular resorption rate. The rib has a cellular turnover rate of 2% per year; long bone, a 10% cellular turnover rate; and bone in the jaws, a turnover rate of 40% or more per year. The resorption process will be fastest in the jaws and slowest for a rib. The dense,

WWW.HIGHDENT.IR

crystalline HA particles may last a lifetime when large in size or volume and under stable pH conditions and in a slow turnover rate system. Amorphous forms of HA may resorb in several months when in small volumes and size and in a fast turnover rate region. Microporous materials are intermediate in resorption times but may require more than 1 year when in larger volumes, such as sinus grafts.

A synthetic 15 residue peptide (P-15) related to a biologically active domain of type I collagen has been identified. Reports suggest that P-15–coated anorganic bovine bone mineral may act as a matrix for bone repair.[182-184]

Prolific literature supporting the use of one alloplast versus another is available in the field of dentistry.[185-194] This may be confusing at times when selecting a specific product. An organized approach should consider the following elements:

- Does the product need to resorb and serve as a scaffold for new formation, or does it need to simply preserve the anatomy?
- If the product needs to resorb for bone growth, what time frame is appropriate for the type of procedure?

Short healing periods need a more readily resorbable product, which therefore should be more porous to facilitate cell-mediated resorption. However, if too easily removed by the recipient site, it may not maintain sufficient space to allow adequate bone formation. In contrast, a more crystalline, less porous product will maintain the space longer but will also take longer to disappear and form bone. Therefore, the choice of product should not necessarily always be from one family or another but is based on the application and the macromolecular and biochemical profile of the product.

Bioactive glass and bioinert ceramics can essentially be grouped with dense crystalline HA because their structure renders them practically unresorbable. Their use is limited to ridge maintenance in areas where no bone growth is needed (e.g., pontics, ridges under a denture).

Partially or slowly resorbable materials may be mixed with readily resorbable materials. In this way, the materials that resorb slowly maintain the space while the body invades the easily resorbed material. An example is in the sinus or membrane grafts with the layered approach of autograft in the host bone, DFDB (30%) and FDB (70%) in the middle layer, and a barrier membrane as a top layer. The material resorbs totally to allow the body to regenerate the whole grafted space, even though this may take years to accomplish.

Natural Barrier Membranes

A thick cortical plate on the graft may act similar to a barrier membrane in guided bone regeneration and prevent the infiltration of epithelium and connective tissue into the graft site. Misch has called this *phase IV* of bone grafting, which allows the cortical block to obtain large volumes of bone, even when the surface of the block has soft tissue covering the site. Autogenous bone is still the gold standard in grafting materials because it may form bone in all four mechanisms (osteogenesis, osteoinduction, osteoconduction, and barrier membrane) and is often readily available.

SUMMARY

The 11 keys of bone grafting are the necessary ingredients to predictable bone augmentation. Because bone modeling is more difficult than remodeling, more keys are required. Bone graft materials help provide several of these keys but are not the entire picture. Instead, the materials and keys are blended for an optimum result. A layered approach to bone augmentation has been developed by Misch. The first layer is the host bone, which has an absence of infection and a surgical RAP before the placement of the bone graft. The defect size and topography is a factor for subsequent consideration. An autograft is placed on the host bone and is immobilized with tent or fixation screws. The autograft and RAP encourage host bone blood vessels to grow into the graft site. Many host growth factors are presented to the graft site as a consequence. The next layer is a mixture of DFDB (30%) and mineralized bone (70%), mixed with PRP. This provides induction, growth factors, and longer space maintenance. A collagen barrier membrane (or cortical bone block) is the next layer, covered with PRP, PPP, or both. Soft tissue closure without tension on the incision line is the next consideration.

A transitional prosthesis, off the soft tissue, is the next step in the process. The last consideration is the undisturbed healing time. When in doubt, "wait longer" is a most important consideration for a successful graft. As a consequence, as many keys and bone graft materials are incorporated into the process as possible, all of which increase the predictable nature of the bone augmentation process.

References

1. Carlsson GE, Thilander H, Hedegard B: Changes in contour of the maxillary alveolar process after extractions with or without insertion of an under immediate full denture, *Acta Odontol Scand* 25:21-43, 1967.
2. Pietrokovski J, Massler M: Alveolar ridge resorption following tooth extraction, *J Prosthet Dent* 17:21-27, 1967.
3. Pietrokovski J, Massler M: Ridge remodeling after tooth extraction in rats, *J Dent Res* 46:222-231, 1967.
4. Schropp L, Wenzel A, Kostopoulos L et al: Bone healing and soft tissue contour changes following single-tooth extraction: a clinical and radiographic 12-month prospective study, *Int J Perio Rest Dent* 23:313-323, 2003.
5. Misch CE: Bone augmentation for implant placement: keys to bone grafting. In Misch CE, editor: *Contemporary implant dentistry*, ed 2, St Louis, 1999, Mosby.

WWW.HIGHDENT.IR

همیار دندانسازان

6. Simion M, Baldoni M, Rossi P et al: A comparative study of the effectiveness of e-PTFE membranes with and without early exposure during the healing period, *Int J Periodontics Restorative Dent* 14:166-180, 1994.
7. Machtei EE: The effect of membrane exposure on the outcome of regenerative procedures in humans: a meta-analysis, *J Periodontol* 72:512-516, 2001.
8. Becker W, Dahlin C, Becker BE et al: The use of a e-PTFE barrier membranes for bone promotion around titanium implants placed into extraction sockets: a prospective multicenter study, *Int J Oral Maxillofac Implants* 9:31-40, 1994.
9. Simion M, Trisi P, Maglione M, et al: A preliminary report on a method for studying the permeability of expanded polytetrafluoroethylene membrane to bacteria in vitro: a scanning electron microscopic and histological study, *J Periodontol* 65:755-761, 1994.
10. Nowzari H, London R, Slots J: The importance of periodontal pathogens in guided periodontal tissue regeneration and guided bone regeneration, *Compend Contin Educ Dent* 16:1042-1046, 1995.
11. Pallasch TJ: The healing pattern of an experimentally induced defect in the rat femur studied with tetracycline labeling, *Calcif Tissue Res* 2:334-342, 1986.
12. Rupprecht S, Petrovic L, Burchhardt B, et al: Antibiotic-containing collagen for the treatment of bone defects, *J Biomed Mater Res B Appl Biomater,* 2007 [e-pub ahead of print].
13. Beardmore AA, Brooks DE, Wenke JC et al: Effectiveness of local antibiotic delivery with an osteoinductive and osteoconductive bone-graft substitute, *J Bone Joint Surg Am* 87:107-112, 2005.
14. Misch CM, Misch CE: The repair of localized severe ridge defects for implant placement using mandibular bone grafts, *Implant Dent* 4:261-267, 1995.
15. Tolman DE: Reconstructive procedures with endosseous implants in grafted bone: a review of the literature, *Int J Oral Maxillofac Implants* 10:275-294, 1995.
16. Schwartz-Arad D, Levin L, Sigal L: Surgical success of intraoral autogenous block onlay bone grafting for alveolar ridge augmentation, *Implant Dent* 14:131-138, 2005.
17. Jovanovic SA, Spiekermann H, Richter EJ: Bone regeneration around titanium dental implants in dehisced defect sites: a clinical study, *Int J Oral Maxillofac Implants* 7:233-245, 1992.
18. Carpio L, Loza J, Lynch S et al: Guided bone regeneration around endosseous implants with anorganic bovine bone mineral: a randomized controlled trial comparing bioabsorbable versus non-resorbable barriers, *J Periodontol* 71:1743-1749, 2000.
19. Haas R, Baron M, Dortbudak O et al: Lethal photosensitization, autogenous bone, and e-PTFE membrane for the treatment or peri-implantitis: preliminary results, *Int J Oral Maxillofac Implants,* 15:374-382, 2000.
20. Locci P, Calvitti M, Belcastro S et al: Phenotype expression of gingival fibroblasts cultured on membranes used in guided tissue regeneration, *J Periodontol* 68:857-863, 1997.
21. Gapski R, Wang HL, Misch CE: Management of incision design in symphysis graft procedures: a review of the literature, *J Oral Implantol* 26:134-142, 2001.
22. Park SH, Wang HL: Clinical significance of incision location on guided bone regeneration: human study. *J Periodontol* 78:47-51, 2007.
23. Silverstein LH: *Principles of suturing,* Mahwah, NJ, 1999, Montage Media.
24. Kurtzman GM, Silverstein LH, Shatz PC et al: Suturing for surgical success, *Dent Today* 24:96-102, 2005.
25. Ethicon sutures. In *Wound closure manual*, Somerville, NJ, 1999, Johnson and Johnson.
26. Leknes KN, Roynstrand IT, Selvig KA: Human gingival tissue reactions to silk and expanded polytetrafluoroethylene sutures, *J Periodontol* 76:34-42, 2005.
27. Leknes KN, Selvig KA, Boe OE et al: Tissue reactions to sutures in the presence and absence of antiinfective therapy, *J Clin Periodontol* 32:130-138, 2005.
28. Yaltirik M, Dedeoglu K, Bilgic B et al: Comparison of four different suture materials in soft tissues of rats, *Oral Dis* 9:284-286, 2003.
29. Selvig KA, Biagiotti GR, Leknes KN et al: Oral tissue reactions to suture materials, *Int J Periodontics Restorative Dent* 18:474-487, 1998.
30. Ivanoff CJ, Widmark G: Nonresorbable versus resorbable sutures in oral implant surgery: a prospective clinical study, *Clin Implant Dent Relat Res* 3:57-60, 2001.
31. Jones JK, Triplett RG: The relationship of cigarette smoking to impaired intraoral wound healing: a review of evidence and implications for patient care, *J Oral Maxillofac Surg* 50:237-239, 1992.
32. Ziran BH, Hendi P, Smith WR et al: Osseous healing with a composite of allograft and demineralized bone matrix: adverse effects of smoking, *Am J Orthop* 36:207-209, 2007.
33. Trombelli L, Scabbia A: Healing responses of gingival recession defects following guided tissue regeneration procedures in smokers and non smokers, *J Clin Periodontol* 24:529-533, 1997.
34. Becker W, Becker BE, Caffesse R: A comparison of demineralized freeze-dried bone and autogenous bone to induce bone formation in human extraction sockets, *J Periodontol* 65:1128-1133, 1994.
35. Becker W, Lynch SE, Lekholm U et al: A comparison of e-PTFE membranes alone or in combination with platelet-derived growth factors and insulin-like growth factor I or demineralized freeze-dried bone in promoting bone formation around immediate extraction socket implants, *J Periodontol* 63:929-940, 1992.
36. Dahlin C, Sennerby L, Lekholm U et al: Generation of new bone around titanium implants using a membrane technique: an experimental study in rabbits, *Int J Oral Maxillofac Implants* 4:19-25, 1989.
37. Nemcovsky CE, Artzi Z, Moses O: Rotated palatal flap in immediate implant procedures. Clinical evaluation of 26 consecutive cases, *Clin Oral Implants Res* 11:83-90, 2000.
38. Chen ST, Darby IB, Adams GG et al: A prospective clinical study of bone augmentation techniques at immediate implants, *Clin Oral Implants Res* 16:176-184, 2005.
39. Buser D, Dula K, Belser U et al: Localized ridge augmentation using guided bone regeneration. 1. Surgical procedure in the maxilla, *Int J Periodontics Restorative Dent* 13:29-45, 1993.
40. Buser D, Dula K, Hirt HP et al: Lateral ridge augmentation using autografts and barrier membranes: a clinical study with 40 partially edentulous patients, *J Oral Maxillofac Surg* 54:420-432; discussion, 432-433, 1996.
41. Fugazzotto P: Ridge augmentation with titanium screws and guided tissue regeneration: technique and report of case, *Int J Oral Maxillofac Implants* 8:335-339, 1993.
42. Prousssaefs P, Lozada J: Use of titanium mesh for staged localized alveolar ridge augmentation: clinical

WWW.HIGHDENT.IR

and histologic-histomorphometric evaluation, *J Oral Implantol* 32:237-247, 2006.
43. Becker W, Becker BE, McGuire MK: Localized ridge augmentation using absorbable pins and e-PTFE barrier membranes: a new surgical technique: case reports, *Int J Periodontics Restorative Dent* 14:49-61, 1994.
44. Mellonig JT, Nevins M, Sanchez R: Evaluation of a bioabsorbable physical barrier for guided bone regeneration. Part II. Material and a bone replacement graft, *Int J Periodontics Restorative Dent* 18:129-137, 1998.
45. Wikesjo UM, Qahash M, Thomson RC et al: Space-providing expanded polytetra fluoroethylene devices define alveolar augmentation at dental implants induced by recombinant human bone morphogenetic protein 2 in an absorbable collagen sponge carrier, *Clin Implant Dent Relat Res* 5:112-123, 2003.
46. Wang H-L, Carroll J: Utilizing absorbable collagen membranes for guided tissue regeneration, guided bone regeneration, and in the treatment of gingival recession, *Compend Contin Educ Dent* 21:399-414, 2000.
47. Stentz WC, Mealey BL, Gunsolley JC et al: Effects of guided bone regeneration around commercially pure titanium and hydroxyapatite-coated dental implants II. Histologic analysis. *J Periodontol* 68:933-949, 1997.
48. Misch CE, Dietsh F: Bone grafting materials in implant dentistry, *Implant Dent* 2:158-167, 1993.
49. Wikesjo UM, Nilveus RE, Selvig KA: Significance of early healing events on periodontal repair: a review, *J Periodontol* 63:158-165, 1992.
50. Anusaksathien O. Giannobile WV: Growth factor delivery to re-engineer periodontal tissues, *Curr Pharm Biotechnol* 3:129-139, 2002.
51. Lin KY, Bartlett SP, Yaremchuk M et al: The effect of rigid fixation on the survival of onlay bone grafts: an experimental study, *Plast Reconstr Surg* 86:449, 1990.
52. LaTrenta GS, McCarthy JG, Breitbart AS et al: The role of rigid skeletal fixation in bone graft augmentation of the craniofacial skeleton, *Plast Reconstr Surg* 84:578, 1989.
53. Schenk RK, Buser D, Hardwick WR et al: Healing pattern of bone regeneration in membrane-protected defects: a histologic study in the canine mandible, *Int J Oral Maxillofac Implants* 9:13-29, 1994.
54. Melcher AH, Dryer CJ: Protection of the blood clot in healing of circumscribed bone defects, *J Bone Joint Surg* 44B:424-429, 1962.
55. Melcher AH, Accurs GE: Osteogenic capacity of periosteal and osteoperiosteal flaps elevated from the parietal bone of the rat, *Arch Oral Biol* 16:573-580, 1971.
56. Ogiso B, Huges FJ, Melcher AH et al: Fibroblasts inhibit mineralized bone nodule formation by rat bone marrow stromal cells in vitro, *J Cell Physiol* 146:442-450, 1991.
57. Frost H: The biology of fracture healing: an overview for clinicians. Part I, *Clin Orthop* 248:283-293, 1989.
58. Frost H: The biology of fracture of healing: an overview for clinicians. Part II, *Clin Orthop* 248:294-309, 1989.
59. Frost H: The regional acceleratory phenomenon: a review, *Henry Ford Hosp Med J* 31:3-9, 1983.
60. Shih MS, Norrdin RW: Regional acceleration of remodeling during healing of bone defects in beagles of various ages, *Bone* 6:377-379, 1985.
61. Martin RB: Osteonal remodeling in response to screw implantation in canine femora, *J Orthop Res* 5:445-454, 1987.
62. Yaffe A, Fine N, Alt I et al: The effect of bisphosphonate on alveolar bone resorption following mucoperiosteal flap surgery in the mandible of rats, *J Periodontol* 66:999-1003, 1995.
63. Rompen BH, Biewer R, Vanheusden A et al: The influence of cortical perforations and of space filling with peripheral blood on the kinetics of guided bone generation. A comparative histometric study in the rat, *Clin Oral Implants Res* 10:85-94, 1999.
64. Nishimura I, Shimizu Y, Ooya K: Effects of cortical bone perforation on experimental guided bone regeneration, *Clin Oral Implants Res* 15:293-300, 2004.
65. Giannobile WW, Ryan S, Shih MS et al: Recombinant human osteogenic protein-1 (OP-1) stimulates periodontal wound healing in class III furcation defects, *J Periodontol* 69:129-137, 1998.
66. Fiorellini JP, Buser D, Riley E et al: Effect on bone healing of bone morphogenetic protein placed in combination with endosseous implants: a pilot study in beagle dogs, *Int J Periodontics Restorative Dent* 21:41-47, 2001.
67. Bogoche E, Gschwend N, Rahn B et al: Healing of cancellous bone osteotomy in rabbits. Part I. Regulation of bone volume and the regional acceleratory phenomenon in normal bone, *J Orthop Res* 11:285-291, 1993.
68. Shih MS, Norrdin RW: Effect of prostaglandin E1 on regional haversian remodeling in beagles with fractured ribs: a histomorphometric study, *Bone* 8:87-90, 1987.
69. Mueller M, Schilling T, Minne HW et al: A systemic acceleratory phenomenon (SAP) accompanies the regional acceleratory phenomenon (RAP) during healing of a bone defect in the rat, *J Bone Miner Res* 6:401-410, 1991.
70. Winet H: The role of microvasculature in normal and perturbed bone healing as revealed by intravital microscopy, *Bone* 19:39S-57S, 1996.
71. Hammerle CH, Schmid J, Olah AJ et al: A novel model system for the study of experimental guided bone formation in humans, *Clin Oral Implants Res* 7:38-47, 1996.
72. Buser D, Dula K, Belser UC et al: Localized ridge augmentation using guided bone regeneration II. Surgical procedure in the mandible, *Int J Periodontics Restorative Dent* 5:10-29, 1995.
73. Lynch SE, Genco RJ, Marx RE: *Tissue engineering applications in maxillofacial surgery and periodontics,* Carol Stream, Ill, 1999, Quintessence.
74. Lee MB: Bone morphogenetic proteins: background and implications for oral reconstruction. A review, *J Clin Periodontol* 24:255-265, 1997.
75. Reddi A, Cunningham NS: Initiation and promotion of bone differentiation by bone morphogenetic proteins, *J Bone Miner Res* 8:S499-S502, 1993.
76. Shimokado K, Raines EW, Madtes DK et al: A significant part of macrophage-derived growth factor consists of at least two forms of PDGF, *Cell* 43:277-286, 1985.
77. Rose LE, Rosenberg E: Bone grafts and growth and differentiation factors for regenerative therapy: a review. *Pract Proced Aesthet Dent* 13:725-734, 2001.
78. Grotendorst GR, Martin GR, Pencev D et al: Stimulation in granulation tissue formation by platelet-derived growth factor in normal and diabetic rats, *J Clin Invest* 76:2323-2329, 1985.
79. Howes R, Bowness JM, Grotendorst GR et al: Platelet derived growth factor enhances demineralized bone matrix induced cartilage and bone formation, *Calcif Tissue Int* 42:34-38, 1988.
80. Marx RE, Carlson BR, Eichstaedt RM et al: Platelet-rich plasma: growth factor enhancement for bone grafts, *Oral*

WWW.HIGHDENT.IR

همیار دندانسازان

Surg Oral Med Oral Pathol Oral Radiol Endod 85:638-646, 1998.
81. Grotendorst GR, Pencev D, Misten GR et al: Molecular mediators of tissue repair. In Hunt TK, Heppenstall RB, Pines E et al, editors: *Soft and hard tissue repair,* New York, 1984, Prager.
82. Martas H: Fibrin seal: the state of the art, *J Oral Maxillofac Surg* 43:605, 1985.
83. Bosch P, Lintner F, Arbes H et al: Experimental investigations of the effect of the fibrin adhesive on the Kiel heterologous bone graft, *Arch Orthop Trauma Surg* 96:177, 1980.
84. Sporn MB, Roberts AB, Shull JB et al: Polypeptide transforming growth factors isolated from bone sources and used in wound healing in vivo, *Science* 219:1329-1331, 1983.
85. Beck LS, Deguzman L, Lee WP et al: Rapid publication. TGF-beta 1 induces bone closure of skull defects *J Bone Miner Res* 6:1257-1265, 1991.
85a. Gerard D, Carlson ER, Gotcher JE et al: Effects of platelet-rich plasma on the healing of autologous bone grafted mandibular defects in dogs, *J Oral Maxillofac Surg* 64: 443-451, 2006.
86. Lind M. Schumacker B, Soballe K et al: Transforming growth factor-beta enhances fracture healing in rabbit tibiae, *Acta Orthop Scand* 64:553-556, 1993.
87. Joyce ME, Roberts AB, Sporn MB et al: Transforming growth factor-beta and the initiation of chondrogenesis and osteogenesis in the rat femur, *J Cell Biol* 110:2195-2207, 1990.
88. Marcopoulou CE, Vavouraki HN, Dereka XE et al: Proliferative effect of growth factors TGR-beta1, PDGR-BB and rhBMP-2 on human gingival fibroblasts and periodontal ligament cells, *J Int Acad Periodontol* 5:63-70, 2003.
89. Ruskin JD, Hardwick R, Buser D et al: Alveolar ridge repair in a canine model using rhTGF-beta 1 with barrier membranes, *Clin Oral Implants Res* 11:107-115, 2000.
90. Howell TH, Fiorellini JP, Paquette DW et al: A phase I/II clinical trial to evaluate a combination of recombinant human platelet-derived growth factor BB and recombinant human insulin-like growth factor-I in patients with periodontal disease, *J Periodontol* 68:1186-1193, 1997.
91. Lee SJ, Park YJ, Park SN et al: Molded porous poly (L-lactide) membranes for guided bone regeneration with enhanced effects by controlled growth factor release, *J Biomed Mater Res* 55:295-303, 2001.
92. Stefani CM, Machado MA, Sallum EA et al: Platelet-derived growth factor/insulin-like growth factor-I combination and bone regeneration around implants placed into extraction sockets: a histometric study in dogs, *Implant Dent* 9:126-131, 2000.
93. Graves DT, Kang YM, Kose K: Growth factors in periodontal regeneration, *Compend Contin Educ Dent Suppl* 18:S672-677, 1994.
94. Jackson RA, Nurcombe V, Cool SM: Coordinated fibroblast growth factor and heparin sulfate regulation of osteogenesis, *Gene* 279:79-91, 2006.
95. Inui K, Maeda M, Sano A et al: Local application of basic fibroblast growth factor minipellet induces the healing of segmental bony defects in rabbits, *Calcif Tissue Int* 63:490-495, 1998.
96. Fiedler J, Brill C, Blum WF et al: IGF-I and IGF-II stimulate directed cell migration of bone marrow-derived human mesenchymal progenitor cells, *Biochem Biophys Res Commun* 45:1177-1183, 2006.
97. Marx RE: Platelet-rich plasma (PRP): what is PRP and what is not PRP? *Implant Dent* 10:225-228, 2001.
98. Gerard D, Carlson ER, Gotcher JE et al: Effects of platelet-rich plasma on the healing of autologous bone grafted mandibular defects in dogs, *J Oral Maxillofac Surg* 64:443-451, 2006.
99. Marx RE, Snyder RM, Kline SN: Cellular survival of human marrow during placement of marrow cancellous bone grafts, *J Oral Surg* 37:712-718, 1979.
100. Garg AK, Gargenese D, Peace I: Using a platelet-rich plasma to develop an autologous membrane for growth factor delivery in dental implant therapy, *Dent Implantol Update* 11:41-44, 2000.
101. Marx RE. Platelet-rich plasma. A source of multiple autologous growth factors for bone grafts. In Lynch SE, Genco RJ, Marx RE, editors: *Tissue engineering application in maxillofacial surgery and periodontics,* Chicago, 1999, Quintessence.
102. Tischler M: Platelet-rich plasma. The use of autologous growth factors to enhance bone and soft tissue grafts, *N Y State Dent J* 68:22-24, 2002.
103. Salata LA, Franke-Stemport V, Rasmusson I: Recent outcomes and perspectives of the application of bone morphogenetic proteins in implant dentistry, *Clin Implant Dent Relat Res* 4:27-32, 2002.
104. Urist MR, Strates BS: Bone morphogenetic protein, *J Dent Res* 50:1392-1406, 1971.
105. Urist MR: Mesenchymal cell reactions to inductive substrates for new bone formation. In Dunphy JE, Van Winkle W Jr, editors: *Repair and regeneration,* New York, 1969, McGraw-Hill.
106. Urist MR, DeLange RJ, Finerman GAM: Bone cell differentiation and growth factors, *Science* 220:680-686, 1983.
107. Lind M: Growth factors: possible new clinical tools. A review, *Acta Orthop Scand* 67:407-417, 1996.
108. Wang EA, Rosen V, D'Alessandro JS et al: Recombinant human bone morphogenetic protein induces bone formation, *Proc Natl Acad Sci USA* 87:2220-2224, 1990.
109. Toriumi DM, Kotler HS, Luxenberg DP et al: Mandibular reconstruction with a recombinant bone-inducing factor, *Arch Otolaryngol Head Neck Surg* 117:1101-1112, 1991.
110. Vohof JS, Haus MT, de Ruijter AE et al: Bone formation in transforming growth factor beta-I-loaded titanium fiber mesh implants, *Clin Oral Implants Res* 13:94-102, 2002.
111. Okubo Y, Bessho K, Fujimura K et al: Expression of bone morphogenetic protein in the course of osteoinduction by recombinant human bone morphogenetic protein-2, *Clin Oral Implants Res* 13:80-85, 2002.
112. Sigurdsson TJ, Lee MB, Kubota K et al: Periodontal repair in dogs: recombinant human bone morphogenetic protein-2 significantly enhances periodontal regeneration, *J Periodontol* 66:131-138, 1995.
113. Nagao H, Tachikawa N, Miki T et al: Effect of recombinant human bone morphogenetic protein-2 on bone formation in alveolar ridge defects in dogs, *J Oral Maxillofac Surg* 31:66-72, 2002.
114. Nevins M, Kirker-Head C, Nevins M et al: Bone formation in the goat maxillary sinus induced by absorbable collagen sponge implants impregnated with recombinant human bone morphogenetic protein-2, *Int J Period Rest Dent* 16:8-19, 1996.
115. Boyne PJ, Marx RE, Nevins M et al: A feasibility study evaluating rhBMP-2/absorbable collagen sponge for

WWW.HIGHDENT.IR

maxillary sinus augmentation, *Int J Periodontics Restorative Dent* 17:25, 1997.
116. Camelo M, Nevins M, Schenk R et al: Periodontal regeneration in human Class II furcations using purified recombinant human platelet-derived growth fact-BB (rh PDGF-BB) with bone allograft, *Int J Periodontics Restorative Dent* 23:213-225, 2003.
117. Nevins M, Giannobile WV, McGuire MK et al: Platelet derived growth factor (rhPDGF-BB) stimulates bone fill and rate of attachment level gain. Results of a large multicenter randomized controlled trial, *J Periodontol* 76:2205-2215, 2005.
118. Nevins M, Camelo M, Nevins ML et al: Periodontal regeneration in humans using recombinant human platelet derived growth factor-BB (rhPDGF-BB) and allogenic bone, *J Periodontol* 74:1282-1292, 2003.
119. Fiorellini JP, Buser D, Riley E et al: Effect on bone healing of bone morphogenetic protein placed in combination with endosseous implant: a pilot study in beagle dogs. *Int J Periodontics Restorative Dent* 21:41-47, 2001.
120. Sigurdsson TJ, Nguyen S, Wikesjo UM: Alveolar ridge augmentation with rh BMP-2 and bone-to-implant contact in induced bone, *Int J Periodontics Restorative Dent* 21:461-473, 2001.
121. Taba M Jr, Jin Q, Sugai JV et al: Current concepts in periodontal bioengineering, *Orthod Craniofac Res* 8: 292-302, 2005.
122. Wei G, Jin Q, Giannobile WV et al: The enhancement of osteogenesis by nano-fibrous scaffolds incorporating rh BMP-7 nanospheres, *Biomaterials* 28:2087-2096, 2007
123. Marks T, Wingerter S, Franklin L et al: Histological and radiographic comparison of allograft substitutes using a continuous delivery model in segmental defects, *Biomed Sci Instrum* 43:194-199, 2007.
124. Dequeker J, Merlevede W: Collagen content and collagen extractability pattern of adult human bone according to age, sex and degree of porosity, *Biochem Biophys Acta* 244:410-420, 1971.
125. Dickerson JWT: The effect of development on the composition of a long bone of the pig, rat, fowl, *Biochem J* 82:47, 1962.
126. Gray JC, Elves MW: Early osteogenesis in compact bone isografts: a quantitative study of contributions of the different graft cells, *Calcif Tissue Int* 29:225-237, 1979.
127. Marx RE, Schiff WM, Saunders TR: Reconstruction and rehabilitation of cancer patients. In Fonseca RJ, Davis WH, editors: *Reconstructive preprosthetic oral and maxillofacial surgery,* ed 2, Philadelphia, 1995, WB Saunders.
128. Holmstrand K: Biophysical investigations of bone transplants and bone implants: an experimental study, *Acta Orthop Scand Suppl* 26:1-92, 1957.
129. Friedenstein AJ, Piatetzky-Shapiro II, Petrakova KV: Osteogenesis in transplants of bone marrow cells, *J Embryol Exp Morphol* 16:381-386, 1966.
130. Marx RE, Wong ME: A technique for the compression and carriage of autogenous bone during grafting procedures, *J Oral Maxillofac Surg* 45:988, 1987.
131. Glowacki J, Mulliken JB: Demineralized bone implants, *Clin Plast Surg* 12:233-241, 1985.
132. Friedlander GE: Bone grafts: the basic science rationale for clinical applications, *J Bone Joint Surg* 69A:786-790, 1987.
133. Covey DC, Albright JA: Clinical induction of bone repair with demineralized bone matrix or a bone morphogenetic protein, *Orthop Rev* 18:857-863, 1989.
134. Takagi K, Urist MR: The role of bone marrow in bone morphogenetic protein-induced repair of massive diaphyseal defects, *Clin Orthop* 171:224-231, 1982.
135. Takagi K, Urist MR: The reaction of the dura to bone morphogenetic protein (BMP) in repair of skull defects, *Ann Surg* 196:100-109, 1982.
136. Mellonig JT, Levey R: The effect of different sizes of freeze dried bone allograft on bone growth, *J Dent Res* 63:222, 1984.
137. Buck BE, Malinin TL: Human bone and tissue allografts, *Clin Orthop* 303:8-17, 1994.
138. Gunther KP, Scharf HP, Pesch HJ et al: Osteointegration of solvent-preserved bone transplants in an animal model, *Osteologie* 5:4-12, 1996.
139. Becker W, Urist M, Becker BE et al: Clinical and histologic observations of sites implanted with intraoral autologous bone grafts or allografts. 15 human case reports, *J Periodontol* 67:1025-1033, 1996.
140. Dalkyz M, Ozcan A, Yapar M et al: Evaluation of the effects of different biomaterials on bone defects, *Implant Dent* 9:226-235, 2000.
141. Acil Y, Springer IN, Broek V et al: Effects of bone morphogenetic protein-7 stimulation on osteoblasts cultured on different biomaterials, *J Cell Biochem* 86:90-98, 2002.
142. Wikesjo UM, Sorensen RG, Kinoshita A et al: RhBMP-2/alpha BSM induces significant vertical alveolar ridge augmentation and dental implant osseointegration, *Clin Implant Dent Relat Res* 4:174-182, 2002.
143. Narang R, Wells H, Laskin DM: Experimental osteogenesis with demineralized allogeneic bone matrix in extraskeletal sites, *J Oral Maxillofac Surg* 40:133, 1982.
144. Cunningham NS, Reddi AH: Biologic principles of bone induction: application to bone grafts. In Habal MB, Reddi HA, editors: *Bone grafts and bone substitutes,* Philadelphia, 1992, Saunders.
145. Mulliken JB, Glowacki J: Induced osteogenesis for repair and construction in the craniofacial region, *Plast Reconstr Surg* 65:553, 1980.
146. Forsell JH: Irradiation of musculoskeletal tissues. In Tomford WW, editor: *Musculoskeletal tissue banking,* New York, 1993, Raven Press.
147. Ijiri S, Yamamuro T, Nakamura T et al: Effect of sterilization on bone morphogenetic protein, *J Orthopaed Res* 12:628-636, 1994.
148. Munting E, Wilmart JF, Wijne A et al: Effect of sterilization on osteoinduction comparison of five methods in demineralized rat bone, *Acta Orthop Scand* 59:34-38, 1988.
149. Mellonig JT, Prewett AB, Moyer MP et al: Allograft safety: viral inactivation with bone demineralization, *Contemp Orthop* 31:257-261, 1995.
150. Swenson CL, Arnoczky SP: Demineralization for inactivation of infectious retrovirus in systemically infected cortical bone, *J Bone Joint Surg Am* 85-A:323-331, 2003.
151. Russo R, Scarborough N: *Inactivation of viruses in demineralized bone matrix*. Presented at U.S. Food and Drug Administration workshop on tissue for transplantation and reproductive tissue, June 1995.
152. Buck BE, Malinin TI, Brown MD: Bone transplantation and human immunodeficiency virus: an estimate of risk of acquired immunodeficiency syndrome (AIDS), *Clin Orthop* 240:129-136, 1989.
153. Mellonig JT, Triplett RG: Guided tissue regeneration and endosseous dental implants, *Int J Periodontics Restorative Dent* 13:108-119, 1993.

WWW.HIGHDENT.IR

همیار دندانسازان

154. Landsberg CJ, Grosskopf A, Weinzeb M: Clinical and biologic observation of demineralized freeze dried bone allografts in augmentation procedures around dental implants, *Int J Oral Maxillofac Implants* 9:586-592, 1994.
155. Becker W, Schenk ND, Higuchi K et al: Variations in bone regeneration adjacent to implants augmented with barrier membranes alone or with demineralized freeze-dried bone or autologous grafts: a study in dogs, *Int J Oral Maxillofac Implants* 10:143-154, 1995.
156. Pinholt EM, Hanaes HR, Roervik M et al: Alveolar ridge augmentation by osteoinductive materials in goats, *Scand J Dent Res* 100:361-365, 1992.
157. Shanaman RH: A retrospective study of 237 sites treated consecutively with guided tissue regeneration, *Int J Periodontics Restorative Dent* 14:293-301, 1994.
158. Shigeyama Y, D'Errico JA, Stone R et al: Commercially-prepared allograft material has biological activity in vitro, *J Periodontol* 66:478-487, 1995.
159. Schwartz Z, Mellonig JT, Carnes DL et al: Ability of commercial demineralized freeze-dried bone allograft to induce new bone formation, *J Periodontol* 67:918-926, 1996.
160. Feighan JE, Davy D, Prewett AB et al: Induction of bone by a demineralized bone matrix gel: a study in a rat femoral defect model, *J Orthop Res* 13:88, 1995.
161. Chesmel KD, Branger J, Wertheim H et al: Healing response to various forms of human demineralized bone matrix in athymic rat cranial defects, *J Oral Maxillofac Surg* 56:857-863, 1998.
162. Edwards JT, Diegmann MH, Scarborough NL: Osteoinduction of human demineralized bone: characterization in rat model, *Clin Orthop Relat Res* 357:219-228, 1998.
163. Callan DP, Salkeld SL, Scarborough N: Histologic analysis of implant sites after grafting with demineralized bone matrix putty and sheets, *Implant Dent* 9:36-44, 2000.
164. Takikawa S, Bauer TW, Kambic H et al: Comparative evaluation of the osteoinductivity of two formulations of human demineralized bone matrix, *J Biomed Mater Res* Am 65:37-42, 2003.
165. Martin GJ, Boden SD, Titus L et al: New formulations of demineralized bone matrix as a more effective bone graft alternative in experimental posterior lateral lumbar spine arthrodesis, *Spine* 24:637, 1999.
166. Sigurdsson TJ, Nygaard L, Tatakis DN et al: Periodontal repair in dogs: evaluation of rh BMP-2 carriers, *Int J Periodontics Restorative Dent* 16:524-537, 1996.
167. Niederwanger M, Urist MR: Demineralized bone matrix supplied by bone banks for a carrier of recombinant human bone morphogenetic protein (rh BMP-2): a substitute for autogeneic bone grafts, *J Oral Implantol* 22:210-215, 1996.
168. Kent JN, Jarcho M: Ridge augmentation procedures with hydroxylapatite. In Fonseca JS, Davis WH, editors: *Reconstructive preprosthetic oral and maxillofacial surgery,* ed 2, Philadelphia, 1995, WB Saunders.
169. Salama R, Burwell RG, Dickson IR: Recombined grafts of bone and marrow, *J Bone Joint Surg* 55:402-417, 1973.
170. Jarcho M: Calcium phosphate ceramics as hard tissue prosthetics, *Clin Orthop* 157:259-278, 1981.
171. Rejda BV, Peelen JGJ, de Groot K: Tri-calcium phosphate as a bone substitute, *J Bioeng* 1:93-97, 1977.
172. LeGeros RZ: Calcium phosphate materials in restorative dentistry: a review, *Adv Dent Res* 2:164-180, 1988.
173. Chang C, Matukas VJ, Lemons JE: Histologic study of hydroxylapatite as an implant material for mandibular augmentation, *J Oral Maxillofac Surg* 41:729-737, 1983.
174. Mercier P, Bellavance F, Cholewa J et al: Long-term stability of atrophic ridges reconstructed with hydroxylapatite: a prospective study. *J Oral Maxillofac Surg* 54:960-968, 1996.
175. Uchida A, Nade SML, McCarney ER et al: The use of ceramics for bone replacement, *J Bone Joint Surg* 66B:269-275, 1984.
176. Klein CPAT, Drissen AA, deGroot K: Biodegradation behavior of various calcium phosphate material in bone tissue, *J Biomed Mater Res* 17:769-784, 1983.
177. Tofe AJ, Watson BA, Bowerman MA: Solution and cell-mediated resorption of grafting materials [abstract], *J Oral Implantol* 17:345, 1991.
178. Cheung HS, Tofe AJ: Mechanism of cell growth on calcium phosphate particles: role of cell mediated dissolution of calcium phosphate matrix, *STP Pharma Sciences* 3:51-55, 1993.
179. Holtrap ME, Cox KA, Glowacki J: Cells of the mononuclear phagocytic system resorb implant's bone matrix: a histologic and ultrastructural study, *Calcif Tissue Int* 34:488-494, 1982.
180. Kwong CH, Burns WB, Cheung HS: Solubilization of hydroxyapatite crystals by murine bone cells macrophages and fibroblasts, *Biomaterials* 10:579-584, 1989.
181. White E, Shors EC: Biomaterial aspects of Interpore 200 porous hydroxyapatite, *Dent Clin North Am* 30:49-67, 1986.
182. Smiler D, Soltan M, Lee JW: A histomorphogenic analysis of bone grafts augmented with adult stem cells, *Implant Dent* 16:42-53, 2007.
183. Scarano A, Degidi M, Iezzi G et al: Maxillary sinus augmentation with different biomaterials: a comparative histologic and histomorphometric study in man. *Implant Dent* 15:197-207, 2006.
184. Thompson DM, Rohrer MD, Prasad HS: Comparison of bone grafting materials in human extraction sockets: clinical, histologic, and histomorphometric evaluations. *Implant Dent* 15:89-96, 2006.
185. Nevins ML, Camelo M, Lynch SE et al: Evaluation of periodontal regeneration following grafting intrabony defects with bio-oss: a histologic report, *Int J Periodontics Restorative Dent* 23:9-17, 2003.
186. Tonetti MS, Cortellini P, Lang NO et al: Clinical outcomes following treatment of human intrabony defects with GTR/bone replacement material or access flap alone. A multicenter randomized controlled clinical trial, *J Clin Periodontol* 31:770-776, 2004.
187. Scarano A, Iezzi G, Petrone G et al: Cortical regeneration with a synthetic cell-binding peptide: a histologic and histomorphometric study, *Implant Dent* 12:318-324, 2003.
188. Lekovic V, Camargo P, Weinlander M et al: Combination porous bone material, enamel matrix proteins, and a bioabsorbable membrane to repair intrabony periodontal defects in humans, *J Periodontol* 72:583, 2001.
189. Paolantonio M, Scarano A, DiPlacido G et al: Periodontal healing in humans using anorganic bovine bone and bovine peritoneum-derived collagen membrane: a clinical and histologic case report, *Int J Periodontics Restorative Dent* 21:505-515, 2001.
190. Mellonig JT: Human histological evaluation of a bovine-derived bone xenograft in the treatment of periodontal

WWW.HIGHDENT.IR

osseous defects, *J Periodontics Restorative Dent* 20:19-29, 2000.

191. Artzi Z, Tal H, Dayan D: Porous bovine bone mineral in healing of human extraction sockets. Part 1: histomorphometric evaluations at 9 months, *J Periodontol* 1015-1023, 2000.
192. Proussaefs P, Lozada J, Kleinmann A et al: The use of titanium mesh in conjunction with autogenous bone graft and inorganic bovine bone mineral (bio-oss) for localized alveolar ridge augmentation: a human study, *Int J Periodontics Restorative Dent* 23:185-195, 2003.
193. Maiorana C, Santoro F, Rabagliati M et al: Evaluation of the use of iliac cancellous bone and anorganic bovine bone in the reconstruction of the atrophic maxilla with titanium mesh: a clinical and histologic investigation, *Int J Oral Maxillofac Implants* 16:427-432, 2001.
194. Kubler A, Neugebauer J, Oh JH et al: Growth and proliferation of human osteoblasts on different bone graft substitutes: an in vitro study, *Implant Dent* 13: 171-179, 2004.

WWW.HIGHDENT.IR
همیار دندانسازان

Chapter 37

Tooth Extraction, Socket Grafting, and Barrier Membrane Bone Regeneration

Carl E. Misch, Jon B. Suzuki

Two fundamental concepts determine the position of an individual implant within a treatment plan: biomechanics and esthetics. Both of these concepts require adequate bone volume to accomplish their goals. Biomechanics requires key implant positions as the canine and first molar sites. When available bone is not present in those locations, bone augmentation is required. When the goal is an FP-1 restoration with ideal crown contour and soft tissue drape, abundant bone is necessary. If the available bone is inadequate, bone augmentation is indicated. Both factors of biomechanics and esthetics often require ideal available bone volumes.

The primary goal of this chapter is to present clinical guidelines to achieve adequate to ideal bone volumes for implant insertion. The process begins with the extraction of a hopeless tooth and socket grafting and continues to the use of barrier membrane (BM) guided bone regeneration (GBR).

ATRAUMATIC TOOTH EXTRACTION

At some point in time, the dental practitioner may decide to remove a natural tooth. Hopeless or unrestorable teeth may be related to periodontal, endodontic, prosthetic, or orthodontic failures. The concept of "treat or extract" was addressed in preprosthetic considerations in a previous chapter. Once the extraction of a natural tooth is indicated, methods to maintain or obtain the surrounding hard and soft tissues are indicated.

Over the last decade, interest has increased in atraumatic tooth extractions and socket grafting to maintain bone for implant insertion. Before extraction, exudates should not be present. Antibiotics, prophylaxis, scaling, and root planing may all be necessary before the extraction/grafting appointment.

The process of atraumatic tooth extraction and maintenance (or augmenting) of soft and hard tissues begins with the soft tissue. The cells of the inner layer of the periosteum are responsible for bone remodeling. Whenever the periosteum is reflected, the cells are injured and need to regenerate before the remodeling process begins. The cortical bone receives more than 80% of its arterial blood supply and faciliates 100% of its venous blood return through the periosteum.[1] When the bone volume is ideal, the periosteum should not be reflected. However, the periosteum also limits the volume of bone formation. When the periosteum is separated from the bone graft by a barrier membrane, more volume of bone is regenerated. The periosteum helps bone remodeling or bone repair, but it limits bone modeling and regeneration.

The soft tissue drape around the teeth is also affected by the reflection of the periosteum and often shrinks to adapt to the residual ridge form. In fact, the soft tissue is more labile to the trauma and reflection of the tissues than the hard tissues. Therefore the sulcular and surrounding soft tissue should remain undisturbed during tooth extraction to prevent further dimensional loss.

The extraction of a natural tooth begins with an incision within the sulcus, preferably with a thin scalpel blade rather than a blunt periotome, 360 degrees around the tooth, to cut the connective tissue attachment fibers above the bone (Figure 37-1). There are 13 different connective tissue fiber groups around a tooth, of which six directly insert into the cementum of the tooth above the bone. If these fibers are not cut before the extraction, trauma to the soft tissue is imminent; soft tissue tearing may occur as well. The tip of the interdental papilla is avascular and should not be incised when possible. The soft tissue should not be reflected, as soft tissue retraction and shrinkage during initial healing is more evident, especially in the interdental papilla region.

The next step in an atraumatic extraction process is to observe the crown and root anatomy, especially in multirooted teeth. When a tooth is extracted next

WWW.HIGHDENT.IR

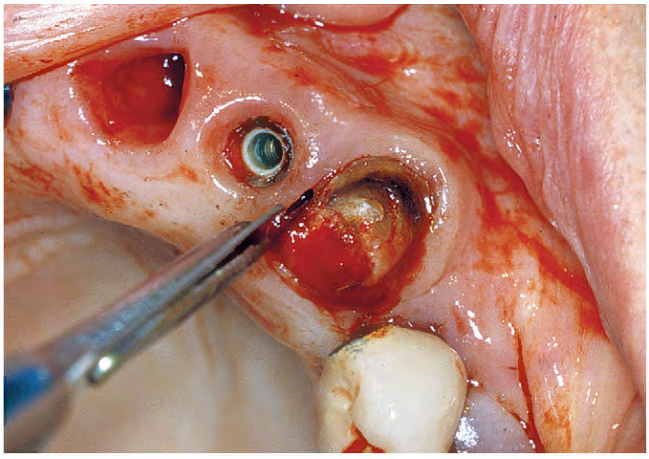


Figure 37-1 The extraction of the tooth begins with a scalpel to incise the sulcular connective tissue fibers above the bone, which are attached to the cementum of the tooth.

Figure 37-2 **A,** The maxillary lateral incisor requires extraction. The pathway of removal may restrict the extraction or chip an adjacent tooth. **B,** The distal portion of the lateral incisor was reduced to allow the tooth to move distal, which allows pressure to the periodontal ligament and bone. In addition, there is less risk of adjacent tooth damage.

to adjacent teeth, the pathway of removal is often obstructed by the position of the adjacent tooth. If the tooth to be extracted is not reduced (especially mesial and distal), instruments may chip the enamel (or restoration) of the adjacent tooth and may cause the extraction of the tooth to take an altered pathway of removal, which is more likely to fracture the roots,

Figure 37-3 **A,** The left central incisor with replacement resorption requires extraction. The single tooth root is reduced mesiodistal and separated in two. **B,** The separated central permits the unresorbed tooth root to be displaced to the mesial when the osteotome is inserted into the distal periodontal ligament space.

bone, or both (Figure 37-2). If the roots of the tooth to be extracted are divergent, they should be sectioned and removed as individual units, rather than risking fracture of the roots or surrounding bone (Figure 37-3). When the roots are fractured, there is an increased risk of bone fracture/removal to retrieve them. If bone removal around the tooth is necessary (because the tooth is fractured or decayed to the bony crest), it ideally should be at the expense of the lingual alveolus, not the more labial bone.

Biomechanic concepts have been used to extract teeth for thousands of years and date back to the days of Aristotle (384-322 BC),[2] who described the mechanics of the extraction forceps, including the advantages of "two levers acting in contrary sense having a single fulcrum." This was 100 years before Archimedes reported on the principles of the lever.

Pierre Fauchard (1690-1761) is credited with being the pioneer of scientific dentistry and gave specific instructions for extracting teeth using a dental elevator, a "pelican," or pincers (forceps).[2,3] He describes loosening the tooth with an elevator, then using the claw of the "pelican" (invented by Chauliac in the fourteenth century).[4] The pelican handle was positioned both on

WWW.HIGHDENT.IR

همیار دندانسازان

the tooth and on the gum below the tooth while it was rocked back and forth (which he called "shaking") before the extraction. Taft described a similar technique using the dental key, which had left and right claws that provided twisting and rocking in both directions.[5] This allowed the tooth to be loosened sufficiently to be pulled from the socket with "pincers" (forceps). Modern extraction forceps date from Tomes in 1840 with the development of the anatomical forceps, complete with a handle and beak to fit the neck of the tooth.

The principle of the dental elevator is also not a modern development. Abulkasim (AD 1050-1122) was the first to apply a single lever (elevator) under the tooth to force it from its "bed." It was improved by Ambroise Paré in the sixteenth century to lift out the tooth before using the pelican.[5] Although these biomechanical methods to remove teeth are effective, a review of the biomechanical principals is in order to decrease the trauma during the tooth extraction process.

The term "simple machine" is often used to describe basic devices that increase the amount of force applied (e.g., the lever, inclined plane, wheel, screw, pulley). They each transmit or modify force or torque. The most common devices used in the extraction of teeth include levers and inclined planes.

The wedge is technically a moving double inclined plane, which overcomes a large resistance by applying a relatively smaller force than the load necessary to move an object. The mechanical advantage of a wedge depends on the ratio of its length to its thickness. A short wedge with a wide angle moves an object faster; however, it requires more force than a long wedge with a smaller angle. Dental elevators and periotomes use the mechanical advantage of a wedge to initiate the luxation of teeth for their removal.

Periotomes are usually longer and thinner wedges compared with dental elevators and should be used to begin the atraumatic extraction process. Periotomes may be used in a similar manner for extraction of intact teeth or removal of retained root fragments. The long axis of the periotome blade should be inserted into the interproximal region along the root long axis (to protect the facial plate of bone), with the tip of the periotome blade located within the crest of the alveolar bone. The instrument is then pushed into the periodontal ligament space or tapped with a mallet into the periodontal ligament space along the mesial and distal root, severing the periodontal ligament immediately below the alveolar crest and wedging the tooth against the opposing cribriform plate (Figure 37-4, *A*).

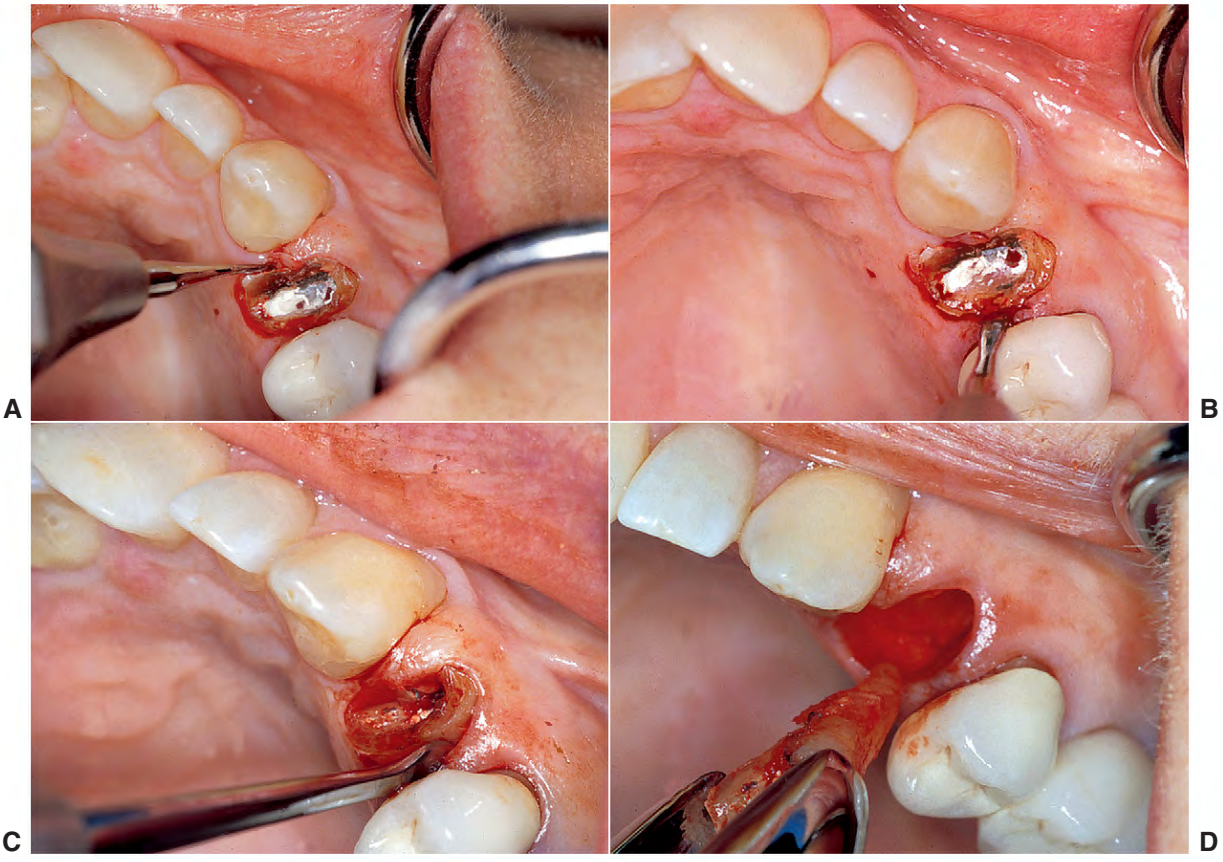


Figure 37-4 **A,** A periotome is inserted along the tooth root on the mesial and pushed (or tapped with a mallet) to wedge the tooth against the opposing cribriform plate. A similar process is performed on the distal interpositional region of the tooth root. **B,** Once the periotome acts as a wedge and is in place for 10 to 30 seconds, it is tapped (with a mallet) farther down along the mesial and distal interproximal root surface. **C,** The periotome is converted into a lever by rotating the handle several degrees, which magnifies the force against the root. The tooth becomes slightly mobile at this stage. **D,** A traditional dental forceps may remove the tooth after initial mobility is created by the periotome.

WWW.HIGHDENT.IR

A period of 10 to 30 seconds is allowed to elapse while the instrument is in place. This allows biomechanical creep to occur to the ligament and reduces its strength, and because the tooth is pushed against the opposing alveolus, it also begins to expand the bone. This process is much more effective when there is no adjacent tooth contact. Reducing the mesial and distal contacts of the tooth to be extracted not only decreases the risk of damage to the adjacent tooth crown, but also aids in the first step to extract the hopeless tooth. The periotome is then pushed farther down into the periodontal ligament toward the root apex, often using a mallet and light tapping force. This process continues along the crestal one third of the tooth. At the completion of this procedure, the tooth is often slightly mobile (see Figure 37-4, *B*).

Once the periotome is used as a moving wedge, it may then be converted to a lever (see Figure 37-4, *C*). The blade of the periotome is often 3 to 4 mm wide. When the handle is rotated, one side of the periotome is applied to the tooth root, the other side to the cribriform plate, and the width of the "wedge" is now the length of a lever, which magnifies the rotation force (moment). The rotation of the periotome handle increases both tooth mobility and the force against the opposite cortical plate to further expand it within physiologic limits.

A single-rooted tooth is most often tapered. As the periotome is tapped down to the cribriform plate and slightly rotated, the tooth often is pushed against the opposing cribriform plate. Because the socket is tapered, the lateral force on one side of the tooth is converted to a coronal direction force on the other side and the root is pushed out of the socket. As a result, the periotome may now be pushed farther apical,[1] toward the root apex. When time elapses between each force application, the tooth may even slide up and completely out of the socket. Additional time and elevation may be required if significant tooth mobility is not achieved.

A traditional dental forceps should not be applied to the tooth until significant tooth mobility is achieved. Once the wedge and lever action of the dental elevator is applied to a tooth, most often a dental forceps is used to ultimately grasp and deliberately rock the tooth back and forth and to rotate it as much as conditions will allow. The combination of these tooth movements expands the bony socket and separates the periodontal ligaments. As a consequence, the tooth may be removed (see Figure 37-4, *D*).

Conventional dental forceps are really two first-class levers connected with a hinge. The forces applied to the forceps handles are the long side of the lever and the beaks on the tooth are the short side of the lever, with the hinge acting as a fulcrum. The force on the handles is magnified to allow the beaks of the forceps to grasp the tooth with great force. None of the force on the forceps handles is used to extract the tooth. Rather, the increased force on the forceps beaks often crushes or fractures the tooth. The forceps hold the tooth, and the surgeon's hand, wrist, and arm are used to move and extract the tooth. This action would be similar to forcibly pulling a bottle cap off a bottle or pulling a nail from a piece of wood, using only a pair of pliers.

The principles of biomechanics are the basis for the development of a different type of dental forceps called *Physics Forceps* (Golden-Misch Instruments, Detroit, Mich.). A *moment of force* in physics represents the magnitude of force applied to a rotational system at a distance from the axis of rotation. The principle of moment *M* is derived from Archimedes's discovery of the operating principle of the lever and is defined as

$$M = rF$$

where *F* is the applied force and *r* is the distance from the force applied to the object. The concept of a moment arm is key to the operation of the lever, which is capable of generating mechanical advantage. This means that the force applied to an object is affected by the length of the lever arms. The lever arm is the distance from the force input to the fulcrum or from the fulcrum to the force output.

The Physics Forceps is a dental extractor that uses first-class lever mechanics. One beak of the device is connected to a "bumper," which acts as a fulcrum during the extraction. The bumper is placed most often on the facial aspect of the dental alveolus, at or above the mucogingival junction. The second beak of the extractor is positioned as low as practical on the tooth root, most often on the palatal (lingual) into the gingival sulcus.

Once the treatment is in position around the tooth root, no squeezing pressure is applied to the tooth. Instead, the handles, once in position, are rotated as one unit facially for a few degrees and stopped for approximately 60 seconds (Figure 37-5, *A*). The torque force generated on the tooth, periodontal ligament, and bone is related to the length of the handle to the bumper (8 cm), divided by the distance from the bumper to the forceps beak (1 cm). As a result, a force on the handle connected to the bumper will increase the force on the tooth periodontal ligament and bone by eight times. No force is required to be placed on the forceps beak, which is on the tooth. Therefore the tooth does not split, crush, or fracture. The 60 seconds of constant force cause biomechanical creep into the bone and periodontal ligament. Once creep has expanded and weakened the periodontal ligament and bone, the forceps handle may be slowly rotated another few degrees. This usually releases and elevates the tooth a few millimeters from the socket within an additional 10 seconds (see Figure 37-5, *B*). At this point the tooth is loose and ready to be removed from the socket using any pincer-like device (e.g., pick-ups, an extraction forceps, a hemostat) (see Figure 37-5, *C*).

The extraction of a tooth using the Physics Forceps is similar to the removal of a nail from wood with use

WWW.HIGHDENT.IR

همیار دندانسازان

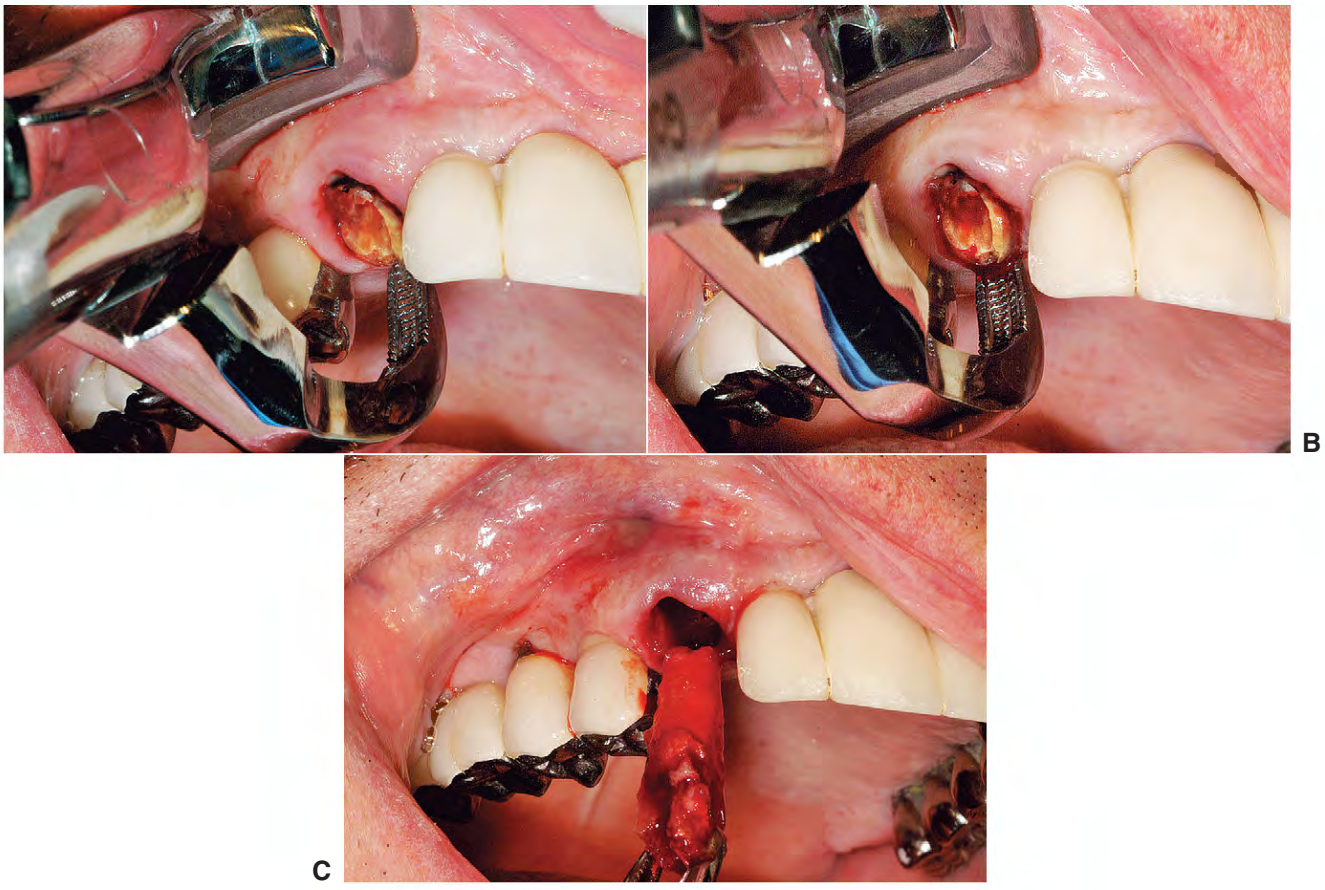


Figure 37-5 **A**, The Physics Forceps has a "bumper" on one beak, which is placed below the tooth and acts as a lever. The other beak is placed on the tooth root. The handles of the Physics Forceps act as a lever to rotate the tooth from the socket. The bumper is placed below the tooth, usually at or above the mucogingival junction. The beak is placed low on the tooth root in the gingival sulcus. **B**, Once in position on the tooth root, the Physics Forceps is used as one unit (no squeezing of the handles). A few degrees of rotation to the facial places moment force on the tooth, which is held for 60 seconds. Another few degrees of rotation are then applied, and the tooth releases and elevates. **C**, Once the tooth releases from the socket, it is removed with a pincer-like device (e.g., pick-ups, extraction forceps, hemostat).

of a carpenter's hammer (instead of pliers). The handle of the hammer is a lever, and the beaks of the hammer fit under the head of a nail (they do not squeeze the head). The hammer head acts as a fulcrum. A rotational force applied to the hammer handle magnifies the force by the length of the handle, and the nail is elevated from the wood.

Unlike a nail in wood, which is parallel and has friction for its full length, a tooth is tapered. Therefore, after it is elevated a few millimeters, the periodontal ligament fibers are broken and the tooth may be easily removed, without additional rotational force. This is important to note, as further rotational force on the forceps may fracture the facial plate of bone.

Creep is a phenomenon whereby a material continues to change shape over time under a constant load. In tooth extraction, creep may occur to bone and the periodontal ligament. Reilly established the creep curve of bone, whereby under a constant load of 60 Mpa, the bone over time responds in three different stages.[6] The majority of bone changes occur within the first minute, whereby the initial strain of bone (the change of length divided by the original length) is modified. The greater the force, the greater the deformation of the bone. This process allows the tooth socket to expand and the tooth to exit the socket.

A secondary creep curve allows the bone to further deform when the force is applied for 1 to 5 minutes. The longer the time, the greater the deformation. However, the secondary deformation is only a 10% to 20% difference compared with the initial strain over the first minute. Eventually, the bone will fracture if the load is applied over a longer time frame, representing creep rupture.

The creep curve of the periodontal complex is similar to the creep curve of the bone, whereby the constant load on a tooth over time increases the strain and decreases the strength of the periodontal complex. Therefore, the clinician should not underestimate the values of time and constant force to the tooth ligament and bone in the extraction process.

HEALING OF EXTRACTION SOCKETS

Once the tooth is extracted, the site may heal by repair or regeneration of the bone. All fibrous tissue

WWW.HIGHDENT.IR

from periodontal disease or endodontic origin should be completely removed, as these tissues impair bone formation and delay bone healing for extended periods. Repair occurs when there is injury or conditions of the bone that cause incomplete bone volume to form in the residual ridge. The most common conditions that cause repair are the absence of a labial plate before or as a consequence of tooth extraction. Other factors include a bony wall that is less than 1.5 mm thick (usually the facial), exudate, gross apical pathology, or excessive heat from a dental drill during root extraction. On rare occasions, bisphosphonate drugs or systemic conditions may cause a repair rather than a regeneration process.

The tooth socket with five bony walls regenerates bone by secondary intention, and bone healing in many aspects is similar to secondary intention soft tissue healing. The healing sequence in both hard and soft tissue includes inflammation, epithelialization, fibroplasia, and remodeling. However, socket healing presents unique microvascular features and a sequential pattern of bone formation before remodeling. The inflammatory stage of healing is initiated by the extraction trauma. Oral epithelium surrounds the crestal aspect of the socket and averages 3 mm in thickness in the absence of periodontal disease (the sulcus, junctional epithelial attachment, and connective tissue attachment accounting for approximately 1 mm each). The cribriform plate is composed of cortical-like bone and, after extraction, it is covered with the residual periodontal ligament. The vessels torn during the extraction fill the socket with blood, which coagulates and protects the bone during initial healing. The epithelium around the crest of the alveolus migrates down the socket walls during the first week. The migration continues until it reaches the bed of granulation tissue situated under the blood clot. It then migrates over this granulation tissue until it makes contact with the epithelium migrating from the other sides.

Ohta has proposed four stages of bone regeneration after a tooth is extracted with a healthy surrounding alveolus.[7] The time frames presented here have been modified from his dog study to reflect the human sigma of bone. The week following extraction is called the *initial angiogenic stage*. Initial angiogenesis develops from the broken ends of blood vessels in the residual periodontal ligament covering the cribriform plate. Blood plasma leaks from these broken vessels, and immature fibroblasts aggregate at the plasma-rich regions. The blood clot begins to shrink, and capillaries form sinusoids and granulation tissue, starting from the apex and the surrounding walls of bone. Fibroplasia begins early in the sequence during the first week as a result of the ingrowth of capillaries and fibroblasts. White blood cells kill bacteria and begin to dissolve foreign bodies and bone fragments. With few exceptions, the angiogenesis begins at the bottom of the socket because this area is not severely injured during the extraction and has the greatest source of blood vessels. Within 5 days of the extraction, early granulation tissue composed of immature capillaries and fibroblasts appears at the bottom of the socket and spreads upward along the socket walls. New bone trabeculae also form at the apical portion of the socket during this early stage.

The new bone formation stage begins as early as the third week after extraction.[7] At this time, the entire socket is filled with granulation tissue. This period demonstrates the greatest sinusoid formation activity. The forming trabeculae of woven bone first start from the bottom of the socket following the meshwork of newly formed anastomosing sinusoidal capillaries. Bone formation is more rapid at this point, creating a three-dimensional lattice pattern of woven bone. It has been observed that, during this time, the cortical bone of the marginal crest of the socket continues to resorb, especially in the interseptal regions and the thinner facial plate.

The bone growth stage starts 4 to 5 weeks after the extraction.[7] New bone trabeculae forming on the walls and apical region have thickened and fill the apical two thirds of the socket. The center of the socket is still woven bone because ossification occurs on randomly formed collagen fibers; hence its lattice appearance. The more organized lamellar bone starts to form from the lining of the socket toward the center of the socket.

The bone reorganization stage occurs at least 6 weeks after extraction.[7] The primary bone trabeculae remodel to form thicker secondary spongiosa. This process always begins at the apex of the extraction socket. The complete cortical lining of bone around the socket is not fully resorbed, and remodeling continues for 4 to 6 months after the initial extraction (Figure 37-6).

The timing for these four stages varies among individuals and clinical situations. The number of bony walls around the socket and size of the alveolus greatly influence the regeneration process. Molar sites take longer to completely form bone compared with smaller-diameter anterior sites. Although the period of regeneration for an extraction socket is variable, the clinical sign that the socket regeneration is complete is when the radiographic lamina dura (which represents the cribriform plate) is no longer present. This period is most often 3 to 6 months, dependent upon tooth size, root number, and trauma of extraction.

SOCKET GRAFTING

Multiple bone graft procedures and studies have been evaluated for socket augmentation at the time of extraction. However, variable results have been observed, not only among different reports, but also within each study.[8-11] Rather than use the same technique regardless of clinical conditions, when bone repair rather than regeneration is likely, the clinician should provide as many keys to bone grafting as possible to increase the socket.

WWW.HIGHDENT.IR

همیار دندانسازان

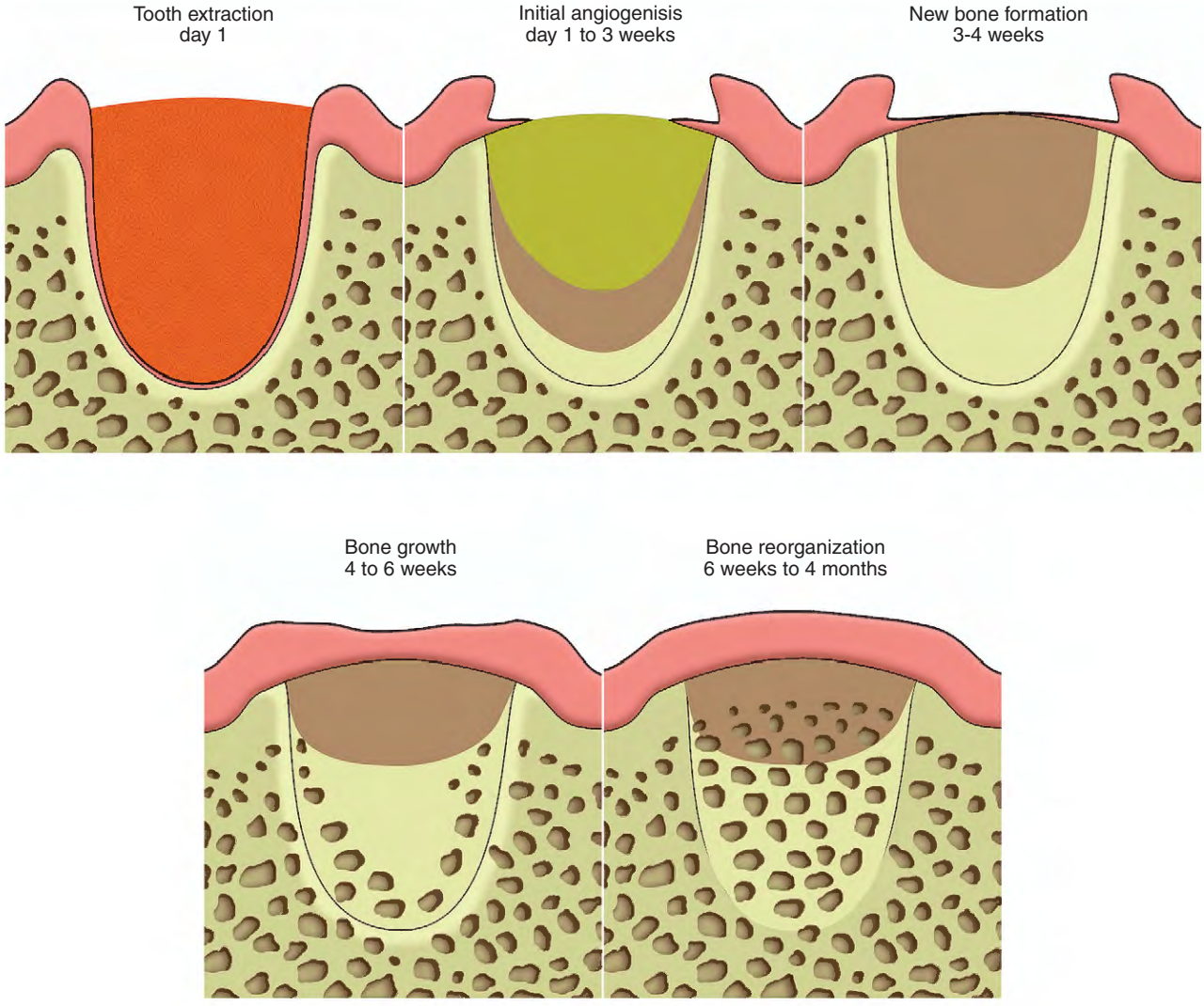


Figure 37-6 The four stages of healthy socket healing include: initial angiogenisis, new bone formation, bone growth, and bone reorganization. The complete process often takes 4 to 6 months.

In 1993, Misch and Dietsh suggested different graft materials and techniques based on the number of bony walls that remained after the tooth is removed.[12] A thick five bony wall defect will grow bone with almost any resorbable graft material (RGM); for example an alloplast, allograft, or autograft. When a wall of bone is less than 1.5 mm or a labial plate is missing (four bony wall defect), an autograft or an alloplast or freeze-dried bone (FDB) with BM and GBR increased the predictability of restoring the original bony contour. Becker et al. evaluated demineralized freeze-dried bone (DFDB) alone in extraction sockets, and no evidence of bone formation was observed.[13] It appears DFDB alone may be a poor choice for socket grafting. LeKovic et al. compared extractions alone to BM with extractions. At 6 months, crestal bone loss (0.38 versus 1.50 mm) and horizontal ridge resorption (–1.31 versus –4.56 mm) were found.[14] A two or three bony wall defect requires an RGM and, at least, some autogenous bone with a barrier membrane. A block graft of cortical autogenous bone fixated into the host bone position is suggested for one bony wall defect (Figure 37-7).

Thick Five Bony Wall Defect

Regeneration restores complete morphology and bone volume to the residual ridge. This most often occurs when there are five thick bony walls around the extraction site. Most of the keys for predictable bone formation are present under these conditions, and the socket often forms bone in the extraction socket without loss of width or height. The atraumatic extraction of a tooth without pathology provides many of the keys necessary for predictable bone regeneration. The extraction process sets up a regional acceleratory phenomenon (RAP) for healing (which increases the rate of repair and adds bone morphogenetic protein [BMP] to the site); the five bony walls protect the graft from mobility; the torn blood vessels in the periodontal complex leak growth factors into the region (platelet-derived growth

WWW.HIGHDENT.IR

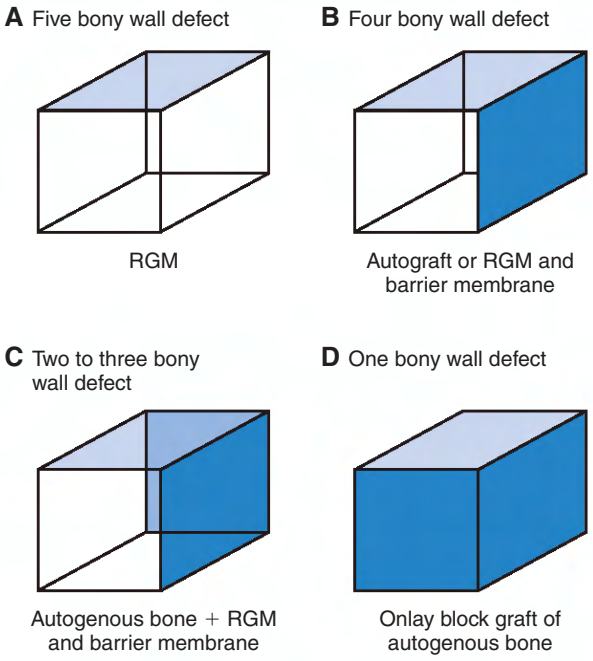


Figure 37-7 The graft materials and techniques for socket grafting are related to the remaining number of bony walls. A thick five wall bony defect may use any resorbable graft material (RGM). Four wall defects require an autograft or mineralized alloplast, allograft, and barrier membrane. A two or three wall bony defect may use some alloplast/mineralized allograft, but should use autograft and a barrier membrane as well. A one wall bony defect is most predictable with a cortical autograft fixated to the host bone.

factor [PDGF], transforming growth factor [TGF]); the space is maintained by the five walls of the bone; the bony walls provide blood vessels from bone into the site; and the defect size is small (i.e., one tooth). As a result, the only key initially missing is soft tissue closure (Figure 37-8). The soft tissue around the extraction site begins to grow over the clot and granulation tissue of the socket and within 2 to 3 weeks covers the site. For example, a mandibular third molar extraction site often has thick lateral walls of bone, and most often the bone area is regenerated in both height and width with no graft material or regeneration technique in the sockets.

Four to Five Wall Bony Socket

When a labial plate around a socket is missing, the absence of the wall prevents space maintenance, reduces host bone vascularization, and replaces it with soft tissue vascularization. The facial bone level will never grow above the height of bone on the facial cortical plate of the tooth. Bone augmentation procedures must be used to obtain an ideal volume and contour of bone. Sockets with a missing lateral wall are significantly compromised and heal by repair rather than regeneration.

When conditions of repair instead of regeneration are present, socket grafting for ridge augmentation at

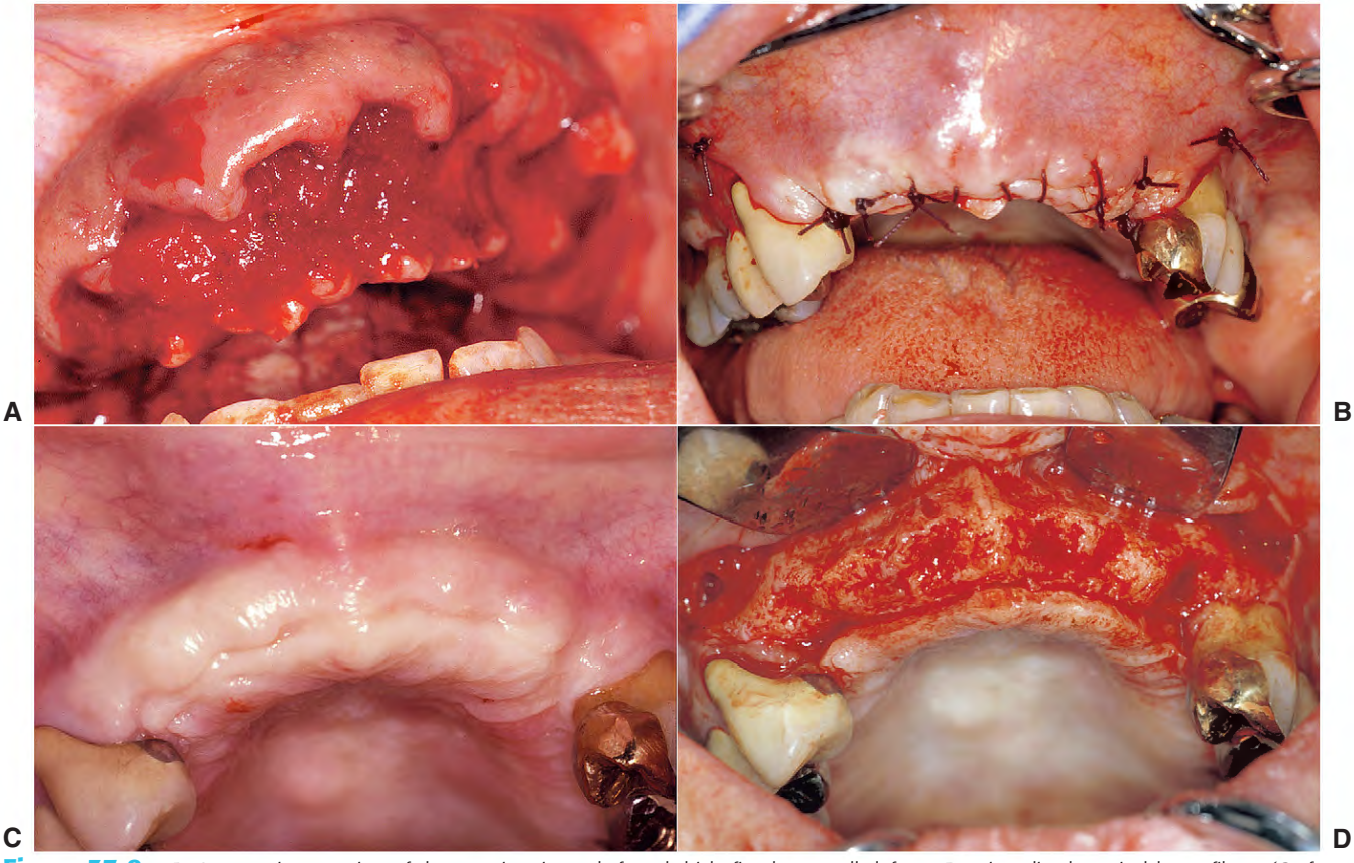


Figure 37-8 **A,** Atraumatic extraction of the anterior six teeth found thick, five bony wall defects. Demineralized, cortical bone fibers (Grafton, Lifecell) are placed into the sockets. **B,** The soft tissue is approximated for primary closure. **C,** The soft tissue healing after 5 months. **D,** Reentry into the residual premaxillary ridge observes abundant bone and ridge preservation.

Continued

WWW.HIGHDENT.IR

همیار دندانسازان

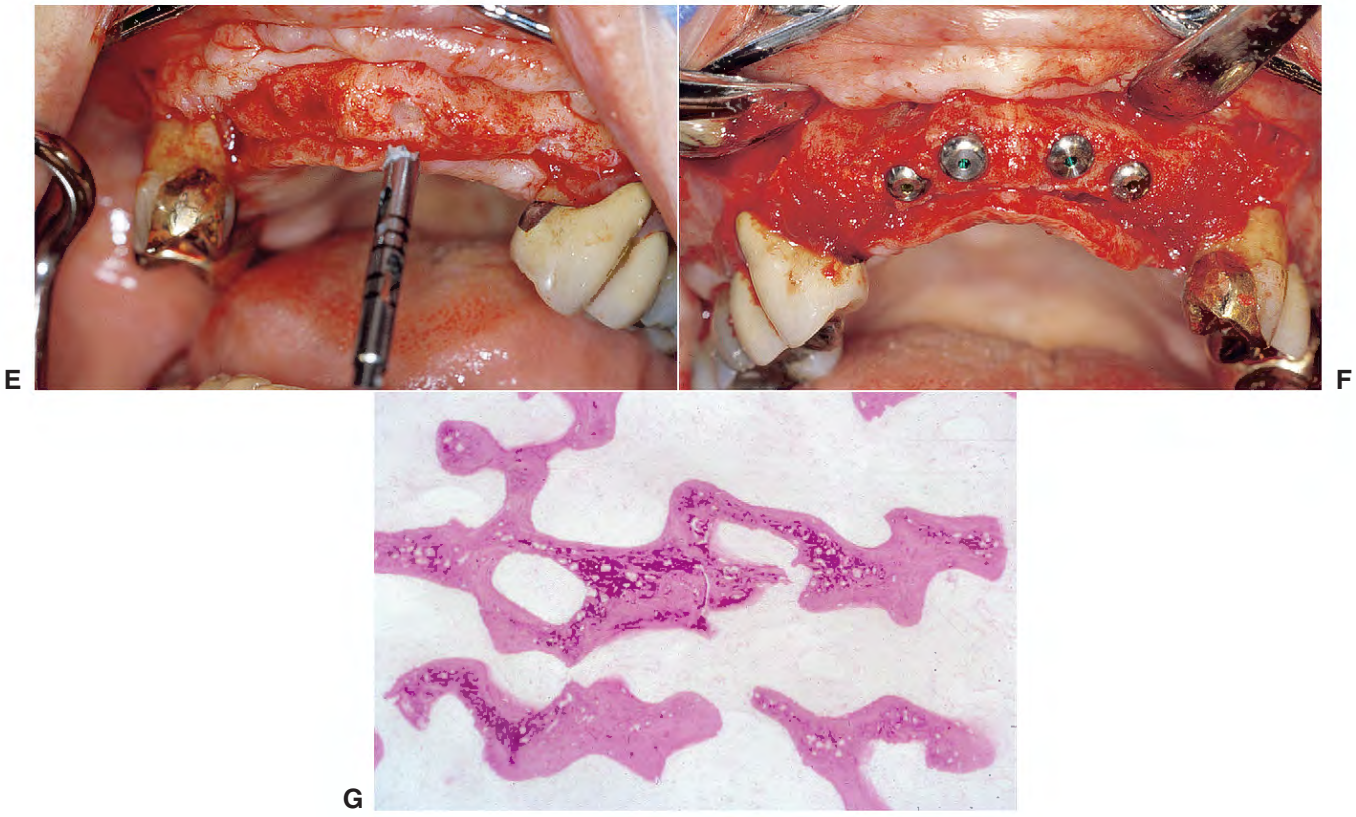


Figure 37-8, cont'd **E,** A core biopsy is taken from a central incisor site. **F,** Implants are inserted into the premaxillary arch. **G,** Histology of the core indicates primarily vital bone trabeculae and some residual demineralized freeze-dried bone at 5 months. *(Courtesy M. Sharawy.)*

the time of extraction is indicated.[12] Tooth extraction without grafting in these conditions results in residual bone loss as a result of resorption. The maxillary anterior region may be reduced 23% in the first 6 months after an extraction and another 11% over the following 5 years.[15] Within 2 years, an average of 40% to 60% of the original height and width of bone may be lost with multiple extractions.[16]

The first determination after the tooth extraction is complete is the assessment of the thickness of labial and palatal plates of bone and their relative height to the ideal volume desires. When one of the lateral plates of bone is thinner than 1.5 mm, or when height is desired, a socket graft is indicated, even in the presence of five bony walls. A similar socket augmentation procedure also may be used when the labial plate of bone is missing. The two techniques of choice are a BM with a mineralized alloplast/freeze-dried bone (FDB) socket fill or a modified socket seal surgery.

Barrier Membrane with Alloplast/ Freeze-Dried Bone

An acellular dermal matrix is selected for a BM when soft tissue augmentation is also desired, or a collagen BM is used when the soft tissue drape is not an issue. A periotome or thin periosteal elevator is used to tunnel under the final bone periosteum and lift the soft tissue off the bone over the thin bony wall. This tunnel should extend several millimeters beyond the desired augmentation site. A BM is then slid into the "pocket" created under the tissue and extends apical, mesial, and distal beyond the extraction site. Approximately 6 to 8 mm of the BM should extend above the marginal tissue.

When the facial plate is thin, the socket may be filled with FDB (e.g., MinerOss, Puros) or a mineralized hydroxyapatite (HA) source (e.g., BioOss, Osteograf-N). When the labial plate is missing, the FDB may be placed in the apical portion, but particulate autologous bone should be placed in the crestal half of the socket. The walls of bone on the mesial, distal, and palatal provide bone blood vessels to this autograft. The extension of collagen or AlloDerm then covers the top of the socket and is tucked below the palatal tissue. Sutures are then placed over the top of the BM. Primary closure of the soft tissues is not obtained, as the tissues would need to be reflected and advanced over the socket, which would affect the soft tissue drape (Figure 37-9, *A* to *H*).

The extraction site may be reentered after 4 to 6 months. The clinical time for reentry is determined by the absence of the cortical lining of the socket (cribriform plate) on a periapical radiograph. Once this has occurred, the implant may be inserted and a

WWW.HIGHDENT.IR

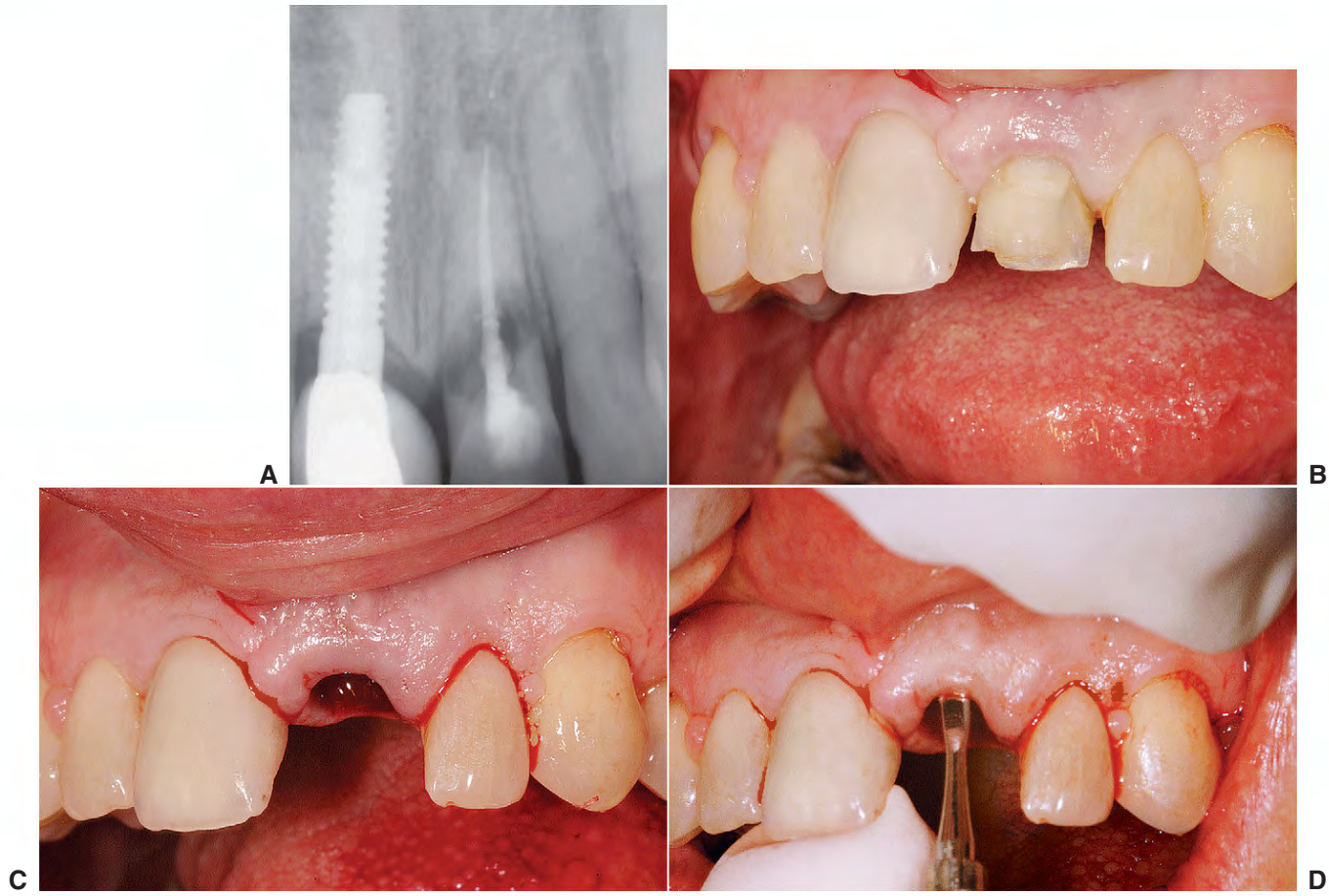


Figure 37-9 **A,** A radiograph of endodontically treated maxillary central incisor with internal resorption and apical radiolucency. **B,** The central incisor crown is reduced on the mesial and distal to favor the atraumatic extraction. **C,** The tooth is atraumatically extracted without soft tissue reflection. The labial wall is less than 1 mm thick. **D,** A periotome forms a tunnel on the facial cortical bone and extends mesial, distal, and apical to the contours of the extraction site.

Continued

regular protocol of healing and restoration used (see Figure 37-9, *I* to *N*).

Maxillary Tuberosity Donor Site

The tuberosity offers a variable amount of trabecular bone. This area is convenient for use in maxillary sinus grafting[12,17,18] and also may be considered for smaller areas of ridge augmentation[19,20] (Figure 37-10). The cancellous nature of the bone allows it to be molded into an alveolar defect, such as an extraction socket.[12] However, the trabecular graft will more often require the use of a barrier membrane in onlay grafting to minimize resorption and stabilize the graft.[21] The tuberosity autograft has growth factors for osteoinduction and to accelerate blood vessel growth in the host site.

The thicker soft tissue in the tuberosity region can mislead the assessment of this donor site. The tuberosity should be evaluated with panoramic or periapical radiographs. The anatomical limitations of this area include the maxillary sinus, pterygoid plates, adjacent teeth when present, and the greater palatine canal. A vertical incision is made posteriorly at the lateral aspect of the maxilla. It extends anteriorly across the tuberosity into the molar region. After reflection of a mucoperiosteal flap, bone may be harvested from the tuberosity with a rongeur or chisel. Removing the graft with a chisel will allow the harvesting of a larger piece of bone. Although the sinus may inadvertently be entered during removal of the graft, coverage of the perforation with the thick mucosa should preclude the development of an oroantral communication. If this is observed at suture removal, the patient is instructed to avoid creating high nasal pressures and should be placed on antibiotics and decongestants to prevent infection and ensure normal drainage. Most often, within 1 month, the oroantral opening will close on its own.

Socket Seal Surgery

A composite graft socket seal surgery has been developed by Misch et al.[22,23] composed of connective tissue, periosteum, and trabecular bone used to seal a fresh extraction socket. A connective tissue graft has the advantage over a keratinized graft by blending into the surrounding attached gingival regions, offering similar color and texture of the epithelium. This is most advantageous in the maxillary anterior region and

WWW.HIGHDENT.IR

همیار دندانسازان

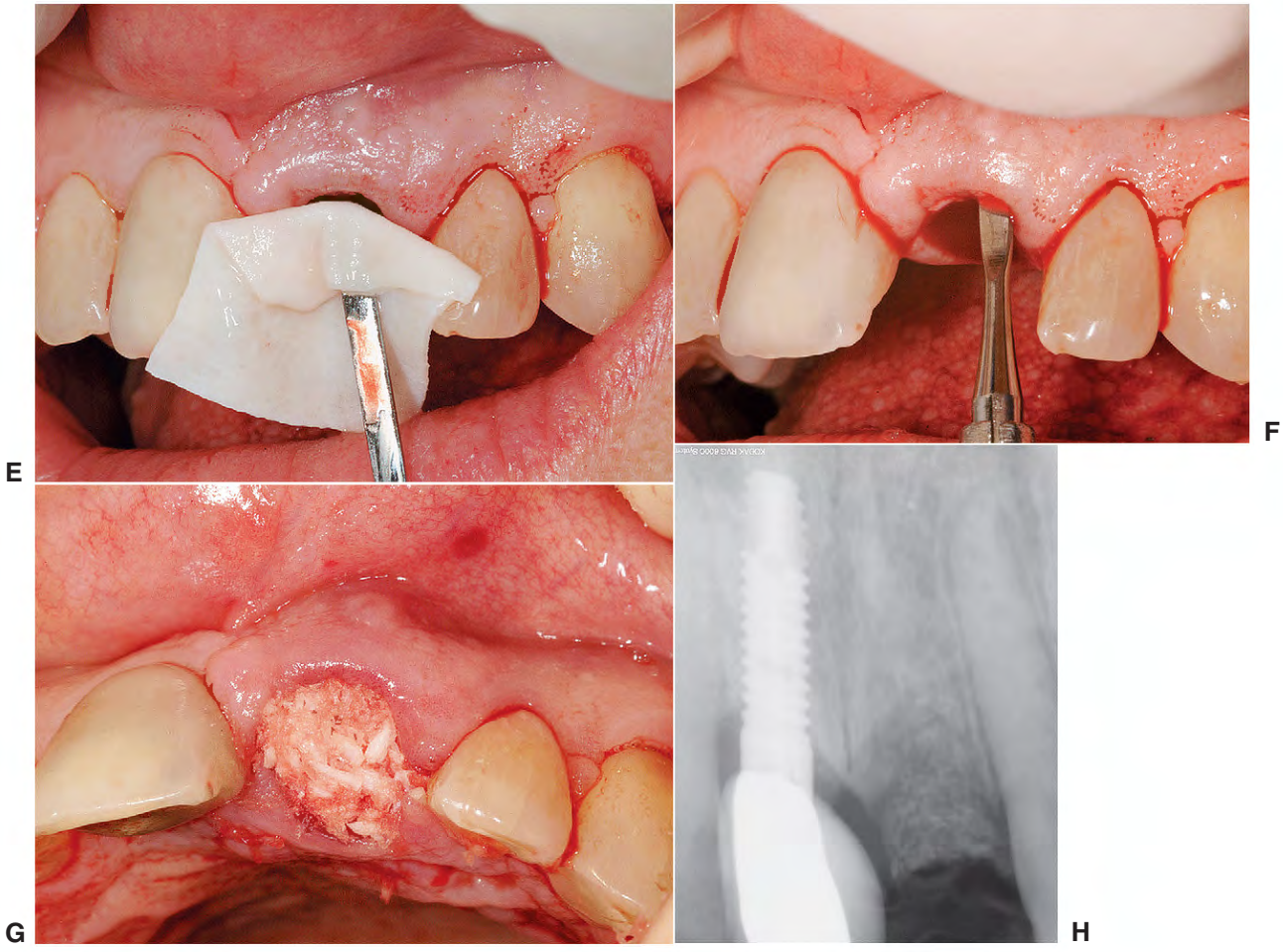


Figure 37-9, cont'd **E,** A piece of acellular dermal matrix (Alloderm) is in position, draped over a periotome. **F,** The acellular dermal matrix is pushed into the facial tunnel and acts as a barrier membrane. **G,** The extraction socket is filled with a resorbable graft material (DFDB/FDB), consisting of a mixture of 30% demineralized freeze-dried bone (Grafton) and 70% mineralized freeze-dried bone (MinerOss). **H,** Postoperative radiograph demonstrates the extraction site and graft material in place.

Continued

other aesthetic areas. The composite graft also contains autogenous bone. The major advantage of autologous bone is a more rapid and predictable bone formation via osteogenesis. This technique may be used any time a tooth is extracted and an implant is planned as replacement.

A 6- to 10-mm trephine bur corresponding to the extraction site diameter is used in a slow-speed, high-torque hand piece to harvest a gingival graft with underlying bone. The most common site for the intraoral composite graft harvest is the maxillary tuberosity region (see Figure 37-10). The trephine drill drills through the unreflected, keratinized, attached gingiva and into the bone of the tuberosity region at the prescribed depth related to the thickness of the tissue and the amount of donor bone available. Care should be exerted not to enter the antrum, but that is of little consequence if the antrum is healthy during the process. A trephine bur may be used as a lever to greenstick fracture the bone core from its base, once it is in position within the bone. A Molt elevator may also be used for this purpose. The bone core (usually 5 to 10 mm in height) and the attached soft tissue (about 3 mm in height) is trimmed of its epithelium with a tissue scissors, leaving 3 to 6 mm of connective tissue attached to the bone core.

If the bone core does not fill the extraction socket completely, a mineralized bone graft material (e.g., FDB, bovine bone) may be used in the apical portion of the socket, provided the labial plate is still intact. Because the new bone forms from the apical portion of the socket, this is the least important region to augment. If no bone plate remains in the apical half of the socket, additional autogenous bone should be harvested from an additional intraoral site to overfill the apical half of the socket. The bone of the composite graft (connective tissue attached to periosteum and bone) is compressed and fitted into the remaining portion of the socket. The tissue of the composite graft will seal the socket and remain above the surrounding gingiva. A mallet and blunt instrument should be used to tap it into place and compress the bony core to conform to the crestal contour of the socket. The connective tissue portion of the graft

WWW.HIGHDENT.IR

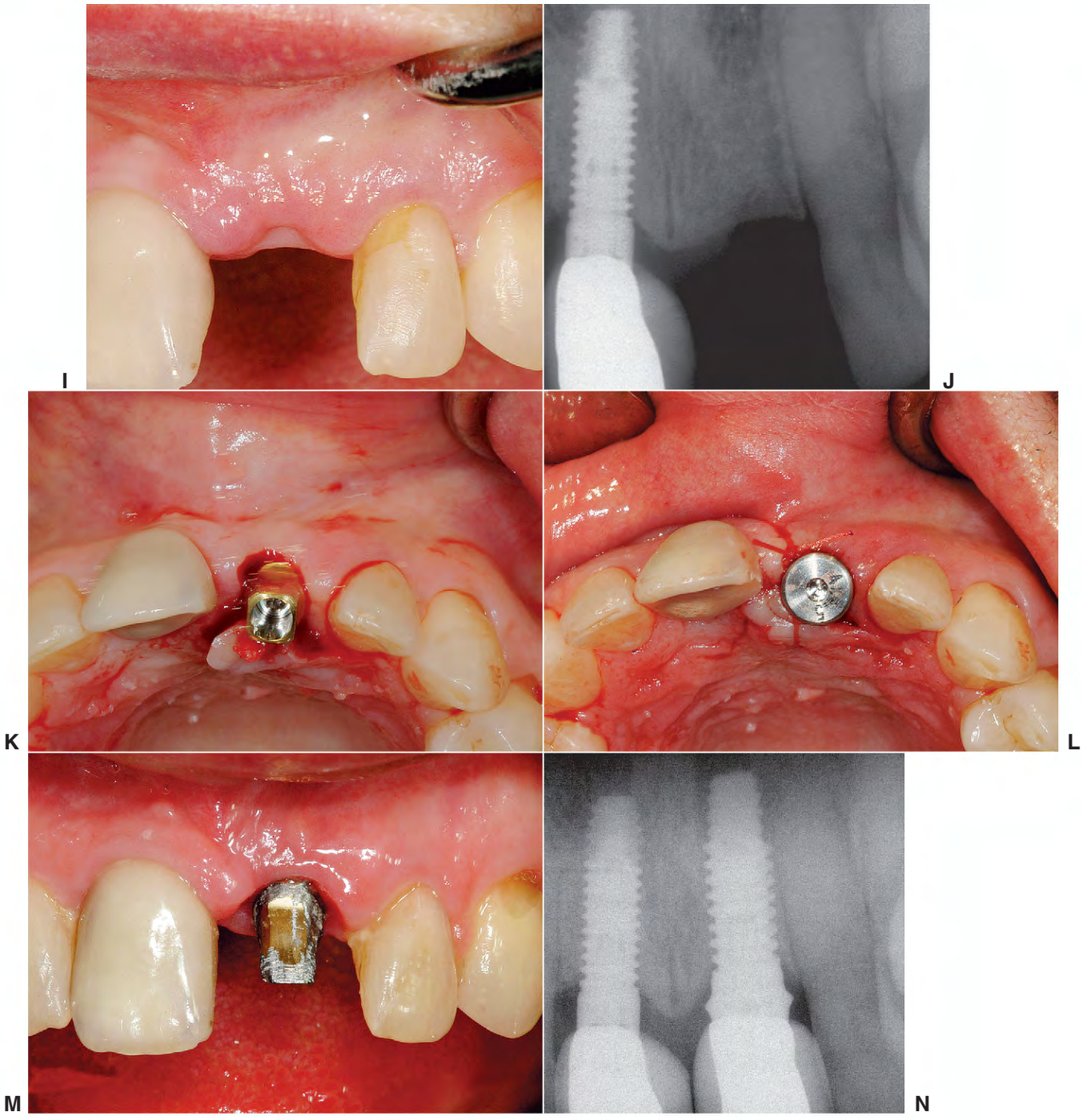


Figure 37-9 *cont'd* **I,** The soft tissue healing after 5 months. **J,** A periapical radiograph indicates the cribriform plate of the extraction socket is no longer visible. This indicates the bone reorganization stage of socket repair is complete. **K,** An implant is inserted into the central incisor site. A "split-finger" tissue flap on the palate is rotated to the mesial. **L,** A permucosal extension is inserted into the implant body and the tissues approximated. **M,** After 4 months of healing, an abutment is placed into the implant body. **N,** A periapical radiograph 9 months after implant insertion. The interseptal bone is maintained adjacent to the implant.

is then sutured to the surrounding gingival tissue with facial and palatal interrupted 4-0 PMA or Vicryl sutures (Figure 37-11). A removable transitional prosthesis should not be permitted to load the tissue during the first few weeks after extraction; otherwise, the composite graft may become mobile and sequestrate. A fixed transitional restoration may use an ovoid pontic design to maintain the interdental papilla height and contour.

The benefits of the composite graft socket seal surgery technique permit the surrounding keratinized gingival tissues to migrate and form a similar color and texture of keratinized tissue over the socket. The blood supply to the composite graft is established from the surrounding soft tissue. In addition, because autogenous bone is used as the graft in the coronal half of the socket where the facial bone is most often very thin or

WWW.HIGHDENT.IR

همیار دندانسازان

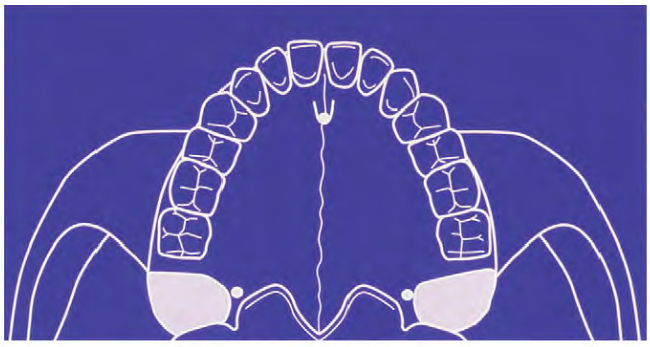


Figure 37-10 The maxillary tuberosity region is a common bone donor site for bone grafting, when trabecular bone is the desired product. Trabecular bone has growth factors for blood vessels and for regeneration of a bone defect.

absent, more predictable results will result than if an allograft were used. The transfer of the bone graft with an intact periosteal layer expedites revascularization and may decrease the healing time.[24,25] As a result, reentry may be in 4 to 5 months, and placement of an ideal implant diameter is often made possible (Figure 37-12).

Two to Three Bony Wall Defects

A two to three bony wall defect is treated very similar to a four bony wall defect. However, because the defect size is larger, more autograft is required in the bone graft. Rather than using the autograft primarily in the crestal region, it is of benefit that the entire first layer of the resorbable graft materials be an autograft. As a consequence, more often a donor site from the mandible is required.

The most common two to three bony wall defects are extraction sites missing more than the labial bony wall. Because the mesial distal bony walls are usually present, the host site is more predictable than a one bony wall defect. Incision line opening is less of a complication, as the residual ridge form has soft tissue support around the defect.

Trephine Bur Bone Harvest

Trephine burs are end-cutting devices of various diameters. Most often, a 6- to 10-mm trephine bur is selected. Once the bone harvest site is reflected, the trephine bur is used to harvest the autograft. When the ramus is selected, a larger trephine is usually selected. One half of the trephine bur is placed over the external oblique and bone, while one half is lateral to the bone and under the reflected masseter muscle, which is elevated off the anterior lateral aspect of the ramus. The trephine bur is used with a straight or angled hand piece at 2500 rpm with copious saline irrigation, 5 to 8 mm deep, making sure it is above and lateral to the position of the inferior alveolar nerve (IAN), artery, and vein. The IAN position may be evaluated on a panoramic radiograph or more precisely by computed tomographic (CT) reformatted images.

The next harvest site is above the half circle created by the first osteotomy and overlays the circle in the top third, or 3 mm from the top. This is repeated in the bottom third of the initial half circle, 3 mm above the bottom. Three interlacing semicircles are created along the external oblique of the ascending ramus. A large No. 8 round carbide in a straight hand piece then scores the lateral aspect of the ramus corresponding to the depth of the trephine bur semicircle cuts, 5 to 8 mm from the anterior border of the ramus. A chisel or surgical curette then may greenstick fracture the donor bone pieces from the ramus. These harvested pieces are usually the correct size to use in the graft site, as they are $5 \times 3 \times 5$ mm large. A collagen sponge (e.g., Avitene, Collatape) may be placed in the donor site and the tissues approximated for primary closure.

A trephine bur harvest over the apex of teeth or from a symphysis is usually with a 6- to 8-mm-diameter trephine. The trephine is used 5 to 8 mm deep, dependent upon the depth of the opposing landmark. Circular rings of the osteotomies are overlapped by 3 to 4 mm with a trephine bur. The first piece of bone is usually the most difficult to harvest. A wider slot is often drilled around the first segment to be harvested with a straight carbide drill (No. 557 surgical bur). The larger groove allows the curette or chisel to be introduced and leverage the piece of bone off the host site by greenstick fracture. Once the first piece is removed, the remaining pieces are tapped off the bone base with a chisel (Figure 37-13).

The two to three wall bony defect is filled with particulate autograft along the bony walls. When inadequate particulate autograft is harvested to completely fill the defect, a 30% DFDB and 70% mineralized bone (e.g., FDB) fills the remainder of the defect, preferably mixed with platelet-rich plasma (PRP). A barrier membrane is then placed over the site (Figure 37-14).

One Bony Wall Defects: Barrier Membranes and Guided Bone Regeneration

The concept of prosthetic-guided treatment plans has been presented as a method for achieving and maintaining predictable results when replacing the natural dentition. Because bone volume is often lost and the position of bone is modified rapidly after tooth loss, bone augmentation prior to or in conjunction with implant insertion is a common scenario when considering implant prostheses.

Several methods have been used to augment an edentulous site. There are advantages and disadvantages to each of the bone regeneration procedures. As a general rule, particulate grafts are easier to learn and to perform; they incur less incision line opening, have less postoperative discomfort from donor sites, have less altered nerve feeling from the donor site, may be more easily adapted to complex bone geometries in the host site, and may be used more easily at the same

WWW.HIGHDENT.IR

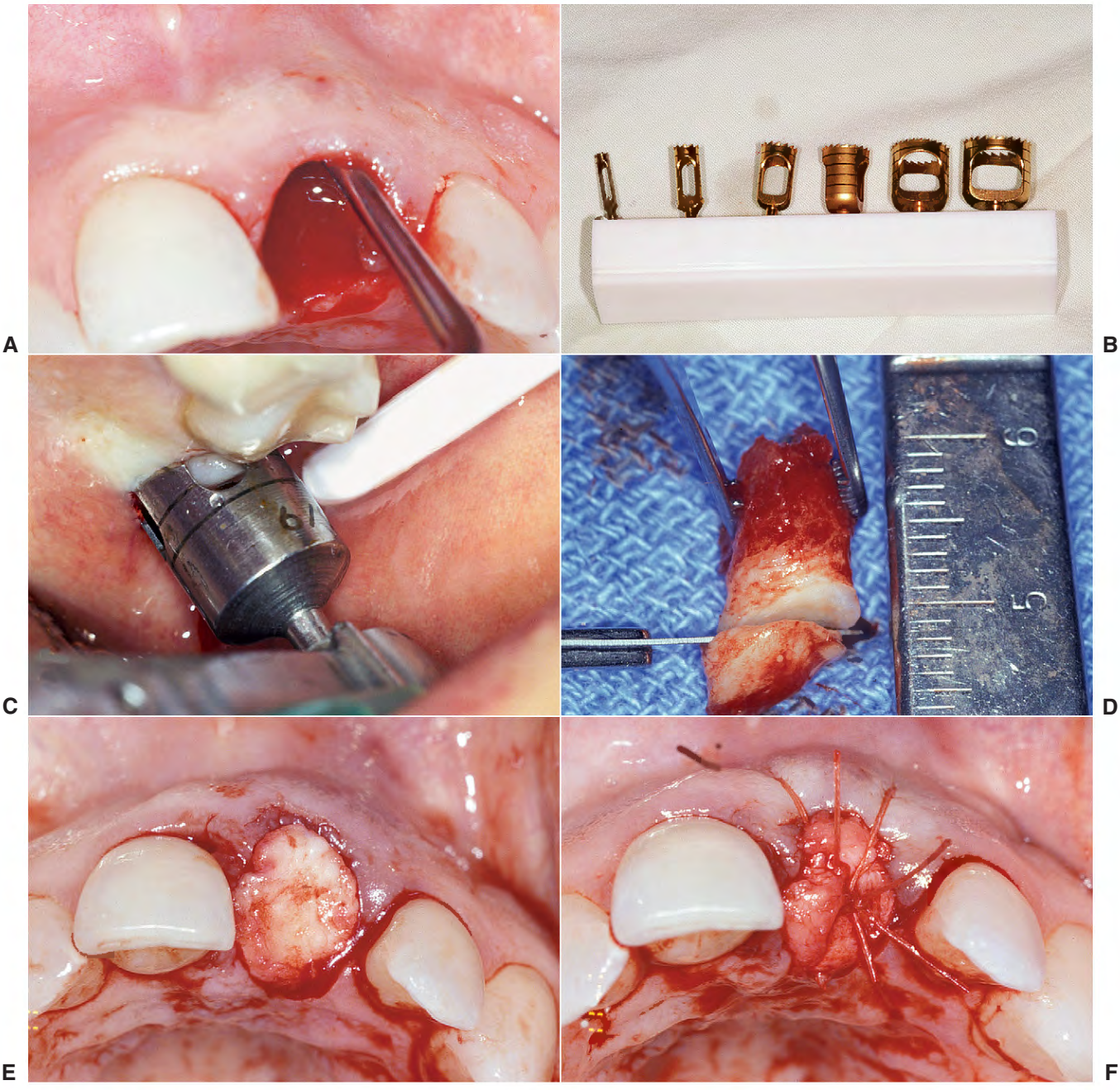


Figure 37-11 **A,** The left maxillary central incisor has an absence of facial cortical plate (a four bony wall defect). **B,** A trephine bur diameter is selected that corresponds to the size of the extraction site (Biohorizons, Birmingham, Ala.). **C,** The trephine bur performs an osteotomy directly through the keratinized attached gingiva and into the tuberosity and proceeds to the floor of the antrum. **D,** The keratinized tissue, mucosa, periosteum, and bone of the composite graft are removed from the trephine bur. The tissue thickness is reduced to 2 to 3 mm above the bone. The surface of the tissue is connective tissue (CT). **E,** The composite graft is inserted into the extraction socket and a blunt instrument (e.g., mirror handle) and mallet taps the composite graft into the socket so that the CT is level with the surrounding tissues. **F,** Sutures are positioned to maintain the composite graft in place.

time as implant insertion or for implant post-prosthetic bone loss. As a result, bone augmentation with GBR techniques is necessary to master.

There are disadvantages to using GBR procedures for all host defects or deficiencies. When bone augmentation is limited to only the GBR approach, limitations of treatment are observed. The larger the defect, the less predictable the graft. Bone height and width augmentation with BM are usually limited to less than 3 to 4 mm. Soft tissue contours are more difficult to predict. Extended healing times are necessary. The bone quality is often less than ideal. Full arches—GBR especially in the mandible—are not predictable. As a consequence, there is a place for both block bone grafts and GBR procedures when augmentation is required to fulfill the prosthetic-guided treatment plan.

WWW.HIGHDENT.IR

همیار دندانسازان

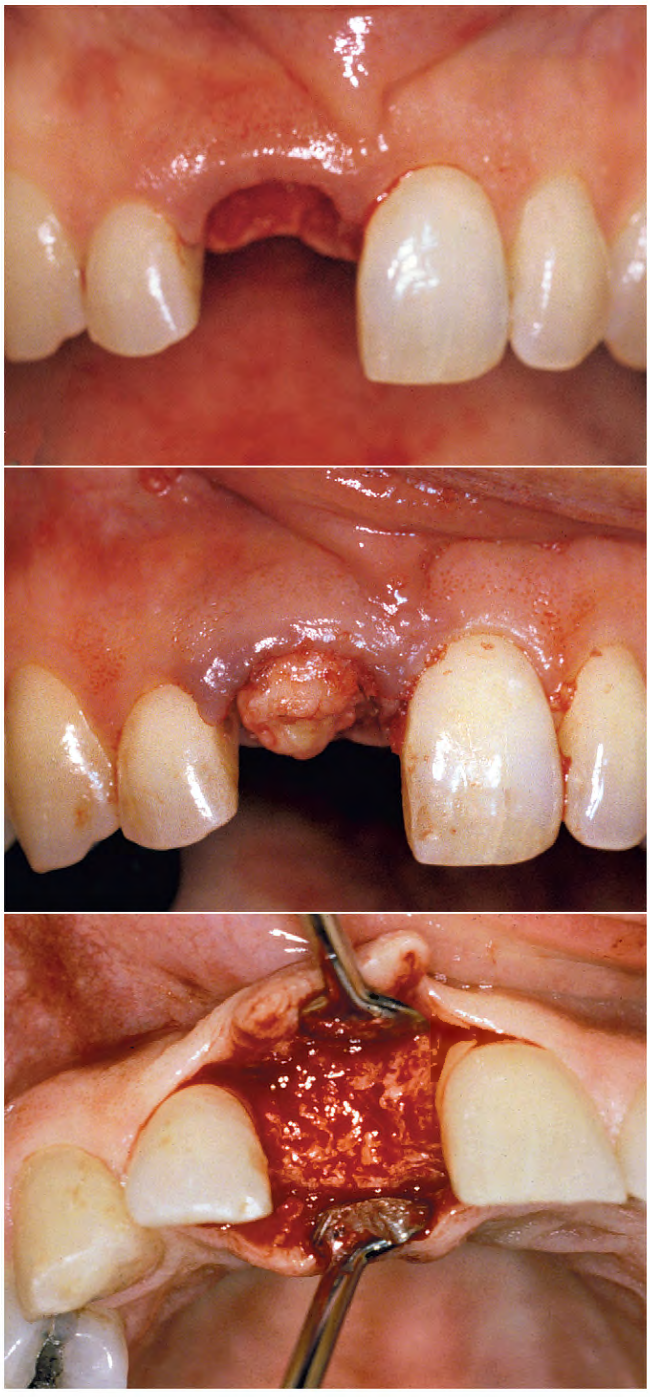


Figure 37-12 **A,** A maxillary central incisor is atraumatically extracted. **B,** A composite graft is positioned into the socket. **C,** After 4 months a reentry into the site observes the residual ridge has been augmented to the desired size.

The underlying concept of BMs has been used in medicine for more than 40 years.[26-33] The goal is to allow cell repopulation of a desired tissue to fill or regenerate a space and prevent unwanted cell types from entering the defect. The original concept for guided tissue regeneration was for nerve regeneration by leaving a tubelike chamber within a surrounding tissue connected to one end of the innervating nerve. The nerve could regenerate under these conditions and grow into the void to reinnervate the structures in the reconstructed defect.[26]

Tatum used a BM with particulate iliac crest bone grafts, placed in metal grids for jaw reconstruction before endosteal implant insertion in the early 1980s.[17] This technique was also used by Boyne with clinical success. The original clinical cases of BM and iliac crest particulate bone graft by Tatum and Boyne used nonresorbable cellulose filters (Millipore Filter, Millipore, Belford, Mass.).[34,35] However, both these clinicians abandoned the technique after several years because of a risk of increased incision line opening and associated risks of infection.

In the late 1980s, Gottlow et al. and Nyman et al. used Millipore, then expanded polytetrafluoroethylene (e-PTFE) (Gore-Tex, W.L. Gore, Flagstaff, Ariz.).[36,37] Most original clinical reports have used the nonresorbable e-PTFE membranes, and many reports have demonstrated predictable results.[36-39] Their research has led to a resurgence in guided tissue regeneration and GBR before or in conjunction with endosteal implants. However, the incidence of incision line opening is increased with this material, as the soft tissue does not readily adhere and attach. When incision line opening over a BM does occur, the amount of desired bone formation may be reduced as much as 60%. The cause of this decrease is often from bacterial contamination of the exposed membranes.[40-48]

The concept for GBR is to place a BM directly over a bone defect and under the soft tissue (including the periosteum) before primary closure. It has been accepted that the periosteum is a source of osteoblasts for bone formation and takes part in the bone augmentation process. However, this apparently is incorrect. When the periosteum is placed directly over a particulate bone graft, bone does not form under the periosteum. Instead fibrous tissue is observed on the surface. When a barrier membrane is placed over the particulate graft, bone is found. The new bone forms from the surrounding walls of host bone and follows the invading blood vessels from the host bone, which grow into the space provided by the membrane or particulate graft.

To create an ideal environment for bone modeling under the barrier membrane, the primary keys for bone grafting should be kept in mind. The GBR provides many of these, such as space maintenance, protection of blood vessels, clot with growth factors, graft stabilization, and exclusion of fibrous tissue in the graft. The barrier membrane should be placed over a particulate graft, rather than an empty or blood clot–filled space. A blood clot, in and of itself, does not participate in bone formation under the BM. Because a blood clot is 95% stagnant red blood cells and 5% platelets, the environment to grow bone is reduced, as red blood cells lyse and reduce the pH in the region.

A wide range of BM exists for GBR. The ideal properties of a BM include: (1) biocompatibility to hard

WWW.HIGHDENT.IR

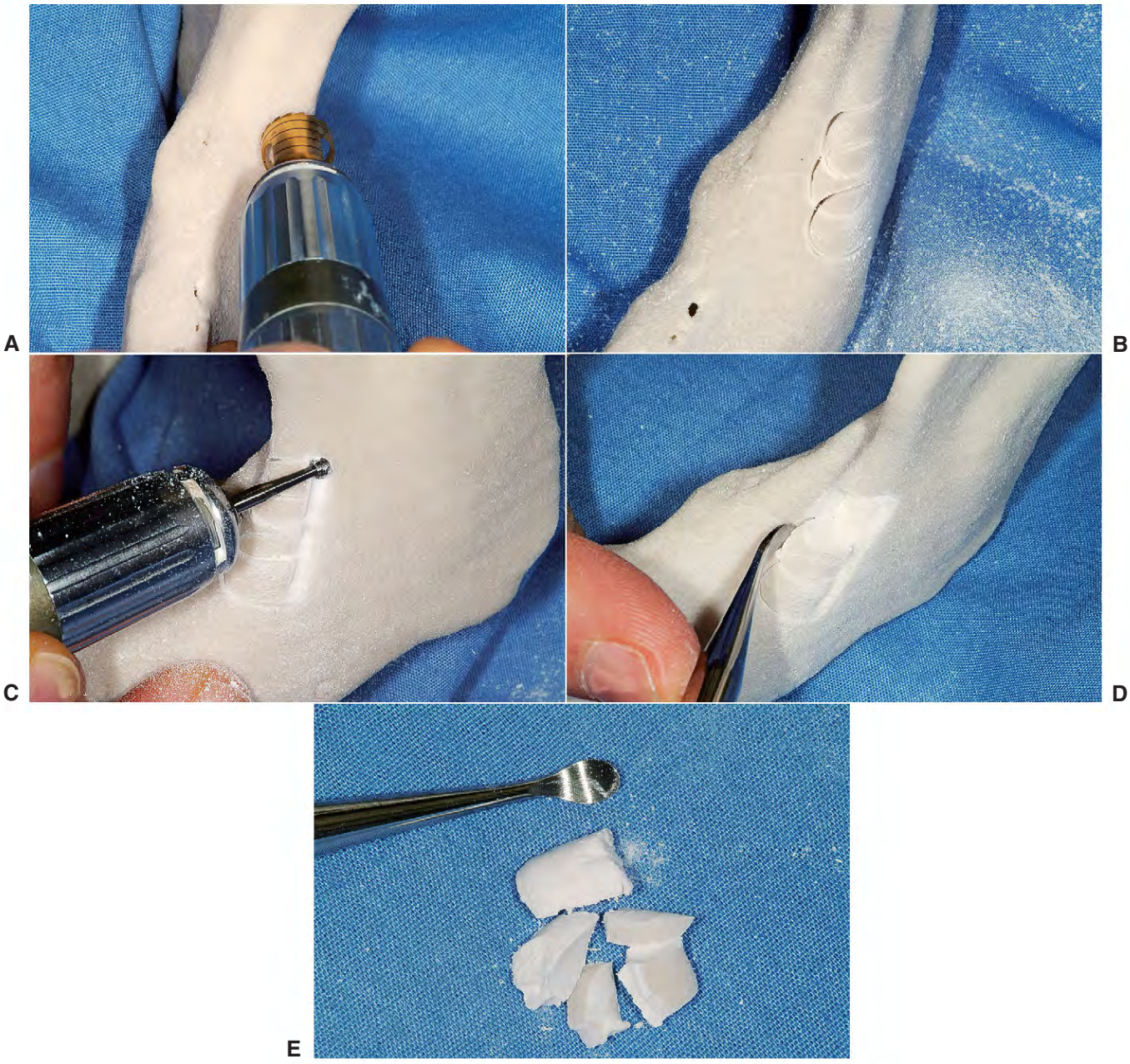


Figure 37-13 **A,** A trephine bur is used in the ramus to harvest autograft. Approximately half the trephine bur is placed on the lateral aspect of the ascending ramus, and penetrates the bone 4 to 8 mm. **B,** The osteotomy created by a trephine bur on the ascending ramus. Overlapping trephine bur osteotomies are made in the lateral aspect of the ascending ramus**. C,** A large round bur scores the lateral aspect of the ramus at the depth of the previous trephine bur osteotomy. **D,** A bone chisel is used to greenstick fracture the segment of particulate bone off the ascending ramus. **E,** The particulate bone pieces are 3 × 5 mm and used as the bottom layer of graft materials for the layered approach to BM GBR.

and soft tissues; (2) ability to occlude unwanted cell or tissue infiltration; (3) ability to maintain a space for the bone augmentation (4) ease of use to modify and place over the graft site; and (5) cost effectiveness.[40,41,46,48-54] In addition, the ideal BM should be absorbable but last long enough for predictable bone formation, decrease tissue movement and, when necessary, increase the tissue thickness over the bone graft. *Resorption* of a material describes the degradation of native tissues (e.g., bone). *Absorption* is the physiologic ability of tissues to degrade or reduce a foreign substance in the body. The absorption of the BM may be through a systematic activity called *biodegradation* or *bioabsorption* by hydroxylation from the surrounding soft tissues.

Although many BM fulfill most of these criteria, no single membrane is completely ideal. However, predictable results may be obtained when enough keys for bone grafting are present. The primary advantage of absorbable membranes is that a second-stage surgery is not necessary because it does not require removal. Although a secondary surgery is often used with GBR (unless the implant is placed at the same time), the

WWW.HIGHDENT.IR

همیار دندانسازان

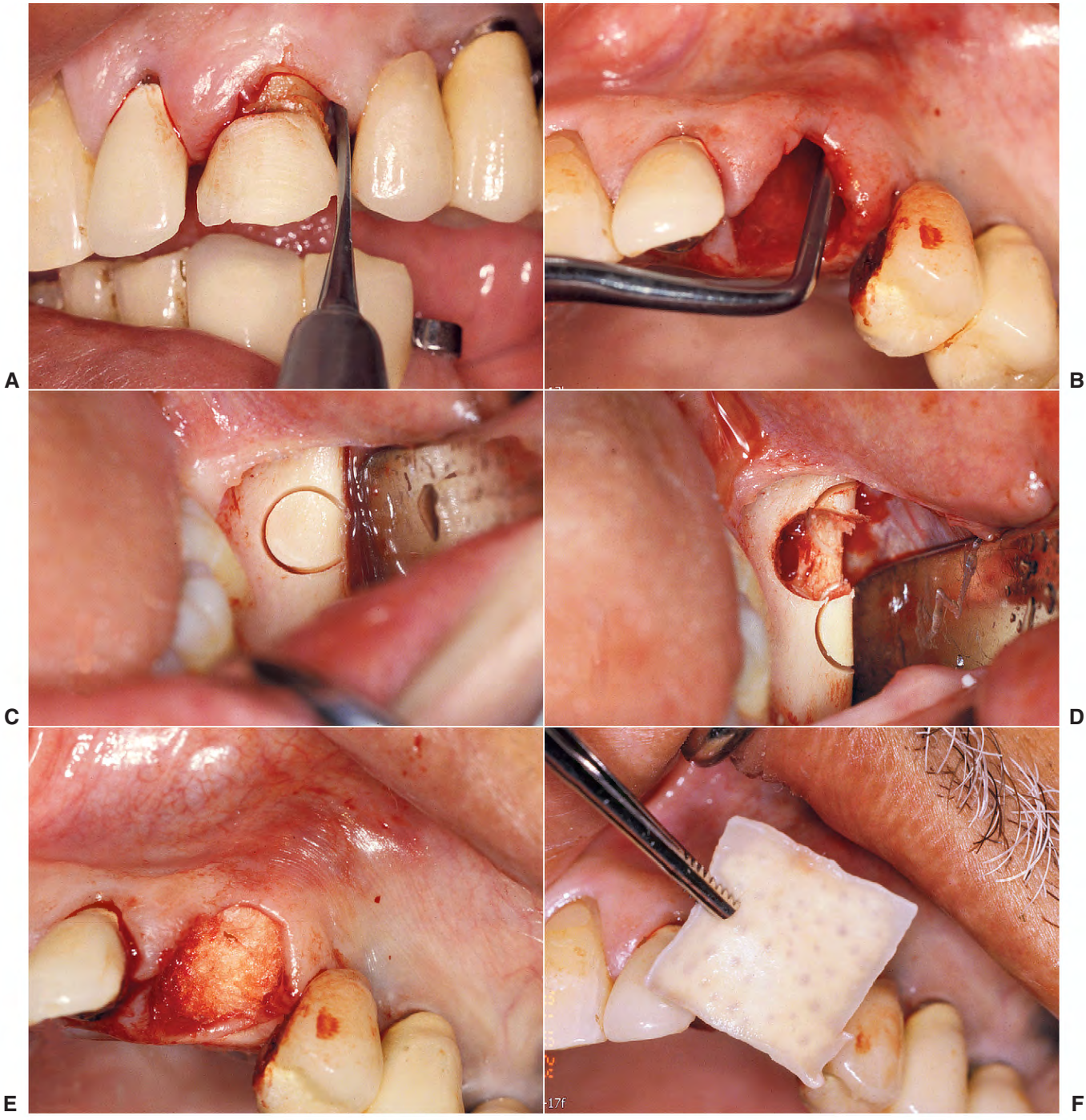


Figure 37-14 **A,** A canine tooth is atraumatically extracted. **B,** The extraction site has the facial wall missing, and the palatal and apical region has more bone loss than a typical extraction site. **C,** An ascending ramus is exposed, and a trephine bur is used along the lateral border. **D,** Overlapping trephine bur osteotomies are made so each bone segment is approximately 3 × 5 mm. **E,** The socket is filled with particulate autograft harvested from the ramus. **F,** The region above the bone is filled with demineralized freeze-dried bone cortical fibers. A barrier membrane (AlloDerm) is inserted along the facial aspect of the ridge; it extends above the crest and is tucked under the palatal tissue to cover the palatal plate. *Continued*

implant surgery may be flapless (when indicated) or the incision reflection of the tissue may be related solely to the indications for the implant not to remove the barrier membrane.[55-57] Absorbable membranes have fewer incision line opening complications, and when they do occur, they do not harbor bacteria, are treated more predictably, and regenerate more bone.[49-52] A second advantage is fewer incision line opening complications; if they do occur, management is more predictable and the amount of bone regenerated is greater than similar complications with nonresorbable materials.[40]

There are three primary categories of absorbable BM for GBR: collagen membranes, polylactic/polyglycolic acid membranes, and acellular dermal matrix. Collagen

WWW.HIGHDENT.IR

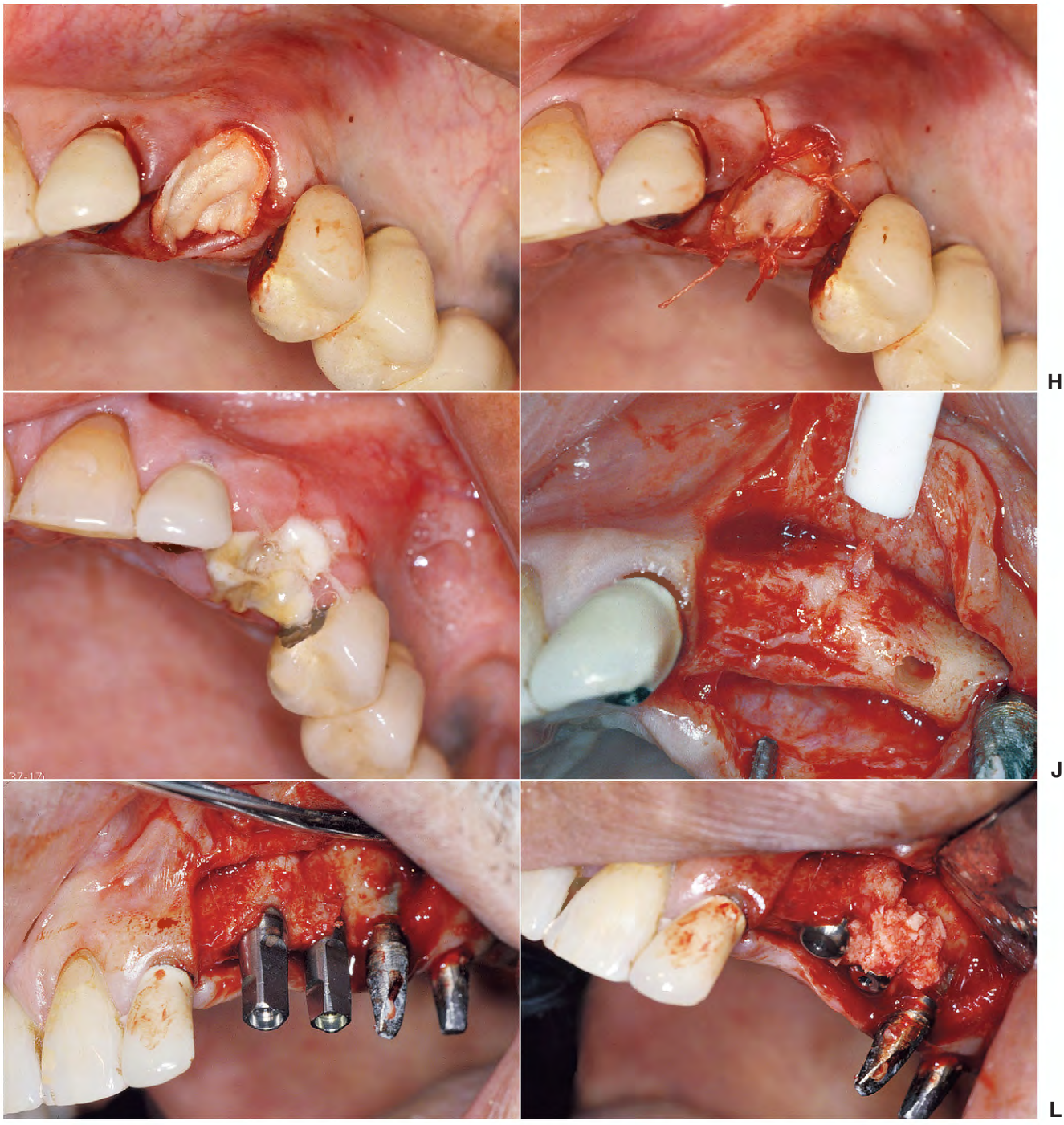


Figure 37-14, cont'd **G,** A barrier membrane of AlloDerm (acellular dermal matrix) is selected to cover the facial and palatal bony plate missing. **H,** The exposed membrane is sutured over the extraction site. **I,** Two weeks later the sutures are removed. The AlloDerm expands and, when exposed, appears soft and often has a taste or odor. One week later the site is improved. **J,** A reentry into the defect site demonstrates adequate bone volume in the canine site. The first premolar site (nongrafted) is deficient in width (Division). **K,** Endosteal implants are inserted into the canine and first premolar sites. Implants were previously positioned in the second premolar and molar sites, supporting a cantilevered restoration. **L,** Cover screws are inserted into the two implants placed at this time, and an autograft is placed over the first premolar implant site, as the facial bone is less than 1.5 mm thick.

membranes have several properties advantageous to GBR, including relative ease of use, hemostasis, and soft tissue compatibility, including the ability to act as a scaffold for migrating cells.[58-62] The most common sources are bovine type I collagen from the deep flexor tendon or dermal sites. After processing, it has minimal antigenicity with human tissue, as processing removes the antigenic portions of the polypeptide chain.

Not all absorbable collagen dressings may be considered a BM. In order for the material to act as a BM for GBR, the membrane should prevent unfavorable tissue cells from entering the bone augmentation site

WWW.HIGHDENT.IR

همیار دندانسازان

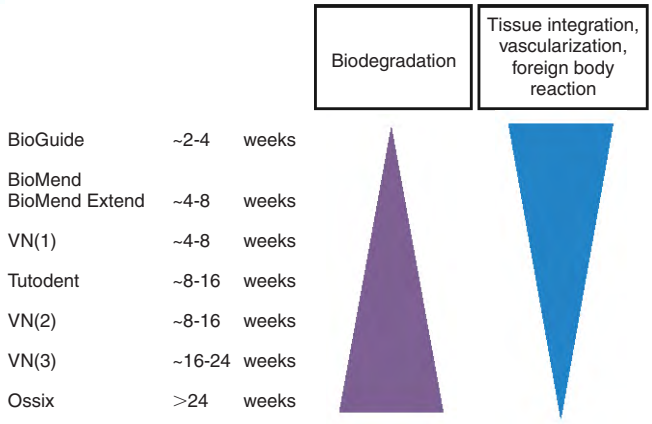


Figure 37-15 Different barrier membranes have different biodegradation times, which are inversely proportional to the tissue integration and vascularization schedule. For example, Biomend has an absorption rate of 4 to 8 weeks, whereas Ossix is more than 6 months. *(From Rothamel D, Schwarz F, Sager M et al: Biodegradation of differently cross-linked collagen membranes: an experimental study in the rat,* Clin Oral Implants Res *16:369-378, 2005.)*

for at least 6 to 8 weeks.[45,63] The greater the porosity of the collagen dressing (e.g., CollaTape, Zimmer Dental, Carlsbad, Calif.), the faster the absorption[64] (Figure 37-15). These materials provide a benefit for hemostasis, soft tissue healing, carry growth factors (e.g., PRP) to the graft site, and maintain the particulate material to a graft site (e.g., the maxillary sinus floor). However, it fully resorbs in 10 to 14 days. These features may help spark the regeneration process of both hard and soft tissue within this time frame, but it is not a BM for GBR. Likewise, PRP has been called a *quasi-membrane* when placed over a bone graft site. Because this material degranulates in 3 to 5 days, it is not a BM for GBR.

More dense and less porous collagen membranes may be fabricated in laminated sheets (e.g., Biomend, Zimmer Dental, Carlsbad, Calif.) and absorb over 4 to 8 weeks. BioGuide (Geistlich Biomaterials, Wohlhusen, Switzerland) is a bilayer collagen membrane from types I and III porcine collagen and absorbs over a 2- to 4-week period. Ossix (Colbar Life Science, Herzliya, Israel) is a porcine barrier membrane that may take more than 6 months to absorb. Clinical studies of GBR with different resorption rates, from 6 weeks to 6 months, often demonstrate similar bone augmentation results. It appears once the blood vessels from host bone invade the bone graft space, the other key factors are more relevant (e.g., graft immobilization).

Acellular dermal allograft (AlloDerm, LifeCell, Branchburg, N.Y.) is a processed human donor skin tissue that causes deepithelialization of the soft tissue and decellularization. Because there are no cells after the processing of the allograft tissue, rejection is eliminated. The acellular dermal matrix is undamaged and the collagen, elastin, and proteoglycans are still present, which augment the overlying soft tissue. As a result, an inert avascular connection tissue is obtained.[64] This tissue has been used in the medical community for treatment of third-degree burns, hernia repair, and tissue augmentation (including breasts).[65] In dentistry, it is often used to create zones of immobile tissue around teeth (or implants), root coverage,[66-70] soft tissue grafting, and augmentation.[71-77] This material also offers unique properties when used for GBR.[78,79]

Because the acellular allograft tissue material may become completely and permanently incorporated into the soft tissue after 6 weeks rather than resorbing as a collagen BM, it may increase tissue thickness over the graft site. This is beneficial in esthetic zones when the soft tissue drape needs to be developed. Because it binds to the overlying soft tissue, it may create a zone of immobile tissue. This is a benefit for particulate graft immobilization and for implant soft tissue maintenance after the prosthesis delivery.

In addition, a few clinical reports expose the acellular dermal matrix to the oral cavity during healing and allow it to act as a scaffold for the soft tissue to migrate over the material. This clinical factor may also be a benefit if incision line opening occurs during initial bone graft healing.[75,79-82]

The disadvantages of acellular dermal matrix include at least 10- to 20-minute preparation time. The material is placed into sterile saline for at least 10 minutes to rehydrate the freeze-dried tissue. After this initial period, another bowl of saline and an additional 10-minute period are suggested, to "wash" from the material the antibiotics that were used as part of the processing. In addition, the dermal matrix tissue swells in thickness and size during initial healing. This may cause incision lines to expose the material to the oral cavity.

SOFT TISSUE HEALING

One of the keys to bone grafting is initial soft tissue coverage and tissue healing. The understanding of the cytokine-mediated processes of connective tissue formation is the basis for the development of new innovative therapeutic strategies.[83] These may include the modulation of chemotaxis, differentiation, proliferation of fibroblasts, synthesis of extracellular matrix proteins, and, as a result, control of soft tissue formation.[83]

Both lack and overexpression of transforming growth factor-β (TGF-β) may be responsible for an impaired healing process.[83] TGF-β attracts fibroblasts, monocytes, and macrophages to the site of inflammation. Furthermore, it provokes a reduced phagocytosis capability of granulocytes and macrophages.[84] During new tissue formation, it induces the expression of integrins, which control the migration of keratinocytes to the wound surface.[85] Additionally, TGF-β stimulates the synthesis of collagen during the wound healing process.[86] In the course of remodeling, the cytokine is

WWW.HIGHDENT.IR

believed to influence composition and cross-linking of permanent collagen structures by regulating the pattern of integrin expression in fibroblasts.

Interleukin 1 (IL-1) fulfills a key role within the inflammatory signal transduction processes.[87] IL-1 is produced by monocytes and macrophages and serves as the main stimulus of extracellular matrix catabolism, production of collagenase and proteinase (matrix metal proteinase), and degranulation of neutrophil granulocytes.[88,89] As reported by Panagakos et al.,[90] increased levels of IL-1 are found during the loss of attachment within the scope of peri-implantitis. Implant-surrounding soft tissue altered by inflammatory processes expresses noticeably higher levels of IL-1β in comparison to the physiologic mucosa.[91] It can be used as a marker for a proinflammatory cellular response in the peri-implant soft tissue.[92]

Vascular endothelial growth factor (VEGF) is a glycoprotein that induces the microvascular permeability and angiogenesis during the stage of proliferation.[93] The cytokine is activated by hypoxic conditions and furthermore by IL-1β and TGF-β.[94,95] The VEGF gene is organized in eight exon subunits separated by seven intron sequences.[96] $VEGF_{165}$ is the prevalent molecular type synthesized and secreted by fibroblasts and keratinocytes.[83] Native $VEGF_{165}$ is a basic, heparin-binding, homodimeric glycoprotein.[97] Johnson et al. found significantly higher concentrations of VEGF in inflammatory gingival tissue compared with unaffected oral mucosa.[98] Suthin et al.[99] described bacterial liposaccharides to be responsible for the stimulation of VEGF secretion. Independent from this involvement in inflammatory processes, VEGF is excreted during every wound healing by keratinocytes.[100]

Whether the surgical implantation itself may cause a potential increase in the expression of inflammatory cytokines such as TGF-β_1, IL-1β, and VEGF in peri-implant soft tissue has not yet been investigated in a clinical follow-up.[83]

Peri-implant tissues become hemorrhagic and edematous in the course of healing disturbance or peri-implantitis.[83] There is little information concerning the biological mechanisms that may produce these alterations.[83] Existing clinical trials have investigated the expression of cytokines in the peri-implant region in inflammatory impaired soft tissues and have reported increased levels of IL-1β, TGF-β_1, and VEGF in the course of inflammation.[90,101-103] In vitro data show increased values of IL-1β, IL-6, and tumor necrosis factor alpha if tissue is incubated with dental implants.[104,105]

In an experimental setting, Shull et al.[106] described severe and uncontrollable inflammatory reactions in TGF-β–negative mice. The phagocytosis activity of neutrophil granulocytes and macrophages may be reduced.[83] High levels of TGF-β_1 can further result in impaired cross-linking of collagen structures and fibrotic transformation of tissue.[107] In experimental models the use of TGF-β–neutralizing antibodies results in a reduction of fibrotic processes during wound healing.[108] In contrast, higher levels of TGF-β_1 itself seem to have a positive influence on successful implant integration: the synthesis of extracellular matrix and the proliferation of fibroblasts is stimulated while the expression of adhesion molecules in mucosal fibroblasts is enhanced.[109] In vitro studies also used topical application of TGF-β for enhancement of healing and strength of granulation skin wounds.[110]

Lipopolysaccharide adsorption processes on the titanium surface of implants may contribute to this cytokine activation, especially because the use of different implant types results in variable expression levels, as reported by Perala et al.[104] Because elevated levels of IL-1 cause tissue destruction, the application of IL-1 antagonists (IL-1 receptor antagonists or monoclonal antibodies) results in a reduction of tissue damage and limited inflammatory processes during experimentally induced periodontitis.[111,112] It has further been shown that IL-1 antagonism results in a reduced progression of inflammatory processes and a reduction in loss of attachment and bone destruction.[113]

VEGF is a powerful mitogen for endothelial cells and fulfills an important role in angiogenesis.[114] The differences in terms of stromal cell positivity for VEGF between healthy and peri-implantitis sites have been studied, and higher values were found in peri-implantitis.[115] Other studies proved VEGF to be an important factor in the initiation and progression of gingivitis to periodontitis, promoting expansion of the vascular network that coincides with progression of the inflammation.[98,116] The demonstration of high expression levels may point to the induction of neoangiogenesis during the periimplant healing processes.[98,115] This capability is also underlined by a study in which intramuscular application of recombinant VEGF improved perfusion and formation of vascular structures.[117]

TGF-β is composed of a family of multifunctional polypeptide growth factors involved in embryogenesis, inflammation, regulation of immune response, angiogenesis, wound healing, and extracellular matrix formation. TGF-β_1 is the most common isoform found in human tissues. A role for TGF-β in the pathogenesis of periodontal disease has been suggested.[101]

TGF-β_1 may be one of the most important factors in the regulation of the infiltrate and in the production of tissue repair with a stimulation of fibroblasts and endothelial cells.[101]

The long-term clinical and esthetic success of implant-supported restorations is determined by osseointegration and optimal remodeling of peri-implant soft tissues. Complications of soft tissue management are often caused by fibrotic regeneration of oral mucosa after multiple surgical procedures.[82] Knowledge of the proliferative processes in wound healing is necessary to attain adequate soft tissue conditions. Successful reconstruction of peri-implant soft tissues is feasible, even in fibrotic conditions when appropriate surgical techniques are selected. The pleiotropic proliferative

WWW.HIGHDENT.IR

همیار دندانسازان

cytokine TGF-β is involved in the regulation of all phases of wound healing and tissue remodeling.[83]

The isoform TGF-β_1 is a cytokine associated with the development of fibrotic tissue. Overexpression of TGF-β_1 causes scarring and fibrosis and results in limited clinical success of intraoral soft tissue management.[118] Experimental therapeutic approaches with neutralizing antibodies to block TGF-β_1 resulted in less scarring and a reduction of fibrosis.[118] Further molecular biological research of cell-matrix-cytokine interactions in wound healing will provide highly specific antifibrotic therapeutic approaches in the future.[118]

The extended elevation in the cytokine expression may be linked to the surgical process or to the existence of an implant-mucosa interface.[83] Clinical implications of these prolonged tissue remodeling processes may be the development of protocols with extended interim solutions to await the completion of soft tissue remodeling before permanent loading.[83]

Inflammatory infiltrate may be important in the evolution of inflammatory processes involving peri-implant tissues. Angiogenesis is an important feature of inflammation and healing, but its role in the development and progression or in the healing of periodontal lesions has not been elucidated. VEGF is a potent inducer of endothelial cell proliferation.[113]

Because of its extensive presence, VEGF is probably a factor in both the maintenance of periodontal physiology and in the progression of peri-implant inflammatory disease.[113]

Recombinant human platelet-derived growth factor (rhPDGF-BB) is a bioactive protein that has been combined with bone graft materials in an effort to regenerate the periodontium to its prediseased state more predictably. The combination of a biocompatible scaffold with this highly purified, recombinant human growth factor capitalizes on the stimulatory effects of PDGF on cellular migration and proliferation, with subsequent matrix formation, to drive the healing process toward regeneration.[119]

When using a growth factor such as rhPDGF-BB with broad wound healing activity, an added clinical benefit is the more rapid soft tissue wound healing pattern observed at the surgical site. This also may be seen as a patient-centered benefit because it allows dentists to provide for a patient's prosthetic care on a timely basis, leading to a faster return to normal function. Also, the patient may resume plaque control efforts earlier, which further facilitates the wound healing process.[119]

Barrier Membrane and Guided Bone Regeneration: The Layered Approach

Defect Size and Topography

The most predictable topography for a barrier membrane bone graft is for (1) an edentulous site that is 0.5 to 3.5 mm inadequate in width, or a Division B bone volume; (2) an extraction socket missing one wall of bone; (3) an implant insertion missing one wall of bone, or an implant repair at stage II uncovery missing one wall of bone. The size of bone defect is most predictable when a four or fewer tooth span is present. Edentulous spans of more than a quadrant become less predictable for BM augmentation. Therefore the more walls of bone missing, or the more width or length of bone desired, the less predictable the BM bone graft.

The clinical procedure for BM GBR should attempt to provide as many keys for bone augmentation as practical. The keys include: (1) absence of infection; (2) soft tissue closure; (3) space maintenance; (4) graft immobilization; (5) RAP; (6) host bone vascularization; (7) growth factors; (8) BMPs; (9) healing time; (10) defect size and topography; and (11) transitional prosthesis. The general principles for these keys are addressed in Chapter 36. The specifics of these factors for BM and GBR will be addressed in this section.

The layered approach to GBR was developed by Misch in the early 1990s.[12,120] This concept provides as many keys of bone grafting as possible to the GBR process. The "layers" in the GBR process include the following:

1. *The host bone.* The host bone provides a wall(s) of surrounding bone. Because it is also decorticated by holes through the cortical plate, the RAP of regeneration is increased and provides host bone blood vessels to the bone graft site. This action carries bone induction growth factors to the graft.
2. *An autograft.* An autograft placed directly on bone allows blood vessels to reach the bone graft to maintain viability for osteogenesis. The autograft also provides FGF to accelerate blood vessel growth from the host bone into the graft. It also has BMPs and other cell enhancement factors to grow bone by osteoinduction. The autograft acts as the spark to grow the bone more predictability and faster.
3. *A mixture of DFDB (30%), FDB (70%), and PRP.* The next layer for GBR provides growth factors (not as much as RAP and autograft) and space maintenance in the graft site. Bone is formed by osteoinduction from the DFDB and PRP and osteoconduction from the FDB (or mineralized bovine source) in this area.
4. *A BM and tent screw.* A BM prevents fibroblasts and connective tissue from invading the graft site for at least 6 weeks. By that time, the graft site is programmed to form bone. A tent screw decreases mobility of the particulate particles and helps ensure space maintenance for bone to form.
5. *Primary closure without tension.* Primary closure prevents contamination and loss of graft material.

Procedure

Host Site Preparation

The recipient site is first prepared for the BM GBR procedure. In the maxilla, the crestal incision is made

WWW.HIGHDENT.IR

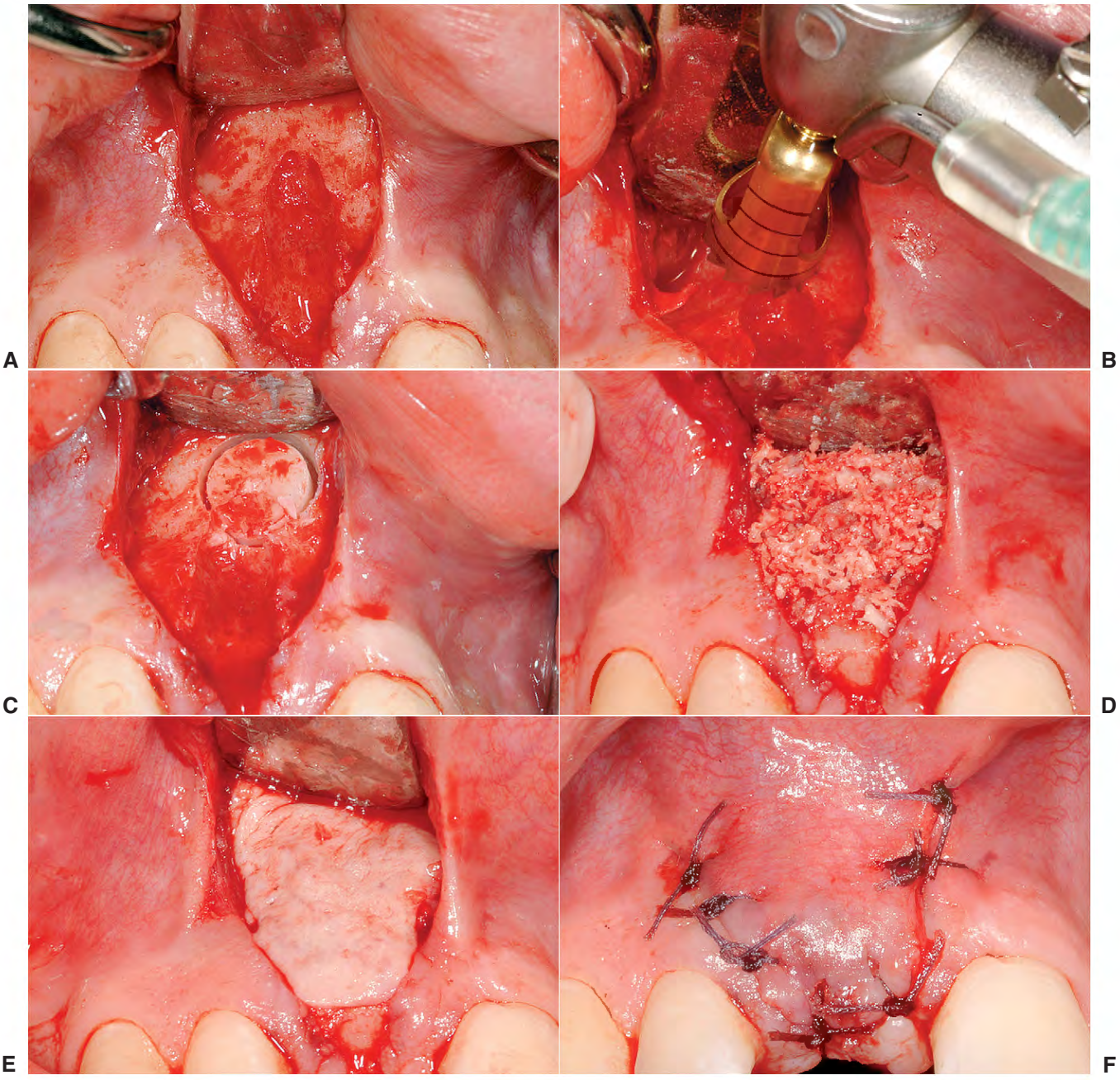


Figure 37-16 **A,** Papilla "saving" incisions are made to augment a central incisor region. **B,** A trephine bur is used to prepare a harvest site from above the apices of anterior teeth. **C,** The osteotomy of the donor site is observed. This bone core is removed and placed directly on the host bone. **D,** A mixture of 30% demineralized freeze-dried bone and 70% mineralized graft (MinerOss) is placed over the autograft. **E,** A barrier membrane (AlloDerm) is placed over the resorbable graft materials. The membrane is soaked in platelet-poor plasma. **F,** The soft tissue is approximated for primary closure.

slightly toward the palate or lingual, to place more keratinized tissue on the facial. Vertical release incisions are dependent upon the interdental papilla height next to the edentulous site. When the interdental papilla are ideal, a papilla-saving incision is made and the interdental papilla is not reflected (Figure 37-16). When the papilla is depressed in the height and contour, the usual vertical release incisions are one tooth distal to the graft site. When the interdental papilla has a probing depth of 6 mm or more, there is a risk the papilla will shrink after reflection and be more difficult to return to an ideal height. If the distal papilla has a probing depth less than 6 mm, the incision is distal to this structure and the papilla is reflected. If the probing depth of the distal papilla is greater than 6 mm and the papilla is in ideal position, it is not reflected and the vertical release incision is made mesial to the papilla. The vertical release incisions are made to the mucogingival junction (MGJ) (Figure 37-17).

A split-thickness reflection is made distal to the graft site and a full-thickness incision is made over the graft

WWW.HIGHDENT.IR

همیار دندانسازان

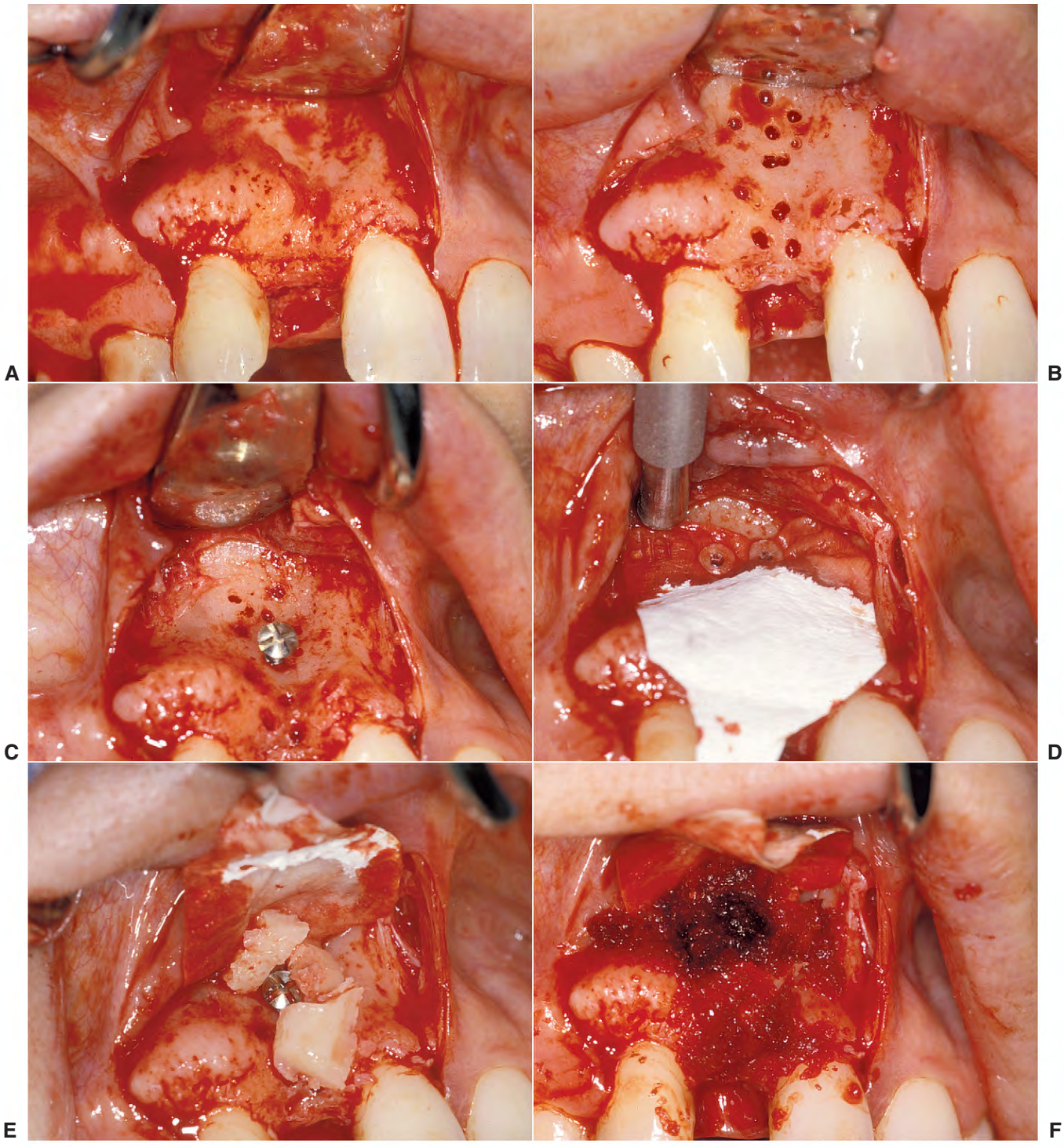


Figure 37-17 **A,** The crestal incision is made slightly toward the palatal slope, to increase the amount of keratinized tissue on the facial of the soft tissue flap. Vertical release incisions are made on the distal line angles of the tooth and the papillae are not reflected, as the augmentation site is limited in length and width. **B,** The host bone is decorticated holes made with a small-diameter bone drill, 3 to 5 mm apart. **C,** A tent screw is positioned in the host site. The screw head is elevated to the height of the desired bone augmentation. **D,** A collagen barrier membrane (Biomend) is fixated at the most coronal position, above the graft site. **E,** The barrier membrane (BM) is elevated and 3- to 5-mm pieces of autograft, collected from the ramus, are positioned directly on the host bone. **F,** A mixture of demineralized freeze-dried bone and mineralized autograft, mixed with platelet-rich plasma, is placed over the autograft.

Continued

site. The palatal/lingual tissue flap is full thickness in the area of the graft site for 5 mm from the crest. A full-thickness reflection of the mucoperiosteal flap is made on the facial in the area of the graft site and extended 5 mm beyond the apical extent of the graft site and at least 5 mm beyond the level of the MGJ.

A small bone drill for a 1.4-mm fixation screw is used in a 20:1 reduction hand piece with copious

WWW.HIGHDENT.IR

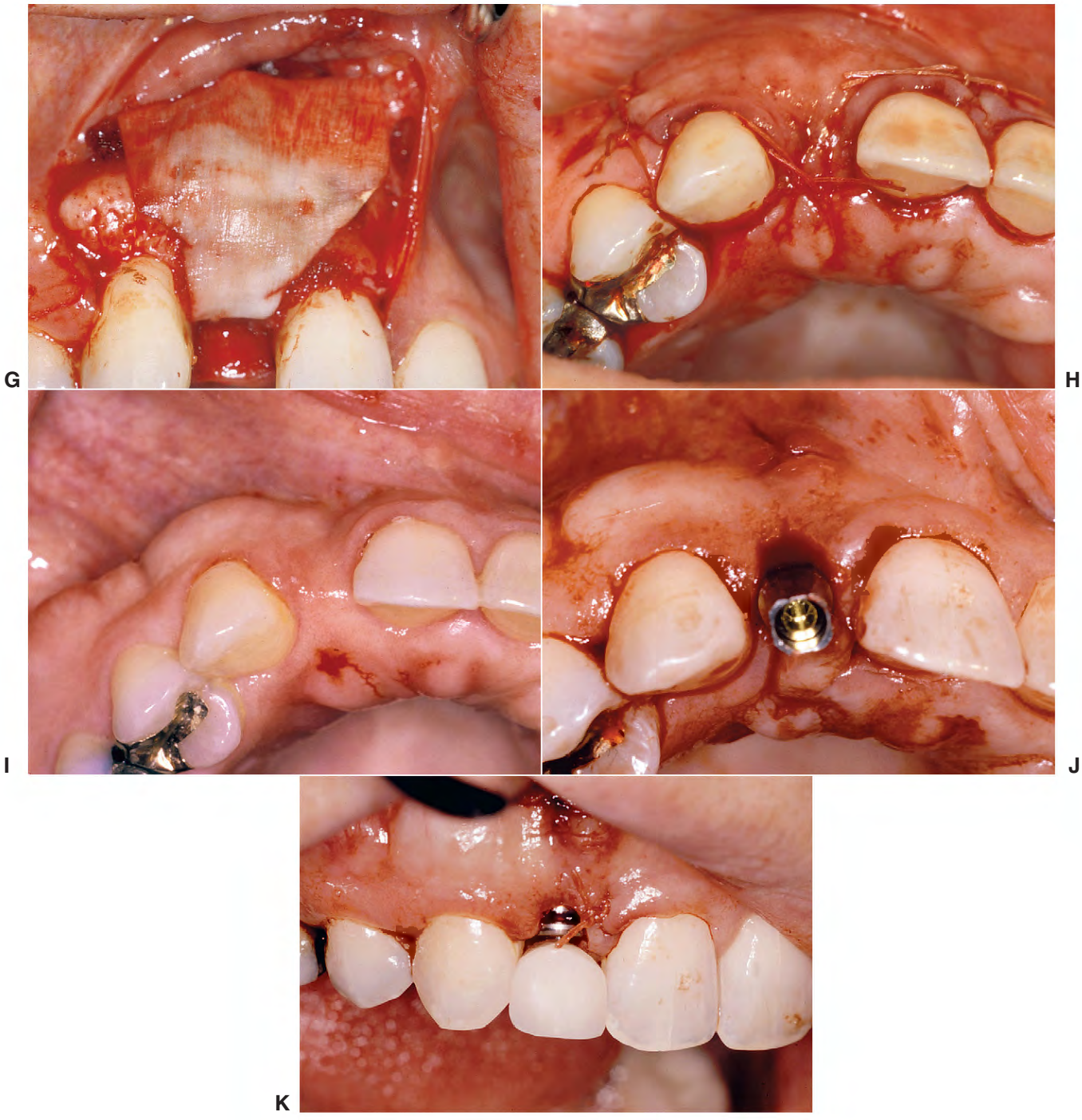


Figure 37-17, cont'd **G,** The BM, soaked in platelet-poor plasma, covers the graft site. **H,** The soft tissues are approximated with 4-0 PMA sutures for soft tissue closure, without tension. **I,** After 6 months, the soft tissue and host site are ready for reentry. **J,** An implant is inserted in the lateral incisor region, which was augmented by the layered approach of BM guided bone regeneration. **K,** The soft tissues are approximated around a permucosal extension for a one-stage surgical approach. The transitional prosthesis does not load the implant or soft tissue.

amounts of saline to prepare multiple holes in the facial cortical plate, 3 to 5 mm apart for the entire site of the graft. The holes should penetrate the lingual plate when bone augmentation is desired on both the facial and lingual aspects of the ridge. Ridge resorption primarily affects the facial plate dimension, but 20% to 30% of the ridge resorption is at the expense of the lingual plate dimension. Remember, for every 0.5 mm of bone grown on the lingual, 0.5 mm less bone needs to be grown in the facial for adequate bone width for an implant. Regardless, at least one to three holes are drilled through the lingual plate, as these sites will be used for tent screws.

A large pear-shaped drill is then used to contour the facial plate and ensure any fibrous tissue attached to the bone in the graft site is removed, including any

WWW.HIGHDENT.IR

همیار دندانسازان

remaining periosteum. The margins of the graft site are especially prepared, to ensure blood vessels from the host trabecular bone can enter the graft site. This prepared host bone surface is the bottom layer of the graft site.

The facial flap is held with a tissue pick-up at the height of the MGJ. The flap is elevated 3 to 5 mm above the height of the MGJ (on the tissue side), and the periosteum is incised with a scalpel 1 to 2 mm deep. The incision is parallel to the crestal incision and extends above and beyond the vertical release incisions.

A soft tissue scissors (e.g., Metzenbaum) with the blades closed is pushed through the periosteal incision and parallel to the surface mucosa for approximately 10 to 15 mm. The blades of the scissors are then opened to form a split-thickness submucosa space to be developed. This procedure allows the facial flap to be advanced over the bone graft for primary closure without tension.

One to three fixation screws are then inserted into the host site and extend above the bone for the desired dimension of the graft after healing. If the desire is to gain 3 mm in width, the screw is elevated 3 mm above the host bone. The tent screw has three primary functions. First it gives a visual indication of how much autograft should be harvested. Second, it helps maintain the space under the barrier membrane during bone formation. Third, it decreases movement of the particulate graft under the transitional prosthesis.

Autologous Donor Site Preparation

The autograft donor site is then addressed. This graft harvest is usually with a trephine bur and, when possible, is near the recipient site. The donor site may be above the graft site in the maxilla or below the site in the mandible. Other sites include the maxillary tuberosity, the ascending ramus, exostoses or tori, debris from the implant osteotomies, the tibia, the mandibular symphysis, palate, and zygomatic process. (In other words, anywhere the surgeon and patient feel comfortable.) Ideally, the pieces of bone harvested are 3 × 5 × 5 mm in size and the amount of bone is enough to cover the bone graft host site to the height of the tent screw.

LAYER GRAFT POSITIONS

The pieces of donor bone are placed directly on top of the host bone site, fill the desired shape and height/width of the desired augmentation graft site and represent the bottom layer of the bone graft. This autograft position is best suited for osteogenesis, osteoinduction, and growth factor for bone and blood vessels to bone.

A mixture of 30% DFDB fibers (Grafton) and 70% FDB (MinerOss) or mineralized HA source (e.g., BioOss, Osteograf N-300) is mixed with PRP harvested from 10 to 20 mL of blood that is collected at the start of the procedure. These materials are all mixed together, inserted into an open bone 3-mL syringe, and transferred to the mouth. The material is placed over the autograft and serves as the third layer of the bone graft.

A BM (collagen [Biomend, Ossix] or acellular dermal matrix [AlloDerm]) is soaked in PRP (when enough is present) or platelet-poor plasma (PPP) and covers the previous layer of the graft site. This is the next layer of the bone graft. The barrier membrane may be sutured under the palatal/lingual flap before placing the autograft and the graft mixture. Once the BM is rotated over the graft material, it may be fixed above the graft site to the facial plate by fixation tacks. The membrane is compressed with a moist surgical sponge to ensure close adaptation of the graft to the host bone. The membrane should not be placed under the vertical release incisions. Instead, the reflected soft tissues should be over the split-thickness tissue and bone. The membrane should also be 2 to 3 mm from the sulcus of a tooth.

The tissues are approximated over the BM for primary closure without tension. If tension on the incision line is observed, a deeper submucosal space is created with blunt dissection using the tissue scissors. The vertical release incisions are first approximated to ensure correct position of the soft tissue around the teeth. A horizontal mattress suture is often placed in the middle of the graft site to overt the margins of the incision and ensure closure without tension. A continuous nonlocking suture usually finalizes the primary closure on the crest. The donor site is then filled with resorbable collagen (CollaTape) and the tissue approximated (Figure 37-18).

Gentle pressure is applied to the graft site for 3 to 5 minutes; any blood below the tissue is milked through the incision line and the incision inspected to ensure no particulate graft or BM material is present.

Reentry into a BM GBR is most often after 6 to 9 months, dependent upon the size of the defect and the amount of autograft (Figures 37-19, 37-20). When incision line opening has occurred during the first month, a longer healing time is required to compensate for the compromised conditions on the crestal portion of the graft site.

Implant Insertion and Guided Bone Regeneration

Guided bone regeneration has been successfully reported at the time of implant insertion in both animal and human trials.[29,121-124] In most of these reports, the threads of the implant were exposed on only one side.[29,121] The procedure is less at risk when the GBR is for width only, not for both width and height. The technique is very similar to that already presented. The osteotomy of the implant site is prepared to the opposing landmark (when

WWW.HIGHDENT.IR

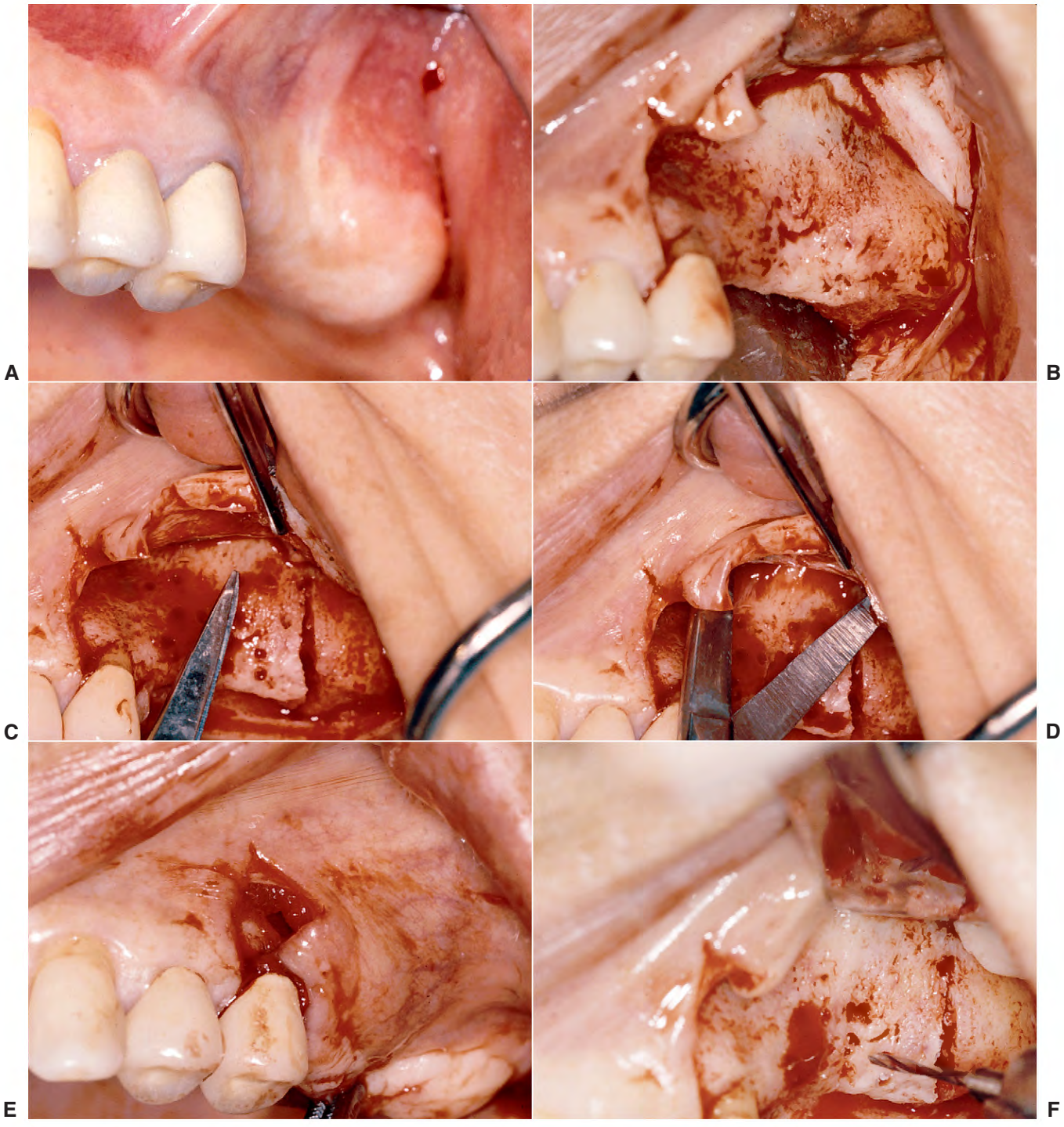


Figure 37-18 **A,** A partially edentulous posterior maxilla with a division B bone defect (one bony wall defect) in the molar site. **B,** A crestal incision is made along the palatal slope and a vertical release incision is made mesial to the adjacent tooth, leaving the interproximal papilla. **C,** A tissue scissors is positioned 3 to 5 mm above the height of the mucogingival junction and pushed into the facial flap, parallel to the surface mucosa. **D,** The tissue scissors are opened and create a submucosal tunnel above the periosteum. **E,** The release facial flap may be advanced over the augmented graft site without tension in the incision line. **F,** The host bone site is decorticated with holes 3 to 5 mm apart created by a small-diameter bone drill.

Continued

possible), and the bone debris is harvested from the drills. The implant length does not need to be as deep as the osteotomy, but the extra depth allows more bone to be harvested.

Holes in the cortical bone distal to the implant (not directly over the intact lateral bone), tent screws, autograft, the second layer of DFDB (30%), FDB (70%), PRP, and the top of the graft are covered with a BM with primary closure of the soft tissue.

The most difficult part of GBR at the time of implant insertion is to ensure that the implant is positioned for the prosthesis without compromise, rather than positioned

WWW.HIGHDENT.IR

همیار دندانسازان

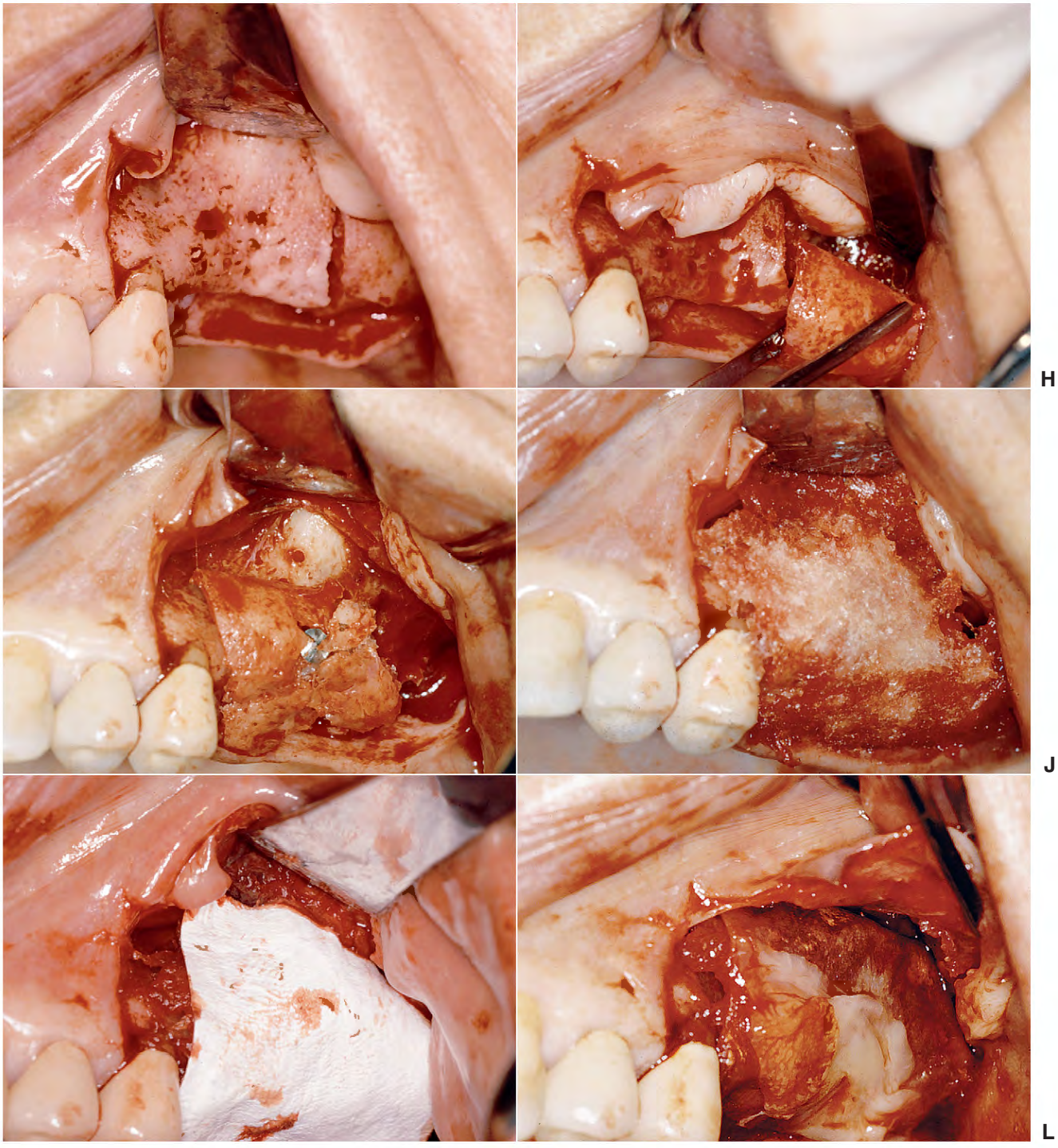


Figure 37-18, cont'd **G,** A large pear-shaped drill contours the facial bone to increase the regional acceleratory phenomenon and eliminate any remaining soft tissue on the surface. **H,** The tuberosity is harvested with chisels and a mallet. **I,** A tent screw is inserted into the facial plate and extends out from the bone the desired distance of augmentation. Autograft from the tuberosity is placed directly on host bone to the level of the tent screw. **J,** A 30% demineralized freeze-dried bone and 70% mineralized bone mixed with platelet-rich plasma (PRP) is placed over the autograft. **K,** A collagen barrier membrane (BM) (Biomend, Zimmer) is placed over the layered graft material. **L,** PRP is placed on the BM. *Continued*

more palatal (lingual) or angled to engage more host bone. The position of the implant should not be compromised so as to improve the success of the bone graft or to improve the osteointegration rate. The implants are useful for the prosthesis, not the bone graft. When the implant cannot be inserted in the correct position (in all three planes) because of the inadequate host bone, only the bone graft should be performed. Only after graft maturity can the implant be inserted.

It should be noted in the anterior regions of the mouth, the GBR procedure (with acellular dermal matrix) is used over the labial plate whenever it is less than 1.5 mm thick. This reduces the risk of marginal bone loss on the facial, which would result in shrinkage

WWW.HIGHDENT.IR

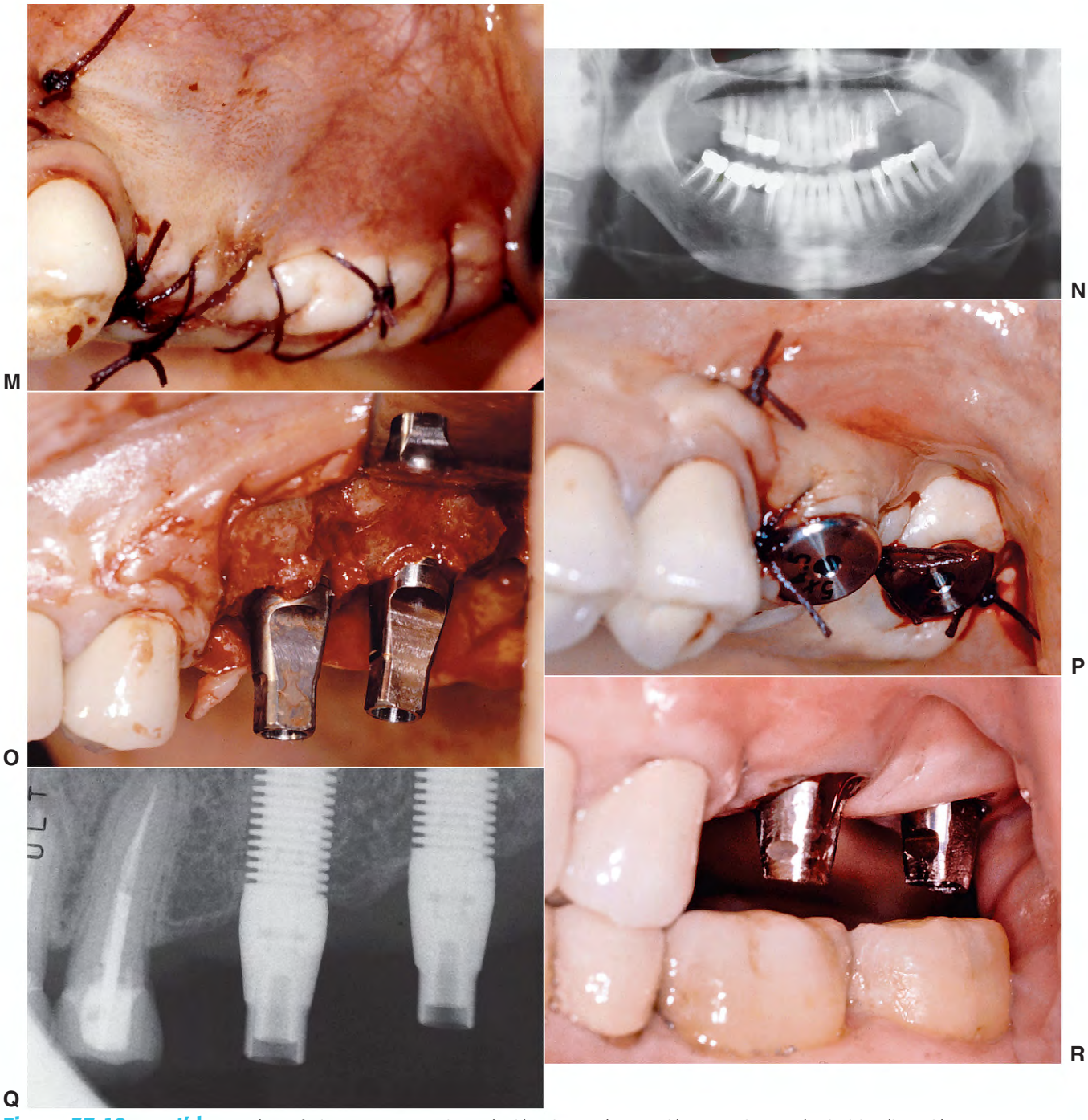


Figure 37-18, cont'd **M,** The soft tissues are approximated with primary closure without tension on the incision line with 3-0 PMA sutures. **N,** A postoperative panoramic radiograph of the maxillary posterior segment with tent screw and layered bone graft. **O,** After 7 months the reentry into the posterior maxilla permits the insertion of two 5-mm-diameter implants (Biohorizons) into the augmented site. **P,** Permucosal extensions and a one-stage surgical approach are used in the region. The tissues are sutured around the healing abutment for primary closure. **Q,** After 5 months, abutments are inserted into the implant and a periapical radiograph confirms proper seating and the maintenance of bone levels. **R,** The abutments are prepared to restore the maxillary first and second molars.

or recessing of the soft tissue at the cervical of the implant crown (Figure 37-21).

Tooth Extraction and Immediate Implant Insertion

The GBR procedure may be used when an implant is immediately placed after a tooth extraction when conditions permit the implant to be positioned without compromise to the prosthesis.[125-127] The osteotomy for the implant is made to the opposing landmark. The autograft is positioned over the implant and completely fills the extraction defect. The second layer of graft material is placed over the facial thin or missing bone. The barrier membrane is placed over the missing (or thin) bony wall, usually the facial. When a facial

WWW.HIGHDENT.IR

همیار دندانسازان

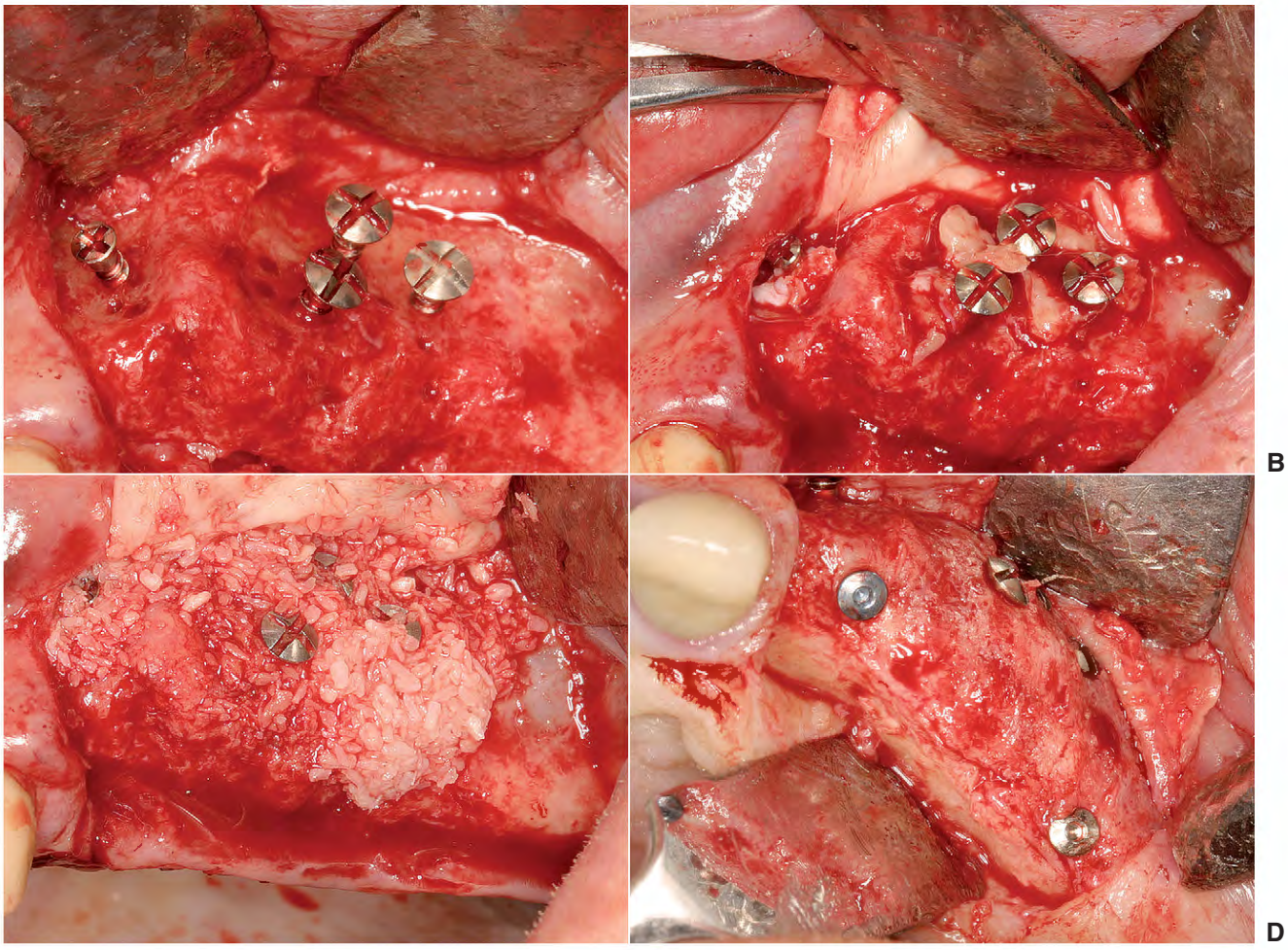


Figure 37-19 **A,** Four tent screws are inserted into an irregular-shaped Division B one bony wall defect. **B,** Autograft is placed on the host bone after decorticating the surface with holes. **C,** 35% demineralized freeze-dried bone cortical fibers (Grafton) and 70% freeze-dried bone (MinerOss) and platelet-rich plasma is placed over the autograft for the second layer. **D,** A 7-month reentry demonstrates the bone augmentation from the layered approach of barrier membrane guided bone regeneration. *(**A,** Courtesy Steve Caldwell.)*

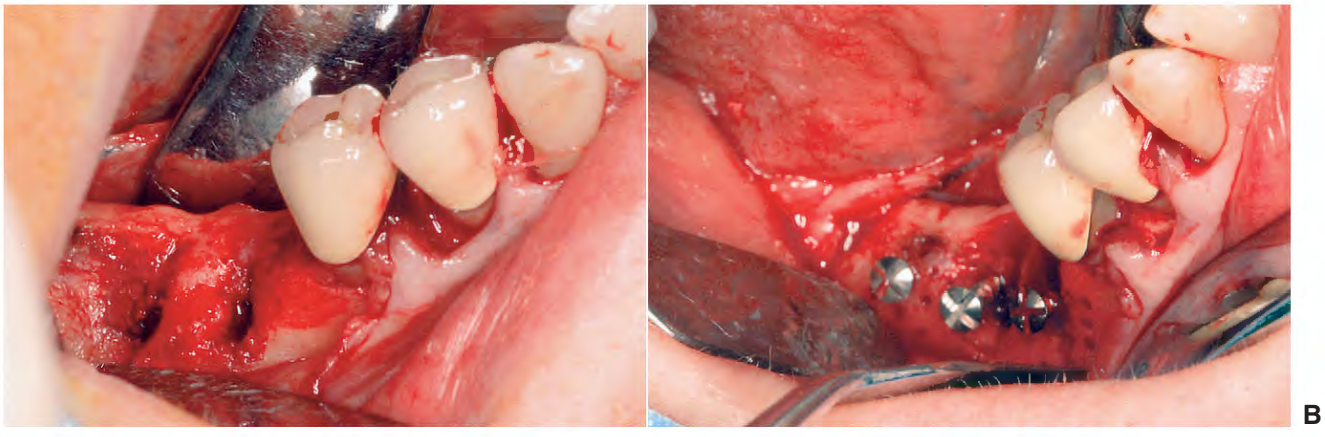


Figure 37-20 **A,** A molar extraction and facial bone dehiscence. **B,** A posterior mandible with a division B one bony wall defect. Three tent screws are positioned for the ideal bone width gain.

Continued

WWW.HIGHDENT.IR

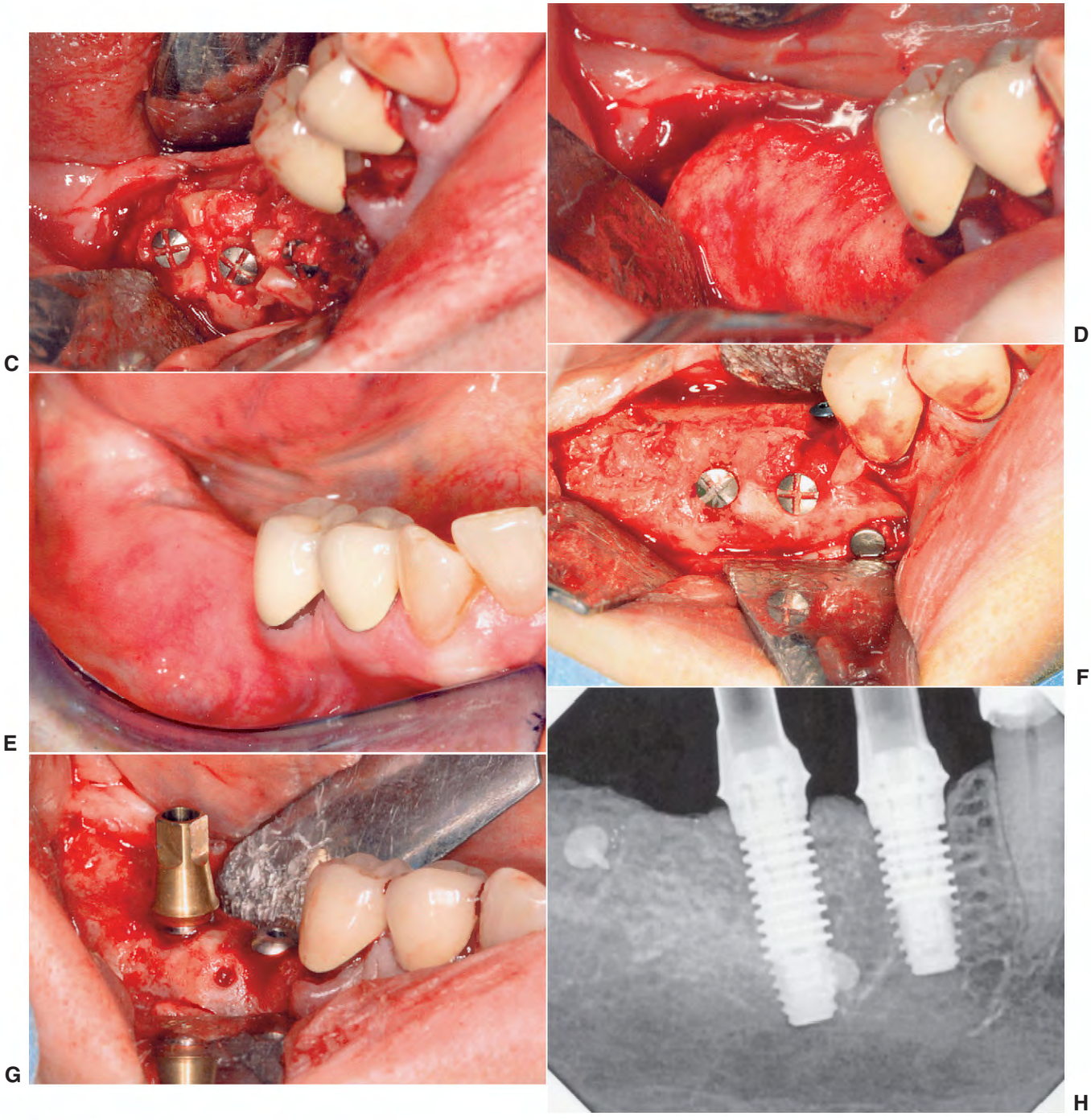


Figure 37-20, cont'd **C,** Particulate autograft is placed on the host bone after decorticating with holes created by a small-diameter drill. **D,** A barrier membrane soaked in platelet-poor plasma is placed over the graft site. **E,** The soft tissue healing is uneventful. **F,** A reentry at 7 months observes the augmented posterior mandible. **G,** Two endosteal implants are inserted into the molar sites (BioHorizons). **H,** A postoperative radiograph of two endosteal implants in the layered bone augmentation site.

plate of the bone is missing, primary closure of the soft tissue is preferred (Figure 37-22).

Transitional Prosthesis

The transitional prosthesis over a particulate graft should not rest on the soft tissue over the graft site. The tent screws provide some protection to the graft site; in addition, an FTP is more predictable for the process. Particulate grafts are more prone to movement during healing, which prevents blood vessels from entering and forming bone in the site.

Healing Time

The healing time of a layered particular BM GBR process is variable and may be from as little as 3 to 4 months to as long as 10 months, depending upon the size of

WWW.HIGHDENT.IR

همیار دندانسازان

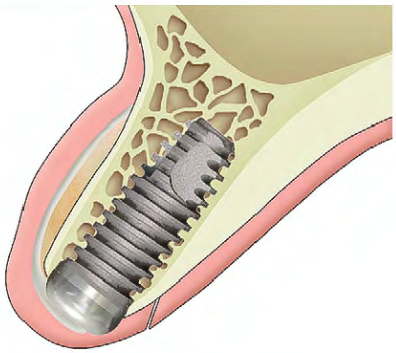


Figure 37-21 An implant may be inserted in the correct position, but the labial plate is less than 1.5 mm thick. A graft and a barrier membrane on the facial of an implant are indicated when the facial plate is thin and bone loss increases the risk of an esthetic compromise.

the augmentation, the number of walls of host bone, and the amount of autograft used in the graft site. As a general rule, it is better to wait 2 months too long than 2 days too little. Many graft sites that are entered too soon give the appearance the technique did not succeed. In reality, the immature bone and cartilage that initially form may be mixed with the bone graft materials, and the creeping substitution process of osteoconduction may be incomplete. Therefore waiting more than 6 months and less than 10 months is the general rule.

The period for graft maturity begins after the soft tissues have healed over the graft site. If incision bone opening occurred during the few weeks of initial healing,

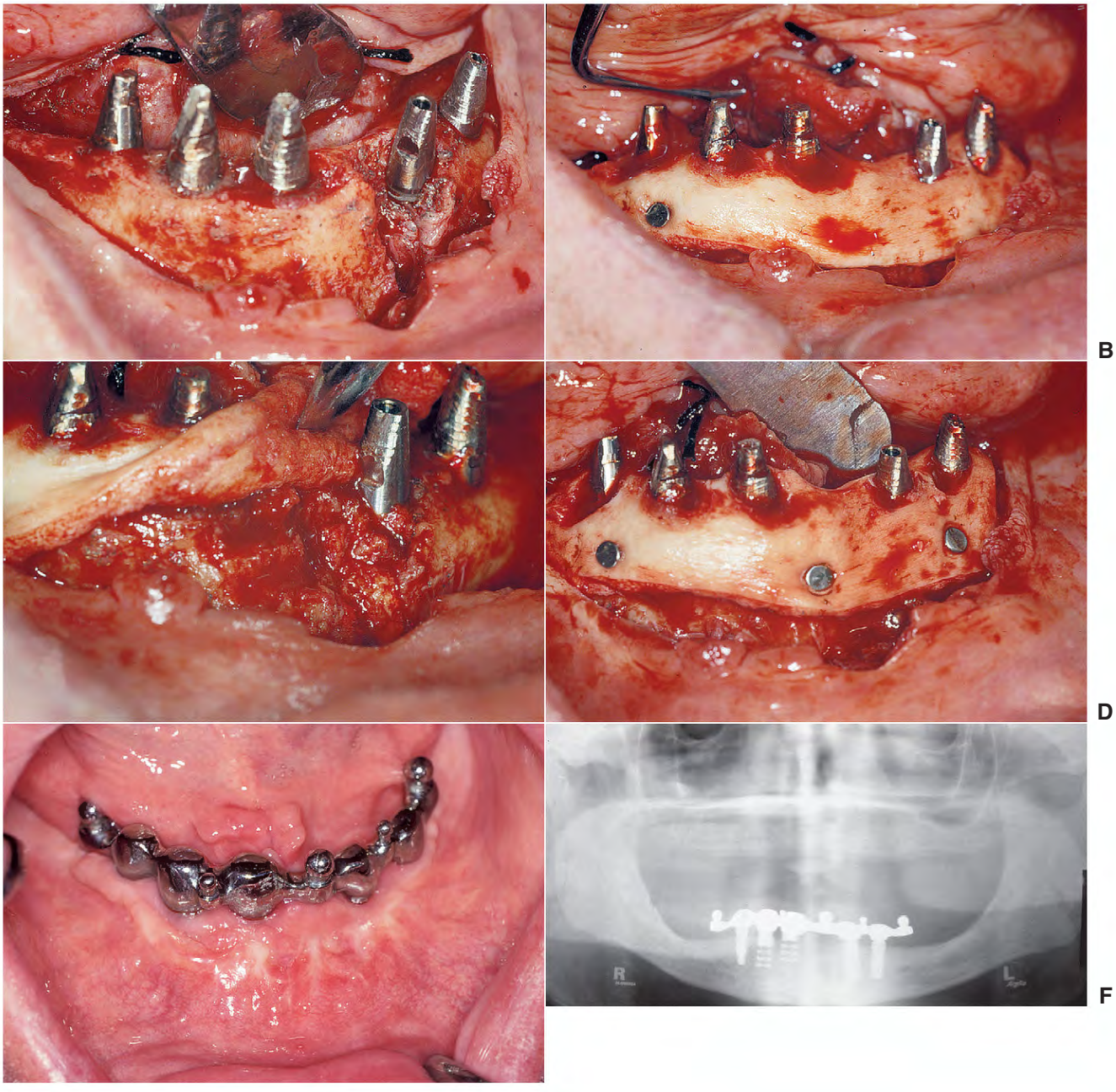


Figure 37-22 **A,** Five implants A, B, C, D, and E are inserted into an anterior mandible, and a large defect is present on the mesial of the D implant. **B,** An acellular dermal matrix barrier membrane (AlloDerm) is placed around the implants. **C,** Autograft is placed into the defect. **D,** The AlloDerm covers the autograft. **E,** Soft tissue healing is uneventful. **F,** A panoramic radiograph of the integrated implants and overdenture bar.

WWW.HIGHDENT.IR

the period of the graft should not begin until the soft tissues are healed over the graft site.

SUMMARY

The use of BMs in implant dentistry is common in conjunction with bone augmentation, implant insertion after extraction, and extraction sites with ridge preservation. A layered approach to BM and GBR improves the success rates and decreases the healing time. Trephine bur particulate bone harvesting from the tuberosity and ramus is often used for this technique. The learning curve of these procedure are easier than block bone grafting and the complications are fewer. As a result, nearly all implant surgeons should be able to use BM as a regular part of a surgical implant treatment protocol.

References

1. Roberts WE, Turley PK, Brezniak N et al: Bone physiology and metabolism, *Calif Dent Assoc J* 15:54-61, 1987.
2. Ruiz ME: *Dentistry: an illustrated history,* St Louis, 1985, Mosby.
3. Fauchard P: *Le chirurgien dentiste, oú traité des dents,* Paris, 1728, PJ Mariette.
4. Chauliac G: *De la grande chirurgie,* Rouen, France, 1649, David du Petit val.
5. Glenner RA, Davis AB, Burns SB: *The American dentist,* Missoula, Mont, 1990, Pictorial Histories.
6. Reilly DT, Burstein AH: The elastic and ultimate properties of compact bone tissue, *J Biomech* 8:393, 1975.
7. Ohta Y: Comparative changes in microvasculature and bone during healing of implant and extraction sites, *J Oral Implant* 3:184-198, 1993.
8. Froum S, Orlowski W: Ridge preservation utilizing alloplast prior to implant placement— clinical and histological case reports, *Pract Periodont Aesthet Dent* 12:393-402, 2000.
9. Bartee BK: Extraction site reconstruction for alveolar ridge preservation, part 1: rationale and materials selection, *J Oral Implantol* 27:187-193, 2001.
10. Foroler EB, Breault LG, Rebitski G: Ridge preservation utilizing an acellular dermal allograft and demineralized freeze-dried bone allograft, part 1: a report of 2 cases, *J Periodontol* 71:1353-1359, 2000.
11. Wang HL, Kiyovobin K, Nerva R: Socket augmentation: rationale and technique, *Implant Dent* 13:286-296, 2004.
12. Misch CE, Dietsh F: Bone grafting materials, *Implant Dent* 2:158-167, 1993.
13. Becker W, Becker BE, Caffesse R: A comparison of demineralized freeze-dried bone and autologous bone to induce bone formation in human extraction sockets, *J Periodontol* 65:1128-1133, 1994.
14. LeKovic V, Keeney EB, Weinlaender M et al: A bone regeneration approach to alveolar ridge maintenance following tooth extraction: report of 10 cases, *J Periodontol* 68:563-570, 1997.
15. Artzi Z, Tal H, Dayan D: Porous bovine bone mineral in healing of human extraction sockets, part I: histomorphometric evaluations at 9 months, *J Periodont* 71: 1015-1023, 2000.
16. McCall RA, Rosenfeld AL: Influence of residual ridge resorption patterns on implant fixture placement and tooth position, *Int J Periodontics Restorative Dent* 11:8023, 1991.
17. Tatum H: Maxillary and sinus implant reconstruction, *Dent Clin North Am* 30:207-229, 1986.
18. Misch CE: Divisions of available bone. In Misch CE, editor: *Contemporary implant dentistry,* ed 2, St Louis, 1993, Mosby.
19. Misch CM: Comparison of intraoral donor sites for onlay grafting prior to implant placement, *Int J Oral Maxillofac Implants* 12:767-776, 1997.
20. ten Bruggenkate CM, Kraaijenhagen HA, van der Kwast WAM et al: Autogenous maxillary bone grafts in conjunction with placement of I.T.I. endosseous implants: a preliminary report, *Int J Oral Maxillofac Surg* 21:81-84, 1992.
21. Buser D, Dula K, Belser UC et al: Localized ridge augmentation using guided bone regeneration. II. Surgical procedure in the mandible, *Int J Periodontics Restorative Dent* 15:11-29, 1995.
22. Misch CE, Dietsh F, Misch CM: A modified socket seal surgery: a composite graft approach, *J Oral Implantol* 25: 244-250, 1999.
23. Tischler M, Misch CE: Extraction site bone grafting in general dentistry: review of application and principles, *Dent Today* 23:108-113, 2004.
24. Knize D: The influence of periosteum and calcitonin on onlay bone graft survival, *Plast Reconstr Surg* 53:190-199, 1974.
25. Zucman P, Mauer P, Berbesson C: The effects of autographs of bone and periosteum in recent diaphyseal fractures, *J Bone Joint Surg* 50B:409, 1968.
26. Maw RB, Loizeaux AD: Functional regeneration of facial nerves in dogs when filter tubes are used, *J Oral Surg* 29:848-852, 1971.
27. Mellonig JT, Triplett RG: Guided tissue regeneration and endosseous dental implants, *Int J Periodontics Restorative Dent* 13:109-119, 1993.
28. Lindhe A, Thoren C, Dahlin C et al: Creation of new bone by an osteopromotive membrane technique: an experimental study in rats, *J Oral Maxillofac Surg* 51:892-889, 1993.
29. Dahlin C, Sennerby L, Lekholm U et al: Generation of new bone around titanium implants using a membrane technique: an experimental study in rabbits, *Int J Oral Maxillofac Implants* 4:19-25, 1989.
30. Buser D, Ruskin J, Higginbottom F et al: Osseointegration of titanium implants in bone regenerated in membrane-protected defects. A histologic study in the canine mandible, *Int J Oral Maxillofac Implants* 10:666-681, 1995.
31. Dahlin C, Lindhe A, Gottlow J et al: Healing of bone defects by guided tissue regeneration, *Plast Reconstr Surg* 81:672-676, 1988.
32. Dahlin C, Gottlow J, Linde A et al: Healing of maxillary and mandibular bone defects using a membrane technique, *Scand Plast Reconstr Surg* 24:13-19, 1990.
33. Nyman S, Lang NP, Buser D et al: Bone regeneration adjacent to titanium dental implants using guided tissue regeneration: a report of 2 cases, *Int J Oral Maxillofac Implants* 5:9-14, 1990.
34. Boyne PJ: Regeneration of alveolar bone beneath cellulose acetate filter implants, *J Dent Res* 1964;43:827.
35. Boyne PJ: Restoration of osseous defects in maxillofacial casualties, *J Am Dent Assoc* 1969;78:767-776.
36. Gottlow J, Nyman S, Karring T et al: New attachment formation as the result of controlled tissue regeneration, *J Clin Periodontol* 11:494-503, 1984.

WWW.HIGHDENT.IR

همیار دندانسازان

37. Nyman S, Gottlow J, Lindhe J et al: New attachment formation by guided tissue regeneration, *J Periodontal Res* 22:252-254, 1987.
38. Dahlin C, Simion M, Nanmark U et al: Histological morphology of the e-PTFE/tissue interface in humans subjected to guided bone regeneration in conjunction with oral implant treatment, *Clin Oral Implants Res* 9:100-106, 1998.
39. Piattelli A, Scarano A, Paolantonio M: Bone formation inside the material interstices of e-PTFE membranes: a light microscopical and histochemical study in man, *Biomaterials* 17:1725-1731, 1996.
40. Bunyarateve P, Wang H-L: Collagen membranes: a review, J Periodontol 72:215-229, 2001.
41. Wang H-L, MacNeil RL: Guided tissue regeneration: absorbable barriers, *Dent Clin North Am* 42:505-522, 1998.
42. Chen Y-T, Want H-L, Lopatin DE et al: Bacterial adherence to guided tissue regeneration barrier membranes exposed to the oral environment, *J Periodontol* 98:172-179, 1997.
43. von Arx T, Broggini N, Jensen SS et al: Membrane durability and tissue response of different bioresorbable barrier membranes: a histologic study in the rabbit calvarium, *Int J Oral Maxillofac Implants* 20:43-53, 2005.
44. Verardi S, Simion S: Management of the exposure of e-PTFE membranes in guided bone regeneration, *Pract Proced Aesthet Dent* 19:111-117, 2007.
45. Simion M, Baldoni M, Rossi P et al: A comparative study of the effectiveness of e-PTFE membranes with and without early exposure during the healing period, *Int J Periodontics Restorative Dent* 14:167-180, 1994.
46. Lekholm U, Becker W, Dahlin C et al: The role of early versus late removal of GTAM membranes on bone formation at oral implants placed into immediate extraction sockets. An experimental study in dogs, *Clin Oral Implant Res* 4:121-129, 1993.
47. Iasella JM, Greenwell H, Miller RL et al: Ridge preservation with freeze-dried bone allograft and a collagen membrane compared to extraction alone for implant site development: a clinical and histologic study in humans, *J Periodontol* 74:990-999, 2003.
48. Wang H-L, Yuan K, Burgett F et al: Adherence of oral microorganisms to guided tissue membranes: an in vitro study, *J Periodontol* 65:211-218, 1994.
49. Wang H-L, Carroll WJ: Guided bone regeneration using bone grafts and collagen membranes, *Implant Dent* 32:504-515, 2001.
50. Zitzmann NU, Naef R, Scharer P: Resorbable versus nonresorbable membranes in combination with Bio-Oss for guided bone regeneration, *Int J Oral Maxillofac Implants* 12:844-852, 1997.
51. Strietzel FP: Risks and complications of membrane-guided bone regeneration. Retrospective analysis. *Mund Kiefer Gesichtschir* 5:28-32, 2001.
52. Oh T-J, Meraw SJ, Lee E-J et al: Comparative analysis of collagen membranes for the treatment of implant dehiscence defects, *J Clin Oral Implants Res* 14:80-90, 2003.
53. Fugazzotto PA: Maintenance of soft tissue closure following guided bone regeneration: technical considerations and report of 723 cases, *J Periodontol* 70: 1085-1097, 1999.
54. Okazaki K, Shimizu Y, Xu H et al: Blood-filled spaces with and without deproteinized bone grafts in guided bone regeneration: a histomorphometric study of the rabbit skull using non-resorbable membrane, *Clin Oral Implants Res* 16:236-243, 2005.
55. Takata T, Wang HL, Miyauchi M: Migration of osteoblastic cells on various guided bone regeneration membranes, *Clin Oral Implants Res* 12: 332-338, 2001.
56. Locci P, Calvitti M, Belcastro S et al: Phenotype expression of gingival fibroblasts cultured on membranes used in guided tissue regeneration, *J Periodontol* 68:857-863, 1997.
57. Wang HL, Miyauchi M, Takata T: Initial attachment of osteoblasts to various guided bone regeneration membranes: an in vitro study, *J Periodontal Res* 37:340-344, 2002.
58. Steinberg AD, LeBreton G, Willey R et al: Extravascular clot formation and platelet activation on variously treated root surfaces, *J Periodontol* 57:516-522, 1986.
59. Postlethwaite AE, Seyer JM, Kang AH: Chemotactic attraction of human fibroblasts to type I, II and III collagens and collagen-derived peptides, *Proc Natl Acad Sci U S A* 75:871-875, 1978.
60. Wang HL, O'Neal RB, Thomas CL et al: Evaluation of an absorbable collagen membrane in treating class II furcation defects, *J Periodontol* 65:1029-1036, 1994.
61. Chen CC, Wang HL, Smith F et al: Evaluation of a collagen membrane with and without bone grafts in treating periodontal intrabony defects, *J Periodontol* 66: 838-847, 1995.
62. Wang HL, O'Neal RB, MacNeil LM: Regenerative treatment of periodontal defects utilizing a bioresorbable collagen membrane, *Pract Periodontics Aesthet Dent* 7: 59-66, 1995.
63. Lim SC, Lee MJ, Yeo HH: Effects of various implant materials on regeneration of calvarial defects in rats, *Pathol Int* 50:594-602, 2000.
64. Rothamel D, Schwarz F, Sager M et al: Biodegradation of differently cross-linked collagen membranes: an experimental study in the rat, *Clin Oral Implants Res* 16:369-78, 2005.
65. Tsai CC, Lin SD, Lai CS et al: The use of composite acellular allodermis-ultrathin autograft on joint area in minor burn patients—one year follow up, *Kaohsiung J Med Sci* 15:651-658, 1999.
66. Henderson RD, Greenwell H, Drisko C et al: Predictable multiple site root coverage using an acellular dermal matrix allograft, *J Periodontol* 72:571-582, 2001.
67. Tal H, Moses O, Zohar R et al: Root coverage of advanced gingival recession: a comparative study between acellular dermal matrix allograft and subepithelial connective tissue grafts, *J Periodontol* 73:1405-1411, 2002.
68. Harris RJ: Clinical evaluation of 3 techniques to augment keratinized tissue without root coverage, *J Periodontol* 72:932-938, 2001.
69. Wei PC, Laurell L, Geivelis M et al: Acellular dermal matrix allografts to achieve increased attached gingival. Part 1. A clinical study, *J Periodontol* 71:1297-1305, 2000.
70. Wei PC, Laurell L, Lingen MW et al: Acellular dermal matrix allografts to achieve increased attached gingival. Part 2. A histological comparative study, *J Periodontol* 72:257-265, 2002.
71. Batista EL, Batista FC, Novaes AB Jr: Management of soft tissue ridge deformities with acellular dermal matrix. Clinical approach and outcome after 6 months of treatment, *J Periodontol* 72:265-273, 2001.
72. Fowler EB, Breault LG: Ridge augmentation with a folded acellular dermal matrix allograft: a case report, *J Contemp Dent Pract* 2:31-40, 2001.

WWW.HIGHDENT.IR

73. Harris RJ: Soft tissue ridge augmentation with acellular dermal matrix, *Int J Periodontics Restorative Dent* 23:87-92, 2003.
74. Fowler EB, Breault LG, Rebitski G: Ridge preservation utilizing an acellular dermal allograft and demineralized freeze-dried bone allograft: part 1. A report of 2 cases, *J Periodontol* 71:1353-1359, 2000.
75. Park J-B: Increasing the width of keratinized mucosa around endosseous implant using acellular dermal matrix allograft, *Implant Dent* 15:275-279, 2006.
76. Harris RJ: Gingival augmentation with an acellular dermal matrix: human histologic evaluation of a case-placement of the graft on periosteum, *Int J Periodontics Restorative Dent* 24:379-385, 2004.
77. Terrence JG, Cheung WS, Hiroshi H: Hard and soft tissue augmentation in implant therapy using acellular dermal matrix, *Int J Periodontics Restorative Dent* 24:353-361, 2004.
78. Novaes AB, Souza, SL: Acellular dermal matrix graft as a membrane for guided bone regeneration: a case report, *Implant Dent* 10:192-196, 2001.
79. Park SH, Wang H-L: Management of localized buccal dehiscence defect with allografts and acellular dermal matrix, *Int J Periodontics Restorative Dent* 26:589-595, 2006.
80. Novaes AB Jr, Papalexiou V, Luczyszyn SM et al: Immediate implant in extraction socket with acellular dermal matrix graft and bioactive glass: a case report, *Implant Dent* 11:343-348, 2002.
81. Cummings LC, Kaldahl WB, Allen EP: Histologic evaluation of autogenous connective tissue and acellular dermal matrix grafts in humans, *J Periodontol* 76:178-186, 2005.
82. Livesey SA, Herndon DN, Hollyoak MA et al: Transplanted acellular allograft dermal matrix. Potential as a template for the reconstruction of viable dermis, *Transplantation* 60:1-9, 1995.
83. Schultze-Mosgau S, Wehrhan F, Wichmann M et al: Expression of interleukin 1-beta, transforming growth factor beta-1, and vascular endothelial growth factor in soft tissue over the implant before uncovering, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 101:565-71, 2006.
84. Gabka CJ, Benhaim P, Mathes SJ et al: An experimental model to determine the effect of irradiated tissue on neutrophil function, *Plast Reconstr Surg* 96:1676-1688, 1995.
85. Gailit J, Welch MP, Clark RA: TGF-beta 1 stimulates expression of keratinocyte integrins during re-epithelialization of cutaneous wounds, *J Invest Dermatol* 103:221-227, 1994.
86. Kishi K, Nakajima H, Tajima S: Differential responses of collagen and glycosaminoglycan syntheses and cell proliferation to exogenous transforming growth factor beta 1 in the developing mouse skin fibroblasts in culture, *Br J Plast Surg* 52:579-582, 1999.
87. Goretsky MJ, Harriger MD, Supp AP et al: Expression of interleukin-1 alpha, interleukin-6, and basic fibroblast growth factor by cultured skin substitutes before and after grafting to full-thickness wounds in athymic mice, *J Trauma* 40:894-899, 1996.
88. Dinarello CA: Interleukin-1 and its biologically related cytokines, *Adv Immunol* 44:153-205, 1989.
89. Mizel SB, Dayer JM, Krane SM et al: Stimulation of rheumatoid synovial cell collagenase and prostaglandin production by partially purified lymphocyte-activating factor (interleukin 1), *Proc Natl Acad Sci U S A* 78: 2474-2477, 1981.
90. Panagakos FS, Aboyoussef H, Dondero R et al: Detection and measurement of inflammatory cytokines in implant crevicular fluid: a pilot study, *Int J Oral Maxillofac Implants* 11:794-799, 1996.
91. Hoenig J, Rordorf-Adam C, Siegmund C et al: Increased interleukin-1 beta (IL-1 beta) concentration in gingival tissue from periodontitis patients, *J Periodontal Res* 24: 362-367, 1989.
92. Black RA, Kronheim SR, Cantrell M et al: Generation of biologically active interleukin-1 beta by proteolytic cleavage of the inactive precursor, *J Biol Chem* 263: 9437-9442, 1988.
93. Konig JE, Tolnay E, Wiethege T et al: Expression of vascular endothelial growth factor in diffuse malignant pleural mesothelioma, *Virchows Arch* 435:8-12, 1999.
94. Li J, Perrella MA, Tsai JC et al: Induction of vascular endothelial growth factor gene expression by interleukin-1 beta in rat aortic smooth muscle cells, *J Biol Chem* 270:308-312, 1995.
95. Pertovaara L, Kaipainen A, Mustonen T et al: Vascular endothelial growth factor is induced in response to transforming growth factor-beta in fibroblastic and epithelial cells, *J Biol Chem* 269:6271-6274, 1994.
96. Tischer E, Mitchell R, Hartman T et al: The human gene for vascular endothelial growth factor. Multiple protein forms are encoded through alternative exon splicing, *J Biol Chem* 266:11947-11954, 1991.
97. Ferrara N, Houck K, Jakeman L et al: Molecular and biological properties of the vascular endothelial growth factor family of proteins, *Endocr Rev* 13:18-32, 1992.
98. Johnson RB, Serio FG, Dai X: Vascular endothelial growth factors and progression of periodontal diseases, *J Periodontol* 70:848-852, 1999.
99. Suthin K, Matsushita K, Machigashira M et al: Enhanced expression of vascular endothelial growth factor by periodontal pathogens in gingival fibroblasts, *J Periodontal Res* 38:90-96, 2003.
100. Shweiki D, Itin A, Neufeld G et al: Patterns of expression of vascular endothelial growth factor (VEGF) and VEGF receptors in mice suggest a role in hormonally regulated angiogenesis, *J Clin Invest* 91:2235-2243, 1993.
101. Bassi F, Marchisella C, Schierano G et al: Detection of platelet-activating factor in gingival tissue surrounding failed dental implants, *J Periodontol* 72:57-64, 2001.
102. Cornelini R, Rubini C, Fioroni M et al: Transforming growth factor-beta 1 expression in the peri-implant soft tissues of healthy and failing dental implants, *J Periodontol* 74:446-450, 2003.
103. Kao RT, Curtis DA, Richards DW et al: Increased interleukin-1 beta in the crevicular fluid of diseased implants, *Int J Oral Maxillofac Implants* 10:696-701, 1995.
104. Perala DG, Chapman RJ, Gelfand JA et al: Relative production of IL-1 beta and TNF alpha by mononuclear cells after exposure to dental implants, *J Periodontol* 63:426-430, 1992.
105. Shida J, Trindade MC, Goodman SB et al: Induction of interleukin-6 release in human osteoblast-like cells exposed to titanium particles in vitro, *Calcif Tissue Int* 67:151-155, 2000.
106. Shull MM, Ormsby I, Kier AB et al: Targeted disruption of the mouse transforming growth factor-beta 1 gene results in multifocal inflammatory disease, *Nature* 359: 693-699, 1992.
107. Eckes B, Zigrino P, Kessler D et al: Fibroblast-matrix interactions in wound healing and fibrosis, *Matrix Biol* 19:325-332, 2000.

WWW.HIGHDENT.IR

همیار دندانسازان

108. Choi BM, Kwak HJ, Jun CD et al: Control of scarring in adult wounds using antisense transforming growth factor-beta 1 oligodeoxynucleotides, *Immunol Cell Biol* 74:144-150, 1996.
109. Schierano G, Bellone G, Manzella C et al: In vitro effect of transforming growth factor-beta on adhesion molecule expression by human gingival fibroblasts cultured in the presence of a titanium abutment, *J Periodontol* 72:1658-1665, 2001.
110. Ammann AJ, Beck LS, DeGuzman L et al: Transforming growth factor-beta. Effect on soft tissue repair. *Ann NY Acad Sci* 593:124-34, 1990.
111. Assuma R, Oates T, Cochran D et al: IL-1 and TNF antagonists inhibit the inflammatory response and bone loss in experimental periodontitis, *J Immunol* 160: 403-409, 1998.
112. Delima AJ, Oates T, Assuma R et al: Soluble antagonists to interleukin-1 (IL-1) and tumor necrosis factor (TNF) inhibits loss of tissue attachment in experimental periodontitis, *J Clin Periodontol* 28:233-240, 2001.
113. Delima AJ, Karatzas S, Amar S et al: Inflammation and tissue loss caused by periodontal pathogens is reduced by interleukin-1 antagonists, *J Infect Dis* 186:511-516, 2002.
114. Connolly DT: Vascular permeability factor: a unique regulator of blood vessel function, *J Cell Biochem* 47: 219-223, 1991.
115. Cornelini R, Artese L, Rubini C et al: Vascular endothelial growth factor and microvessel density around healthy and failing dental implants, *Int J Oral Maxillofac Implants* 16:389-393, 2001.
116. Booth V, Young S, Cruchley A et al: Vascular endothelial growth factor in human periodontal disease, *J Periodontal Res* 33:491-499, 1998.
117. Takeshita S, Pu LQ, Stein LA et al: Intramuscular administration of vascular endothelial growth factor induces dose-dependent collateral artery augmentation in a rabbit model of chronic limb ischemia, *Circulation* 90:II228-II234, 1994.
118. Schultze-Mosgau S, Blatz MB, Wehrhan F et al: Principles and mechanisms of peri-implant soft tissue healing, *Quintessence Int* 36:759-769, 2005.
119. Rosen PS: Using recombinant platelet-derived growth factor to facilitate wound healing. *Compend Contin Educ Dent* 27:520-525, 2006.
120. Misch CE: Subantral augmentation. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.
121. Becker W, Becker BE: Guided tissue regeneration for implants placed into extraction sockets and for implant dehiscences: surgical techniques and case report, *Int J Periodontics Restorative Dent* 10:376-391, 1990.
122. Blanco J, Alonso A, Sanz M: Long-term results and survival rate of implants treated with guided bone regeneration: a 5-year case series prospective study, *Clin Oral Implants Res* 16:294-301, 2005.
123. Von Arx T, Kurt B: Implant placement and simultaneous peri-implant bone grafting using a micro titanium mesh for graft stabilization, *Int J Periodontics Restorative Dent* 8:117-127, 1998.
124. Moses O, Pitaru S, Artzi Z et al: Healing of dehiscence-type defects in implants placed together with different barrier membranes: a comparative clinical study, *Clin Oral Implants Restorative Dent* 16:210-219, 2005.
125. Covani U, Crespi R, Cornelini R et al: Immediate implants supporting single crown restoration: a 4-year prospective study, *J Periodontol* 75:982-988, 2004.
126. Kohal RJ, Mellas P, Hurzeler MB et al: The effects of guided bone regeneration and grafting on implants placed into immediate extraction sockets. An experimental study in dogs, *J Periodontol* 69:927-937, 1998.
127. Nemcovsky CE, Artzi Z: Comparative study of buccal dehiscence defects in immediate, delayed, and late maxillary implant placement with collagen membranes: clinical healing between placement and second-stage surgery, *J Periodontol* 73:754-761, 2002.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 38

Maxillary Sinus Anatomy, Pathology, and Graft Surgery

Carl E. Misch, Randolph R. Resnik, Francine Misch-Dietsh

Maxillary posterior partial or complete edentulism is one of the most common conditions in dentistry.[1] Thirty million people in the United States, or 17.5% of the adult population, are missing all of their maxillary teeth. In addition, 20% to 30% of the adult partially edentulous population older than 45 years is missing maxillary posterior teeth in one quadrant, and 15% of this age group is missing the maxillary dentition in both posterior regions. In other words, approximately 40% of adult patients are missing at least some maxillary posterior teeth. This chapter addresses the treatment concepts specific to the maxillary posterior partial or complete edentulous regions.

The maxillary posterior edentulous region presents many unique and challenging conditions in implant dentistry. Most noteworthy surgical methods include sinus grafts to increase available bone height, onlay grafts to increase bone width, and modified surgical approaches to insert implants in areas with poorer bone density.[2] Grafting of the maxillary sinus to overcome the problem of reduced vertical available bone has become a very popular and predictable procedure over the last decades. After the initial introduction by Tatum in the mid 1970s and the initial publication of Boyne and James in 1980, many studies have been published about sinus grafting with positive results.[3-38] In 1983, Misch[2] observed the most predictable intraoral region to grow bone height is on the maxillary sinus floor, once the sinus mucosa has been elevated. This statement still holds true today.

ANATOMICAL CONSIDERATIONS OF THE POSTERIOR MAXILLA

The maxilla has a thinner cortical plate on the facial compared with any region of the mandible. In addition, the trabecular bone in the posterior maxilla is finer than other dentate regions. The loss of maxillary posterior teeth results in an initial decrease in bone width at the expense of the labial bony plate. The width of the posterior maxilla decreases at a more rapid rate than in any other region of the jaws.[39] The resorption phenomenon is accelerated by the loss of vascularization of the alveolar bone and the existing fine trabecular bone type. However, because the initial residual ridge is so wide in the posterior maxilla, even with a 60% decrease in the width of the ridge, adequate-diameter root form implants usually can be placed. However, the ridge progressively shifts toward the palate until the ridge is resorbed into a medially positioned narrower bone volume.[40] The posterior maxilla continues to progressively remodel toward the midline as the bone resorption process continues. This results in the buccal cusp of the final restoration often being cantilevered facially to satisfy esthetic requirements at the expense of biomechanics in the moderate to severe atrophic ridges.[41]

DENTAL CONTRAINDICATIONS FOR IMPLANT TREATMENT OF THE POSTERIOR MAXILLA

A rule in traditional prosthodontics is that a fixed prosthesis is contraindicated when the canine and two adjacent teeth are missing. Therefore, when the canine and both premolars are missing, a fixed restoration is contraindicated. A removable prosthesis that has no movement under function is considered a fixed prosthesis and therefore should follow the rules of treatment planning for a fixed prosthesis in relation to implant number and position. A minimum of a healthy natural canine tooth or an implant abutment in the canine region is required before posterior implants are considered in the quadrant.

WWW.HIGHDENT.IR

همیار دندانسازان

Abnormal intraoral conditions may compromise the final outcome of sinus-grafting procedures and/or the survival rates of dental implants placed in the grafted sinuses. These contraindications are similar to those reported for standard implant treatment of edentulous patients and may be summarized as follows:

- Inadequate oral hygiene
- Untreated periodontal disease of the residual dentition
- Severe malocclusion
- Severe bruxism with loss of incisal guidance
- Active infection

Decreased Crown Height Space

The crown height space (CHS) should be evaluated before implant placement. Once the occlusal plane is properly restored or modified, the CHS should be greater than 8 mm (Figure 38-1). If less space is available for prosthodontic reconstruction, then a gingivectomy is first considered because it is not uncommon for excess tissue thickness to be present in this region. However, if tissue reduction cannot correct the CHS problem, then osteoplasty and/or vertical osteotomy of the maxillary posterior alveolar process are indicated to improve the vertical ridge orientation before implant surgery.

Poor Bone Density

In general, the bone quality is poorest in the posterior maxilla compared with any other intraoral region.[42] A literature review of clinical studies from 1981 to 2001 reveals the poorest bone density may decrease implant loading survival by an average of 16%, and it has been reported as low as 40%.[43] The cause of these failures is related to several factors. Bone strength is directly related to its density, and the poor-density bone of this region is often five to 10 times weaker compared with bone found in the anterior mandible.[44] Bone densities directly influence the bone-implant contact percent (BIC), which accounts for the force transmission to the bone. The BIC is least in D4 bone, and the stress patterns in this bone migrate farther toward the apex of the implant (Figure 38-2). As a result, bone loss is more pronounced and occurs also along the implant body, rather than only crestally as in other denser bone conditions. D4 bone also exhibits the greatest biomechanical elastic modulus difference when compared with titanium under load.[44] As such, strategic choices to increase BIC are suggested.

In the posterior maxilla, the deficient osseous structures and an absence of cortical plate on the crest of the ridge is often observed, which further compromises the initial implant stability at the time of insertion. The labial cortical plate is thin, and the ridge is often wide. As a result, the lateral cortical BIC to stabilize the implant is often insignificant. Implant surgery more often uses bone compression rather than bone extraction to create the implant osteotomy to compensate for these deficiencies. As a consequence, initial healing of an implant in D4 bone is often compromised.

Figure 38-1 The crown height space (CHS) in the posterior maxilla should be at least 8 mm for prosthetic reconstruction. Reduced CHS requires osteoplasty before implant insertion, which reduces the existing available bone height.

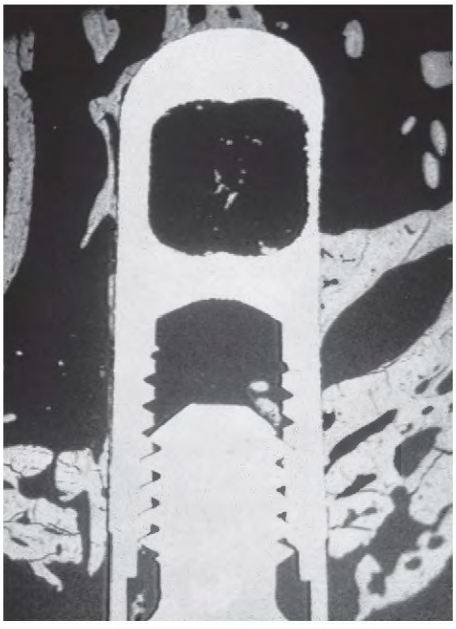


Figure 38-2 The bone-implant contact percent is often reduced in the posterior maxilla, because the quality of bone is poorer than other regions of the mouth.

Implant Size

The ideal length of the implant is directly related to the implant width, the design, the amount of the forces, and the bone density. Because implant success after loading is reduced in implants shorter than 10 mm, it is logical

WWW.HIGHDENT.IR

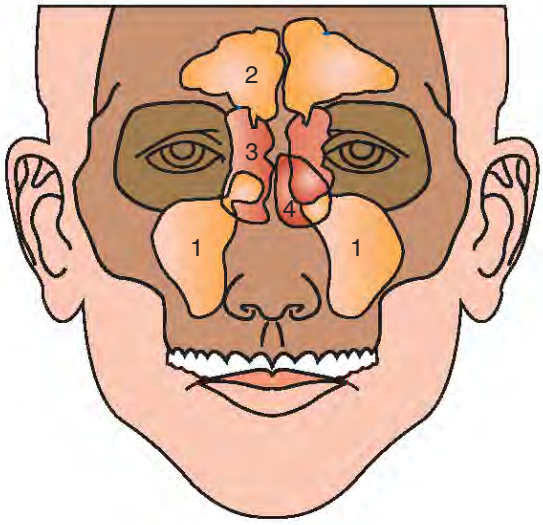


Figure 38-3 The maxillary sinuses *(1)* are the largest of the paranasal sinuses, followed by the frontal *(2)*, ethmoid *(3)*, and sphenoid sinuses *(4)*.

to treatment plan longer implants in the region. In general, 4-mm threaded root form implants should be at least 12 mm in length when the bone density is poor. This usually provides adequate BIC to dissipate the loads applied to the prosthesis. Implants longer than 16 mm are not necessary, even in the softest bone type, because the stress transfer is not dissipated beyond this length. Therefore the implant most often should be 12 to 16 mm long in this region of the mouth. Sinus grafting is usually required before or in conjunction with implant placement to gain adequate bone height for implants of adequate length in this region. The poor bone density requires implants of a larger size, including length. The implant length requirement is greater because the transmission of stress is expressed farther down the implant length.

MAXILLARY SINUS

The anatomy of the maxillary sinuses was first illustrated and described by Leonardo da Vinci in 1489 and later documented by the English anatomist Nathaniel Highmore in 1651.[45] The maxillary sinus (or antrum of Highmore) lies within the body of the maxillary bone and is the largest of the paranasal sinuses, as well as the first to develop (Figure 38-3).

Maxillary Sinus Development

A primary pneumatization of the maxillary sinus occurs at about 3 months of fetal development by an out-pouching of the nasal mucosa within the ethmoid infundibulum. At this time the maxillary sinus is a bud situated at the infralateral surface of the ethmoid infundibulum between the upper and middle meatus.[46] Prenatally, a secondary pneumatization occurs. At birth, the sinuses are filled with fluid and the maxillary sinus is still an oblong groove on the mesial side of the maxilla, just above the germ of the first deciduous molar.[47]

Postnatally and until the child is 3 months of age, the growth of the maxillary sinus is closely related to the pressure exerted by the eye on the orbit floor, the tension of the superficial musculature on the maxilla, and the forming dentition. As the skull matures, these three elements influence its three-dimensional (3D) development. At 5 months, the sinus appears as a triangular area medial to the infraorbital foramen.[48]

During the child's first year, the maxillary sinus expands laterally underneath the infraorbital canal, which is protected by a thin bony ridge. The antrum grows apically and progressively replaces the space formerly occupied by the developing dentition. The growth in height is best reflected by the relative position of the sinus floor. At 12 years of age, pneumatization extends to the plane of the lateral orbital wall, and the sinus floor is level with the floor of the nose. The main development of the antrum occurs as the permanent dentition erupts and pneumatization extends throughout the body of the maxilla and the maxillary process of the zygomatic bone. Extension into the alveolar process lowers the floor of the sinus about 5 mm (Figure 38-4). Anteroposteriorly, the sinus expansion corresponds to the growth of the midface and is completed only with the eruption of the third permanent molars when the young person is about 16 to 18 years of age.[46-48]

In the adult, the sinus appears as a pyramid of five thin, bony walls. The base of this pyramid faces the lateral nasal wall and often measures 33 × 33 mm; its apex extends approximately 23 mm toward the zygomatic bone.[49,50] The dentate adult maxillary sinus has an average volume of 15 mL, although the range is 9.5 to 20 mL.[51] The floor of the maxillary sinus cavity is reinforced by bony or membranous septa joining obliquely or transversely from the medial and/or lateral walls with buttresslike webs. They may be genetic or develop as a result of stress transfer within the bone over the roots of teeth. These have the appearance of reinforcement webs in a wooden boat and rarely divide the antrum into separate compartments. These elements are present from the canine to the molar region, and Misch[41] has observed they tend to disappear in the maxilla of the long-term edentulous patient when stresses to the bone are reduced. Karmody et al.[52] found that the most common oblique septum is located in the superior anterior corner of the sinus or infraorbital recess (which may expand anteriorly to the nasolacrimal duct). The medial wall is juxtaposed with the middle and inferior meatus of the nose.

Although the adult maxillary sinus maintains its overall size while the teeth are present, a rather rapid expansion phenomenon of the maxillary sinus occurs

WWW.HIGHDENT.IR

همیار دندانسازان

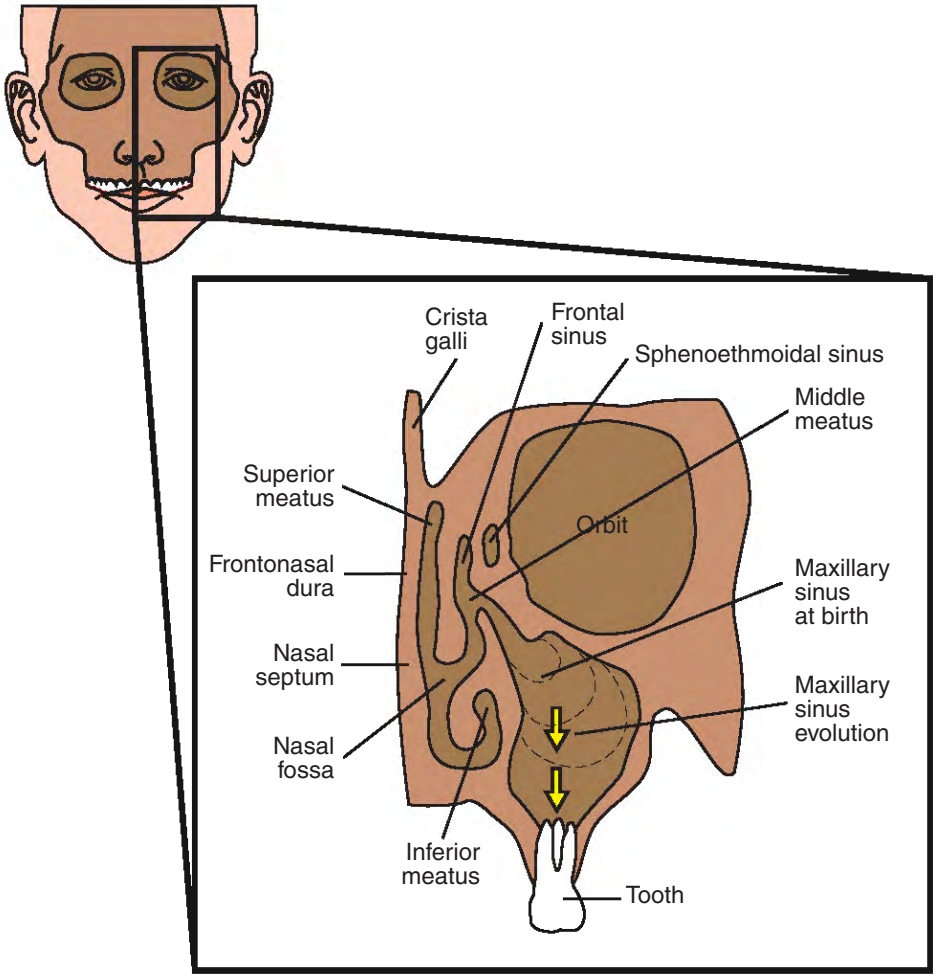


Figure 38-4 The maxillary sinus development increases in size during the growth of the midface of the maxilla and is also related to the eruption of the deciduous teeth, followed by the permanent dentition.

with the loss of posterior teeth. In fact, even with the loss of a single molar, the sinus expands between the adjacent tooth roots. In the edentulous maxilla, the antrum expands in both inferior and lateral dimensions and may even invade the canine eminence region and proceed to the lateral piriform rim of the nose. This results in a lack of available bone in the posterior maxilla and a greatly decreased height from both the sinus expansion and the crestal resorption. Frequently, less than 10 mm remains between the alveolar ridge crest and the floor of the maxillary sinus in the edentulous posterior maxilla. This limited dimension is compounded by the problem of bone of reduced quality and the resultant medial posterior position of the ridge after resorption of bone width. As a result, compromises in the long-term prognosis of many endosteal implant systems are reported.[43]

Maxillary Sinus Anatomy

Maxillary sinus grafts have become a predictable procedure before or in conjunction with implant insertion. However, infections of the maxillary sinus are common and may not only cause implant failure but also may be life-threatening to the patient. As a result, the dentist should be knowledgeable of both the macroscopic and microscopic anatomy, as well as the pathologic conditions that may occur before or postoperative to the sinus graft procedure.

Six bony walls that contain many structures of concern during sinus graft surgery surround the maxillary sinus.[45,53] Knowledge of these structures is crucial for both preoperative assessment and postsurgical complications.

Anterior Wall

The anterior wall of the maxillary sinus consists of thin, compact bone extending from the orbital rim to just above the apex of the cuspid. With the loss of the canine, the anterior wall of the atrum may approximate the crest of the residual ridge. Within the anterior wall and approximately 6 to 7 mm below the orbital rim, with anatomic variants as far as 14 mm from the orbital rim, is the infraorbital foramen (Figure 38-5).

WWW.HIGHDENT.IR

Figure 38-5 The infraorbital foramen is located approximately 10 mm below the orbital rim. The anterior wall of the maxillary sinus in this skull has a horizontal cut just below the infraorbital foramen. The thin anterior wall is often used for surgical access to the maxillary sinus for Caldwell-Luc procedures.

Figure 38-6 A frontal section of a head demonstrates the frontal sinus above and between the orbits and the maxillary sinuses directly below the orbits. A thin bony wall acts as the floor of the orbit and superior wall of the maxillary sinus. On the left, the infraorbital canal is in the outer third of the superior wall. On the right, the infraorbital canal is located at the junction of the apex of the superior and lateral-wall connection. The lateral wall of the sinus in this edentulous adult is the thickest wall but is only 1 mm thick. The medial wall of the maxillary sinus is thin, separates the maxillary sinus from the nares, and houses the inferior meatus in the bottom half of the sinus. Located in the superior-most aspect of this wall is the primary ostium that connects the maxillary sinus to the nares. The inferior wall represents the base of the existing posterior available bone and is the floor of the maxillary sinus.

The infraorbial nerve runs along the roof of the sinus and exits through the foramen. The infraorbital blood vessels and nerves lie directly on the superior wall of the interior and within the sinus mucosa. Tenderness to pressure over the infraorbital foramen or redness of the overlying skin may indicate inflammation of the sinus membrane from infection or trauma. The anterior wall of the maxillary sinus may serve as surgical access during Caldwell-Luc procedures to treat a preexisting or post–sinus graft, pathologic condition.

Superior Wall

The superior wall of the maxillary sinus is shared with the thin orbital floor (Figure 38-6). The orbital floor slants inferiorly in a mediolateral direction and is convex into the sinus cavity. A bony ridge is usually present in this wall that houses the infraorbital canal containing the infraorbital nerve and associated blood vessels. Dehiscence of the bony chamber may be present, resulting in direct contact between the infraorbital structures and the sinus mucosa. Ocular symptoms may result from infections or tumors in the superior aspects of the sinus region and may include proptosis (bulging of the eye) and diplopia (double vision). When these problems occur, the patient is closely supervised and a medical consult is advised to decrease the risk of severe complications that may result from the spread of infection in a superior direction. Superior spreading infections may lead to a brain abscess and death. As a result, when these symptoms appear, aggressive therapy to decrease the spread of infection is indicated. Overpacking the maxillary sinus with bone graft material during a sinus graft may result in pressure against the superior wall if a sinus infection develops.

Posterior Wall

The posterior wall of the maxillary sinus corresponds to the pterygomaxillary region, which separates the antrum from the infratemporal fossa. The posterior wall usually has several vital structures in the region of the pterygomaxillary fossa, including the internal maxillary artery, pterygoid plexus, sphenopalatine ganglion, and greater palatine nerve. The posterior wall should always be identified on the radiograph. When lack of a posterior wall is present, a pathologic condition (including neoplasms) is to be suspected.

It should be noted that pterygoid implants placed through the posterior sinus wall and into this region might approach vital structures, including the maxillary artery. Therefore a blind surgical technique to place a pterygoid implant through the posterior wall may have increased surgical risk. Because pterygoid implants are often positioned in the third or fourth molar region, they are of benefit primarily when third or fourth molars are needed for prosthetic reconstruction or sinus grafts are contraindicated and available bone posterior to the antrum is present.

WWW.HIGHDENT.IR

همیار دندانسازان

Medial Wall

The medial wall of the antrum coincides with the lateral wall of the nasal cavity and is the most complex of the various walls of the sinus (see Figure 38-6). On the nasal aspect, the lower section of the medial wall corresponds to the lower meatus and floor of the nasal fossa; the upper aspect corresponds to the middle meatus. The medial wall is vertical and smooth on the antral side. Located in the superior aspect of the medial wall is the maxillary or primary ostium. This structure is the main opening through which the maxillary sinus drains its secretions via the ethmoid infundibulum through the hiatus semilunaris into the middle meatus of the nasal cavity. The infundibulum is approximately 5 to 10 mm long and drains via ciliary action in a superior and medial direction. This opening corresponds to the initial site of embryonic evagination from the nasal chamber. The ostium diameter averages 2.4 mm in health; however, pathologic conditions may alter the size to vary from 1 to 17 mm. Smaller, accessory or secondary ostia may be present that are usually located in the middle meatus posterior to the main ostium. These additional ostia are usually the result of chronic sinus inflammation and mucous membrane breakdown. They are present in approximately 30% of skulls, range from a fraction of a millimeter to 0.5 cm, and are commonly found within the membranous fontanelles of the lateral nasal wall.[54] Fontanelles are usually classified either as anterior fontanelles (AFs) or posterior fontanelles (PFs) and termed by their relation to the uncinate process. These weak areas in the sinus wall are sometimes used to create additional openings into the sinus for treatment of chronic sinus infections. Primary and secondary ostia may, on occasion, combine and form a large ostium within the infundibulum.

Lateral Wall

The lateral wall of the maxillary sinus forms the posterior maxilla and the zygomatic process (see Figure 38-6). This wall varies greatly in thickness from several millimeters in a dentate patient to less than 1 mm in an edentulous patient. The lateral wall houses an endosseous anastamosis of the infraorbital and posterior superior alveolar artery. The Tatum lateral-wall approach sinus graft procedure uses this area for entrance into the maxillary sinus (Figure 38-7).

Inferior Wall

The inferior wall or floor of the maxillary sinus is in close relationship with the apices of the maxillary molars and premolars (see Figure 38-6). The teeth usually are separated by the sinus mucosa by a thin layer of bone; however, on occasion, teeth may perforate the floor of the sinus and be in direct contact with the sinus lining. Studies have shown that the first molar has the most common dehiscent tooth root, occurring approximately 2.2% of the time. In dentate patients, the floor is approximately at the level of the nasal floor. In edentulous posterior maxillae, the sinus floor is often 1 cm below the level of the nasal floor. Complete or incomplete bony septa may exist on the floor in a vertical or horizontal plane. Almost 30% of the dentate maxillae have septa, with three fourths appearing in the premolar region.[55,56] Complete septa separating the sinus into compartments are very rare, occurring in only 1.0% to 2.5% of maxillary sinuses.

Maxillary Sinus: Vascular Supply and Innervation

The blood supply to the maxillary sinus comes directly from the maxillary artery, which emanates from the external carotid artery. The maxillary artery supplies the bone surrounding the sinus cavity and also the sinus membrane. Branches of the maxillary artery, which most often include the posterior superior alveolar artery and infraorbital artery, form endosseous and extraosseous anastomoses that encompass the maxillary sinus. The endosseous anastomosis is found within the lateral wall of the sinus and supplies this structure and the lateral aspect of the sinus membrane. A posterior lateral nasal artery (a branch of the sphenopalatine artery that also rises from the maxillary artery) also supplies this region from the medial aspect of the sinus.

The formation of the endosseous and extraosseous anastomoses in the maxillary sinus is termed the *double arterial arcade*. The extraosseous anastomosis is less common (present in 44% of the population) and is found near the periosteum of the lateral wall. The extraosseous anastomosis is superior to the endosseous unit, which is approximately 15 to 20 mm from the dentate alveolar crest. In addition to the double arterial arcade, a blood supply from the sphenopalatine artery supplies the central and medial parts of the sinus

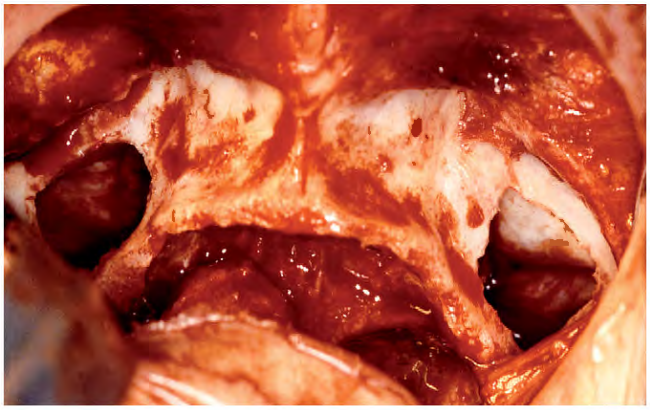


Figure 38-7 The lateral wall of the maxillary sinus most often extends from the second premolar region to the posterior wall of the maxilla. This wall serves as the surgical access to the sinus floor and medial wall for the sinus graft procedure. In this intraoral view, the lateral sinus wall has been infractured and now serves as part of the ceiling of the sinus graft or as the floor of the smaller maxillary sinus.

WWW.HIGHDENT.IR

Box 38-1 Arterial Supply to Posterior Maxilla (Double Arterial Arcade)

Endosseous Anastamosis (within the Lateral Wall of Sinus)

Lateral wall of sinus lateral and sinus membrane supplied by

Posterior superior alveolar artery
Infraorbital artery

Extraosseous Anastamosis (within Periosteum and Below the Zygomatic Arch)

Sinus mucous membranes supplied by

Posterior superior alveolar artery
Infraorbital artery
Posterior lateral nasal artery (medial and posterior wall)

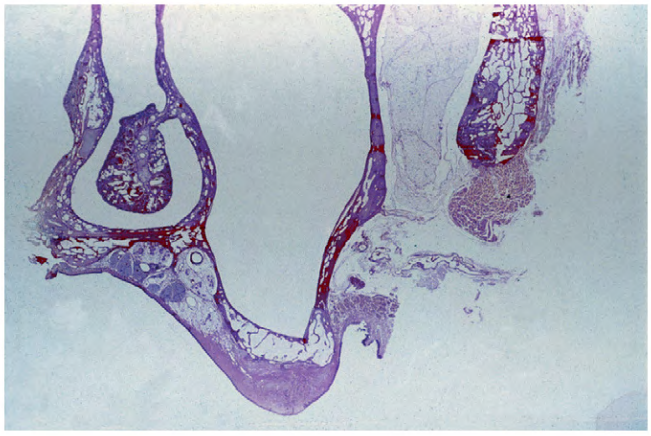


Figure 38-8 A maxillary ridge with advanced resorption results in a thinner lateral wall with no endosseous anastomosis. In this histologic cross section of a posterior edentulous maxilla, the available bone below the antrum is 2 mm in height and the lateral wall is 0.5 mm thick; a lack of endosseous anastomosis is evident.

membrane. This artery is also a branch of the maxillary artery and enters the maxillary sinus on the medial aspect via the maxillary ostium[57] (Box 38-1).

In an edentulous maxilla with posterior vertical bone loss, the endosseous anastamosis may be 5 to 10 mm from the edentulous ridge. The endosseous artery was able to be observed on computed tomography (CT) scans in approximately one half of the patients requiring a sinus graft.[58] In a long-term edentulous patient with a thin lateral wall, the artery may be atrophied and almost nonexistent. On rare occasions, this anatomical structure may be a concern for arterial bleeding complications during lateral-approach sinus elevation surgery.

Most often, implants of 12 to 16 mm in length should be used to support prostheses in the posterior maxilla, because the bone quality is poor and the occlusal force factors are high. Because the endosseous anastomosis is often present and ranges from 5 to 20 mm from the residual crest of the ridge, the lateral-access window for the sinus graft procedure often violates this arterial supply. However, this is usually of little concern during the surgical procedure and may even be of benefit postsurgery to provide a blood supply to the graft.

The size of the lateral-wall arterial anastomoses usually approximates 1.5 mm in a dentate patient and has little arterial pressure. Although the artery is often in the pathway of surgery for the Tatum lateral-wall approach to sinus grafting, endosseous bleeding is rarely observed as a pulsating rhythm and may be easily arrested by the following: (1) cutting the bone and vessel with a high-speed diamond and no irrigation (which cauterizes the vessel), (2) using electrosurgery on the vessel, and/or (3) elevating the head and using a surgical sponge (gauze) and slight pressure over the region for a few minutes. A hemostat may also be used to clamp the vessel, because it is readily observed; however, fracture of the lateral wall may occur.

The medial and posterior aspects of the maxillary sinus mucosa receive their blood supply from the posterior lateral nasal artery. The sphenopalatine artery is the third or fourth branch of the maxillary artery and enters the nasal cavity through the sphenopalatine foramen, which is near the posterior portion of the superior meatus of the nose. Once through the foramen, the sphenopalatine artery branches into the posterior lateral nasal artery and the posterior septal artery. The posterior lateral nasal artery supplies the medial and posterior walls of the antrum (nasal cavity).

The entire circulatory system in the maxillary sinus is a vital part of the healing and regeneration of bone after a sinus graft. Different factors can alter the vascularization in this area. With increasing age, the number and size of blood vessels in the maxilla decrease. As bone resorption increases, the cortical bone becomes thin, resulting in less vascularization. As the lateral wall becomes thinner, the blood supply to the lateral wall and lateral aspect of the bone graft comes primarily from the periosteum, resulting in a compromised vascularization to the region (Figure 38-8). After surgery the arterial supply in the lateral outer wall (in spite of being severed during the procedure) may help vascularize the sinus graft, because the anastomoses may vascularize the graft from both the posterior region and anterior region.

The venous drainage of the maxillary sinus originates from a vascular plexus near the maxillary ostium that drains anteriorly into the facial vein and posteriorly into the maxillary and jugular veins.

The nerve supply to the maxillary sinus is via numerous branches of the second division of the trigeminal nerve, including the posterior alveolar nerves, anterior palatine, and infraorbital nerves. The infraorbital nerve is of concern in sinus elevation surgery because of its anatomical position. This nerve enters the orbit via the inferior orbital fissure and continues anteriorly; the

WWW.HIGHDENT.IR

همیار دندانسازان

nerve lies in a groove in the orbital floor (which is also the maxillary sinus superior wall) before exiting the infraorbital foramen. Anatomical variants have been reported to include a dehiscence and malpositioned infraorbital foramen, along with the nerve transversing the lumen of the maxillary sinus rather than coursing through the bone within the sinus ceiling (orbital floor).

Maxillary Sinus Mucosa: Microscopic Anatomy

The microscopic primary epithelial cells of the maxillary sinus are a continuation of the nasal mucosa and classified as a pseudostratified, ciliated columnar epithelium. However, the epithelial lining of the maxillary sinus is much thinner and contains fewer blood vessels compared with the nasal epithelium. This accounts for the membrane's paler color and bluish hue. Five primary cell types exist in this tissue: (1) ciliated columnar epithelial cells, (2) nonciliated columnar cells, (3) basal cells, (4) goblet cells, and (5) seromucinous cells. The ciliated cells contain approximately 50 to 200 cilia per cell. In a healthy patient, they help to clear mucus from the sinus and into the nose. The nonciliated cells comprise the apical aspect of the membrane, contain microvilli, and serve to increase surface area. These cells have been theorized to facilitate humidification and warming of inspired air. The basal cell's function serves most likely as a stem cell that can differentiate as needed. The goblet cells produce glycoproteins that are responsible for the viscosity and elasticity of the mucus produced. The maxillary sinus has the highest concentration of goblet cells in comparison with the other paranasal sinuses.

Although the sinus mucosa has been called a *mucoperiosteum*, Sharawy and Misch[59] suggested that the periosteal portion of this membrane is not similar to the periosteum covering the cortical plates of the maxillary or mandibular residual ridges of the jaws and should not be considered a mucoperiosteum (Figure 38-9). The very minimal and usual absence of osteoblasts in this tissue and, instead, the presence of osteoclasts, may account for the enlargement of the antrum after tooth loss. The maxillary sinus membrane also exhibits few elastic fibers attached to the bone,[59] which simplifies elevation of this tissue from the bone. The thickness of the sinus mucosa varies, but is generally 0.3 to 0.8 mm.[60] In smokers, it varies from very thin and almost nonexistent to very thick, with a squamous type of epithelium.

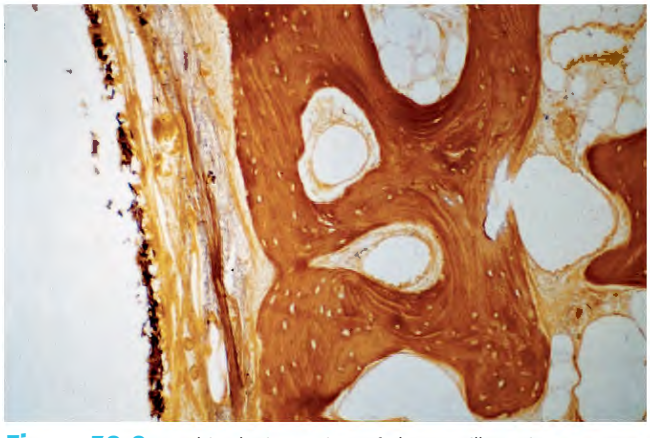


Figure 38-9 A histologic section of the maxillary sinus mucosa demonstrates cilia to the left. An absence of blood vessels and elastic fibers exists in the tissue adjacent to the surrounding bone. No osteoblasts are evident between the sinus mucosa and the surrounding walls of bone.

Maxillary Sinus Mucous Clearance

The mucus of the maxillary sinuses is produced from serous and goblet cells, which produce 1 L of mucus each day in healthy conditions. The cilia in the maxillary sinus beat toward the ostium. A blanket of mucus is propelled toward the ostium by the beating motion of the ciliated lining cells. The mucous material of the sinus in health has two layers: (1) a top mucoid layer and (2) a bottom serous layer (Figure 38-10). The top layer is sticky and collects bacteria and other debris, whereas the serous layer is thin and acts as a lubricant. The cilia of the columnar epithelium beat toward the ostium at 15 cycles per minute, with a stiff stroke through the serous layer, reaching into the mucoid layer. The cilia recover with a limp reverse stroke within

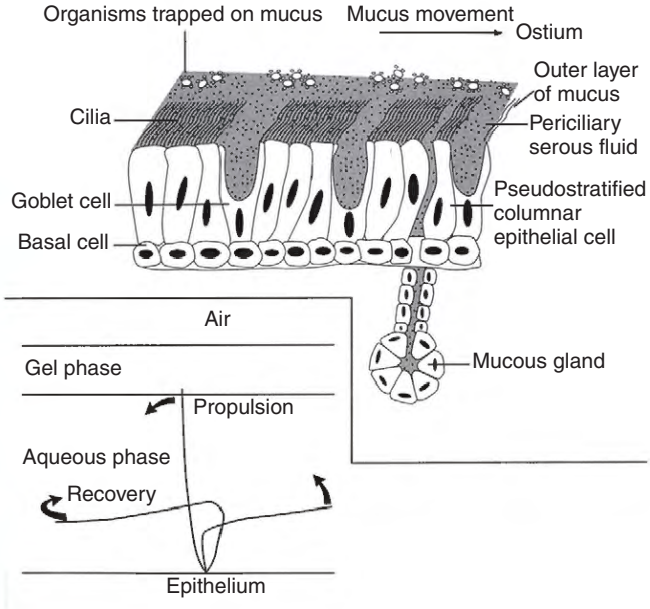


Figure 38-10 The pseudostratified columnar epithelium cells have 50 to 200 cilia per cell that beat toward the ostium to help clear 1 L of mucus from goblet and mucous glands each day from the sinus. In health, the mucous has two layers: a bottom serous layer and top mucoid layer. The cilia beat with a stiff stroke in the mucoid layer toward the ostium and a relaxed recovery stroke within the serous layer.

WWW.HIGHDENT.IR

the serous layer. This mechanism slowly propels the mucoid layer toward the ostium at a rate of 9 mm per minute and into the middle meatus of the nose.[60]

Various elements may decrease the number of cilia and slow their beating efficiency. Viral infections, pollution, allergic reactions, and certain medications may affect the cilia in this way.[61-63] Genetic disorders (e.g., dyskinetic cilia syndrome) and factors such as long-standing dehydration, anticholinergic medications and antihistamines, cigarette smoke, and chemical toxins also can affect ciliary action. Certain pathogens *(Haemophilus influenzae, Streptococcus pneumoniae, Pseudomonas aeruginosa)* associated with sinusitis have been shown to release a compound that slows and disorganizes ciliary beating along with affecting mucous transport. Various medications have also been shown to affect ciliary action. Decongestant medications may alter the various layers of the sinus membranes, thus interrupting normal ciliary activity. On the other hand, long-term antibiotic medications have been shown to have a significant increase in ciliary beat frequency.[61]

In a healthy sinus, an adequate system of mucus production, clearance, and drainage is maintained. The key to normal sinus physiologic condition is the proper function of the cilia, which are the main component of the mucociliary transport system. These cilia work to move contaminants toward the natural ostium and then to the nasopharynx. The flow of mucus is toward the ostium, which leads to the infundibulum, through the hiatus semilunaris, into the middle meatus of the nose, and ultimately into the nasopharynx (Figure 38-11). An alteration in the sinus ostium patency or the quality of secretions can lead to disruption in ciliary action resulting in sinusitis. For clearance to be maintained, adequate ventilation is necessary. Ventilation and drainage is dependent on the osteomeatal unit, which is the main sinus opening. Ciliary movements of ciliated epithelial cells dictate clearance of the maxillary sinus.

The maxillary ostium and infundibulum are part of the anterior ethmoid middle meatal complex, the region through which the frontal and maxillary sinuses drain, which is primarily responsible for mucociliary clearance of the sinuses to the nasopharynx.[64] As a result, pathogenesis of sinusitis is usually the development of obstruction in one or more areas of this complex.[65] Symptomatic sinusitis corresponds to the blockage of the osteomeatal complex that drains the paranasal sinuses. Symptoms of sinusitis may be minimal, even in the presence of extensive radiographic findings, as long as the patency of the ostium is maintained.[66]

Mucoid fluid is transported toward the ostium of the maxillary sinus and drained into the nasal cavity, eliminating inhaled small particles and microorganisms. This mucociliary transport system is an active transport system that relies heavily on oxygen. The amount of oxygen absorbed from the blood is not adequate to maintain this drainage system. Additional oxygen has to be absorbed from the air in the sinus; this is why the patency of the ostium is pivotal in maintaining this transport system. Mucociliary transport may be compromised by abnormalities in the cilia, which include a decrease in the number and poor coordination of movement. After sinus infections, the quantitative loss of cilia is reversed except when severe empyema with scarring occurs, which results in the ciliated epithelium being replaced by squamous epithelium.

Maxillary Sinus Bacterial Flora

Much debate concerns the bacterial flora of the maxillary sinus. Maxillary sinuses have been considered to be sterile in health; however, bacteria can colonize within them without producing symptoms. In theory, the mechanism by which a sterile environment is maintained includes the mucociliary clearance system, immune system, and the production of nitrous oxide within the sinus cavity. In a study of patients who underwent surgical repositioning of the maxilla, Cook and Haber[67] reported that 80% of the patients showed no bacterial growth. The remaining 20% had some bacterial growth but in negligible numbers. All specimens showed some degree of inflammatory cell infiltrate, more acute in the presence of microorganisms. They concluded that the asymptomatic adult maxillary sinus is usually sterile, but a few transient bacteria may exist in a clinically asymptomatic antrum.

However, recent endoscopically normal sinuses were shown to be nonsterile, with 62.3% exhibiting bacterial colonization. The most common bacteria cultured were *Streptococcus viridans*, *Staphylococcus epidermidis*, and *Streptococcus pneumoniae*.[68] The culture findings for secretions in acute maxillary sinusitis yielded high numbers of leukocytes, *S. pneumoniae,* or *Streptococcus pyogenes*, and *Haemophilus influenzae* was recovered from the purulent exudates with lower numbers of staphylococci. In fact, the macroscopic appearance of the secretion should not be used to screen samples, because several cases with *H. influenzae* grew also from nonpurulent samples. Other reports have indicated the bacterial flora of the maxillary sinus consists of nonhemolytic and α-hemolytic streptococci, as well as *Neisseria* spp. Additional microorganisms identifiable in various quantities belong to the staphylococci, *Haemophilus* spp., pneumococci, *Mycoplasma* spp., and *Bacteroides* spp. This is important to note because the sinus graft procedure often violates the sinus mucosa, and bacteria may contaminate the graft site.

Maxillary Sinus: Clinical Assessment

To establish an adequate osseous morphologic condition for the placement of endosteal implants in the resorbed maxillary posterior region, various grafting techniques have been developed to increase bone volume. In

WWW.HIGHDENT.IR

همیار دندانسازان

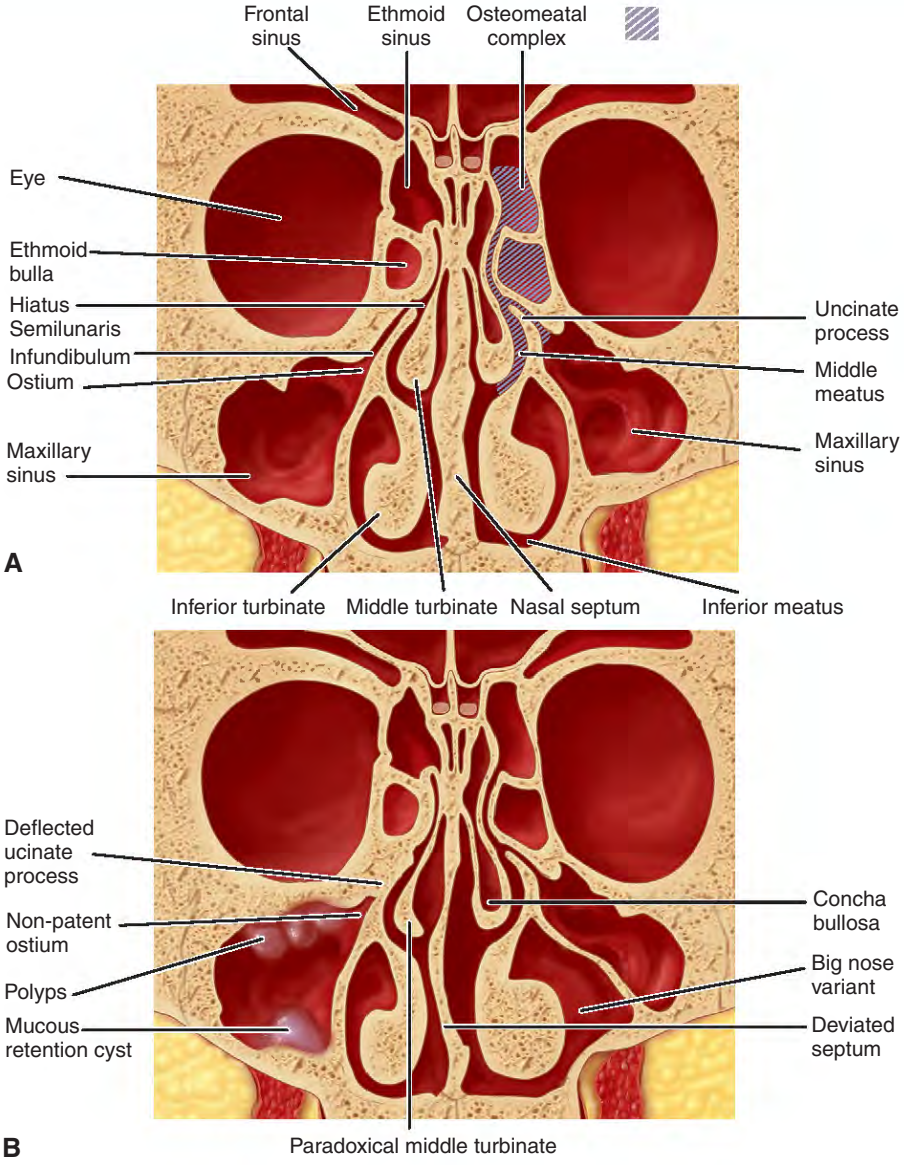


Figure 38-11 **A,** Normal paranasal anatomy. **B,** Paranasal pathology and anatomical variants.

1987, Misch[69] developed four different categories for the treatment of the posterior maxilla (termed *subantral* [SA]), as SA-1 through SA-4 (Figure 38-12). The SA-1 posterior maxilla allows implant placement inferior to the sinus cavity without sinus manipulation, thus not altering the sinus floor or membrane. As such, if the patient has a preexisting maxillary sinus condition or develops a sinus infection after implant placement, then implants are not at risk of becoming contaminated. However, the SA-2 to SA-4 surgical procedures do alter the sinus membrane and sinus floor. With these treatment options, a thorough preoperative evaluation is completed to rule out existing pathologic condition in the maxillary sinus. In this way, the risk of mucus and bacteria contaminating the graft and creating a bacterial smear layer on the implant, which results in impaired bone formation during healing, is reduced. In addition, because of the proximity of the maxillary sinus to numerous vital structures, postoperative complications can be very severe and even life threatening.

Pathologic conditions associated with the paranasal sinuses are common ailments and afflict more than 31 million people each year. Approximately 16 million people will seek medical assistance related to sinusitis; yet sinusitis is one of the most commonly overlooked diseases in clinical practice.[70] Potential infection in the region of the sinuses may result in severe complications. Infections in this area have been reported to result in sinusitis, orbital cellulitis, meningitis, osteomyelitis, and cavernous sinus thrombosis. In fact, paranasal sinus infection accounts for approximately 5% to 10% of all brain abscesses reported each year.[70]

WWW.HIGHDENT.IR

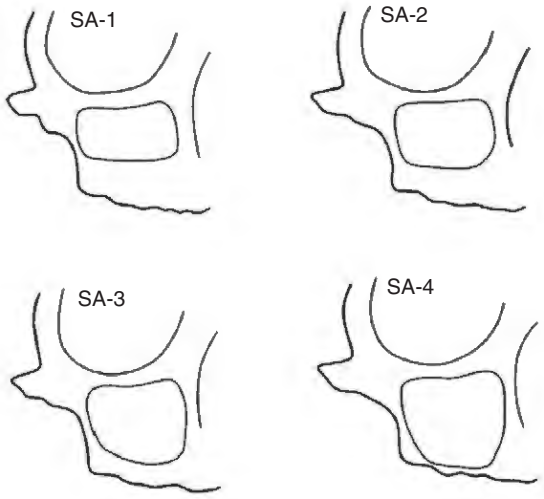


Figure 38-12 Misch classified the subantral (SA) region of the posterior maxilla in four categories: SA-1 has adequate vertical bone for endosteal implants (>12 mm), SA-2 has 0 to 2 mm less than ideal height of bone (10 to 12 mm), SA-3 has 5 to 10 mm of bone below the antrum, and SA-4 has less than 5 mm of vertical bone below the maxillary sinus.

Table 38-1 Preoperative and Postoperative Physical Examination

SITE	SIGNS OF INFECTION
Inferior wall	Bulge in hard palate, ill-fitting denture, loose teeth, hypesthesia or nonvital teeth, bleeding, palatal erosion, oroantral fistula
Medial wall	Nasal obstruction, nasal discharge, epistaxis, cacosmia, visible mass in nostril
Anterior wall	Swelling, pain, skin changes
Lateral wall	Trismus, bulging mass, exudate from incision line
Posterior wall	Midface pain, hypesthesia of one half of face, loss of function of lower cranial nerves
Superior wall	Diplopia (double vision), proptosis (eye bulging out), chemosis, pain or hypesthesia, decreased visual acuity

A physical examination of the maxillary sinus evaluates the middle third of the face for the presence of asymmetry, deformity, swelling, erythema, ecchymosis, hematoma, or facial tenderness (Table 38-1). Nasal congestion or obstruction, prevalent nasal discharge, epistaxis (bleeding from the nose), anosmia (the loss of the sense of smell), and/or halitosis (bad breath) are noted.

The clinical examination for maxillary sinusitis concerns the regions surrounding the maxillary antrum. The examination is conducted to assess each wall surrounding the maxillary sinus separately. The infraorbital foramen on the facial wall of the antrum is palpated through the soft tissue of the cheeks or intraorally to determine whether tenderness or discomfort is present. The intraoral examination assesses the floor of the antrum by alveolar ulceration, expansion, tenderness, paresthesia, and oroantral fistulae. The eyes are examined to evaluate the superior wall of the sinus for proptosis, pupillary level, lack of eye movement, and diplopia. The nasal fluids may be used to evaluate the medial wall of the sinus by asking the patient to blow the nose in a waxed paper. The mucus should be clear and thin in nature. A yellow or greenish tint or thickened discharge indicates infection. Infected maxillary sinuses typically are symptomatic, which can exhibit exudate in the middle meatus and may be inspected with a nasal speculum and headlight (rhinoscopy) through the nares.

Other methods of examination of the infected maxillary sinus may include transillumination, nasoendoscopy, bacteriology, cytology, fiber optic antroscopy, and radiography (conventional, CT, or magnetic resonance imaging [MRI]).

Maxillary Sinus Radiographic Evaluation

The maxillary sinus is often pneumatized in partial or completely edentulous patients and requires grafting. Therefore visualization of the maxillary sinus and surrounding structures are crucial for the proper diagnosis and treatment planning of the partial or complete edentulous posterior maxilla. Numerous preoperative radiographic modalities are available to evaluate the posterior maxilla and maxillary sinuses.

Waters' Projection Radiographs

The most common medical plain-film radiograph used for the evaluation of the maxillary sinus is the occipitomental projection, also termed the *Waters' projection*. This film is taken with the patient's head tilted upwards approximately 40 degrees, allowing a clear evaluation of the superior, lateral, and medial aspects of the maxillary sinus. Before the advent of CT technology, this projection was used routinely for the evaluation of maxillary sinus disease. The Waters' projection is often complimented with similar plain films such as the Caldwell (frontal view), lateral view, and submentovertical (base view). However, the posterior teeth and/or alveolar residual bony process frequently obscure the posteroinferior part of the sinus cavity. Because the sinus floor is a critical portion of the sinus for implant treatment, a Waters' projection radiograph has little use for diagnosis or treatment planning in implant dentistry. It has been reported that 75% of these plain films are inaccurate in evaluating the pathologic condition of the sinuses.[71]

Panoramic Radiographs

The panoramic radiograph is often used as a preliminary diagnostic radiograph in implant dentistry. This

WWW.HIGHDENT.IR

همیار دندانسازان

radiograph can provide direct visualization of the anterior, lateral, and inferior regions of the maxillary sinus. It is often sufficient to evaluate the amount of bone present below the maxillary sinus (Figure 38-13).

When a panoramic radiograph is first evaluated, attention is brought to the amount of bone height in the posterior maxilla from the crest of the ridge to the floor of the maxillary sinus. This available bone height may place the patient in a category of SA-1 (obviously enough bone below the antrum for implants) or SA-4 (obviously not enough bone for implants). The SA-2 and SA-3 categories are less obvious, because the specific bone height range of 5 or 10 mm of bone may be influenced by film magnification.

The hard palate appears as two radiopaque lines on the panoramic radiograph: one line represents the structure on that side, and a second, lighter and fuzzier line represents the ghost image from the other side. When these lines are observed several millimeters above the floor of the antrum, the posterior maxilla has good surgical access for a lateral-wall approach for an SA-3 or SA-4 sinus graft option. When the palate line is near the crest of the edentulous ridge, surgical access is more difficult and the surgical lateral-access wall may even be within the zygomatic process.

The panoramic radiograph is useful for preliminary evaluation of the posterior maxilla. However, it is not the standard radiograph used to evaluate the maxillary sinus for abnormalities or pathologic conditions. Ghost and overlapping images will often obscure or distort the anatomy of the maxillary sinus. If the patient is positioned upward with respect to the Frankfort horizontal plane, then the hard palate and the shadow of the occipital bone will obscure the sinus. Another common positioning mistake is not placing the tongue in the roof of the mouth. This will result in radiolucency over the maxilla that represents the palatoglossal air space, as well as a radiopaque region from the tongue. This radiopaque region may be misinterpreted as a pathologic condition. The posterior wall of the maxillary sinus is often misinterpreted as the panoramic innominate line. This artifact is a thin, vertical radiopaque line in the posterior one third of the maxillary sinus that corresponds to the superimposition of the zygomatic process of the maxilla and the frontal process of the zygoma. The posterior region behind this innominate line is often more radiopaque and may be misinterpreted as available bone "behind the sinus" for endosteal implants (Figure 38-14).[72]

The panoramic radiograph may be used as the only radiographic tool in an SA-1 treatment option. Because the sinus and/or floor is not manipulated during the surgery, the condition of the sinus proper is much less

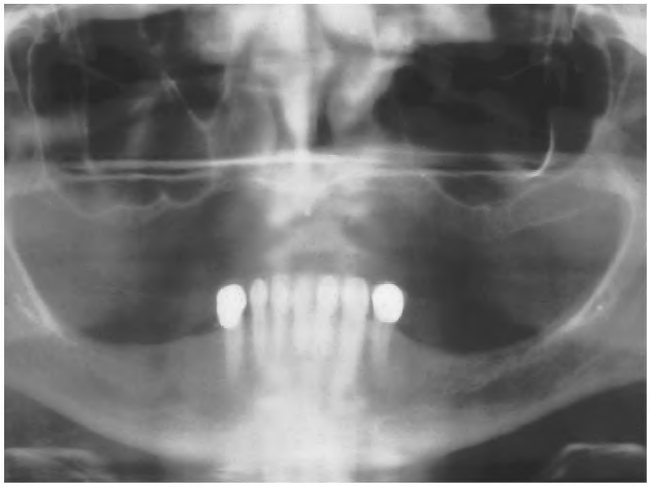


Figure 38-13 A panoramic radiograph is often the first radiograph used in the treatment of an edentulous patient. It is obvious that the patient's right side (*left side of the picture*) does not have adequate bone below the antrum for an endosteal implant. The left side of the patient is less obvious, because magnification and distortion do not permit exact measurements. The two radiopaque horizontal lines of the palate are above the sinus floor and indicate the posterior maxillary access to the lateral wall for a sinus graft is adequate for surgical access.

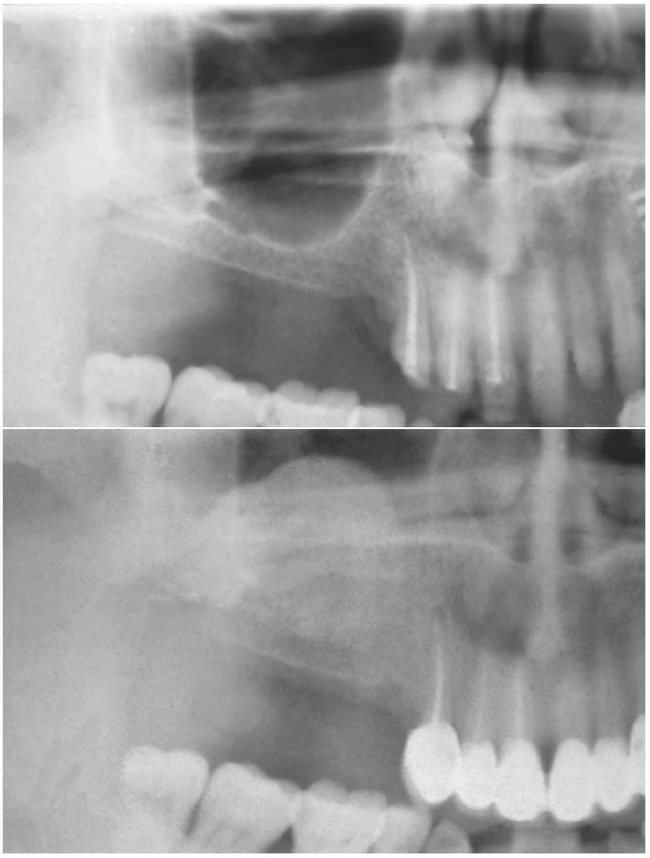


Figure 38-14 **A,** A panoramic radiograph of a posterior maxilla that demonstrates the vertical innominate line. This is not the posterior wall of the maxillary sinus. It is actually the juxtaposition of the zygomatic process and temporal bone lateral to the sinus. The radiopaque region behind this line is not bone. **B,** A sinus graft was performed on this patient. The reader should note that the sinus graft extends posterior to the innominate line on the panoramic image.

WWW.HIGHDENT.IR

relevant to implant insertion. Because the SA-3 or SA-4 sinus graft procedure augments the posterior maxilla, it transforms the region to an SA-1 condition after graft maturity. Therefore after a sinus graft has positively modified the existing bone volume, a panoramic radiograph is often the only radiographic modality used before implant surgery.

Periapical Radiographs

Periapical (PA) radiographs are of little value in the posterior maxilla, because the observed area is small and inadequate. Cysts and inflammation of the membrane can sometimes be seen in association with the apex of the tooth. In addition, certain cases of odontogenic sinusitis may be clearly seen on PA radiographs. Although the PA radiograph can indicate a pathologic condition in the sinuses, it usually does not provide enough information for reliable diagnosis. In general, plain-film PA radiographs of the sinuses are of little value in precise osseous measurements and evaluation of pathologic conditions in the maxillary sinuses.

Computerized Tomography

Currently, no radiographic modality provides more information about the paranasal sinuses than CT. This type of radiography provides much more detailed information about the anatomy and pathologic condition of the sinuses compared with plain films. CT is the best option for viewing the surrounding osseous structures and pathologic condition in the maxillary sinuses.[65,73,74] With the addition of reformatted images, axial, panoramic, cross-sectional, and 3D images provide comprehensive evaluation of the entire sinus cavity and the existing bone below the antrum. In medical evaluation of the sinus cavity, images are taken in the coronal plane. For dental reformatted images, scans are usually taken in numerous transaxial slices (Figure 38-15).

Magnetic Resonance Imaging

MRI is an imaging technique that allows for better differentiation of the soft tissues within the sinuses. MRI has advantages over CT scanning for the evaluation of neoplasms, tumor mapping, and evaluation of fungal sinusitis. However, these images have a high false-positive finding, show very poor bony imaging, and have a higher associated cost. As a consequence, CT reformatted images are superior for implant dentistry.

Ultrasound Evaluation

Mode ultrasound may be used to evaluate for fluid in the maxillary sinus and for mucosal thickening or soft tissue masses in the sinus. Therefore ultrasound can be used for diagnosing acute maxillary sinusitis. However, the diagnostic properties vary greatly and are highly operator dependent. Studies have shown ultrasound sensitivity and specificity to be equal to standard, plain-film radiographs.

Maxillary Sinus: Computed Tomography Radiographic Anatomy

Over the years, preoperative CT scan evaluation has become the standard method for evaluating the pathologic condition of the sinuses before sinus graft procedures. Therefore the practitioner should have an understanding of the CT radiographic anatomy and the pathologic conditions associated with the posterior maxilla and maxillary sinus regions.

Maxillary Sinus Membrane

A CT scan of normal, healthy paranasal sinuses reveal a completely radiolucent (dark) maxillary sinus. Any radiopaque (whitish) area within the sinus cavity is abnormal, and a pathologic condition should be suspected. The normal sinus membrane is radiographically invisible, whereas any inflammation or thickening of this structure will be radiopaque. The density of the diseased tissue or fluid accumulation will be proportional to varying degrees of gray values.

Ostiomeatal Complex

The ostiomeatal unit is composed of the maxillary ostium, ethmoid infundibulum, anterior ethmoid cells, and the frontal recess. The main drainage avenue of the maxillary sinus is through the ostium. The maxillary ostium is bounded superiorly by the ethmoid sinuses and inferiorly by the uncinate process. The uncinate process is a bony knifelike projection that is attached inferiorly to the inferior turbinate and posteriorly has a free margin. Drainage continues through the ostium into the infundibulum, which is a narrow passageway leading into the middle meatus. The middle meatus is the radiolucent space bounded by the middle and inferior turbinates.

Within the nasal cavity, three nasal turbinates or conchae (superior, middle, inferior) exist that are small downward projections of bone. Between the turbinates is a space or recess termed a *meatus*. The respiratory epithelium covers the turbinates and meatus and warms, moistens, and cleans the air that is respirated into the lungs.

The nasal septum is the bony partition that creates a barrier between the right and left sides of the nasal cavity (see Figure 38-11, *A*). Obstructions within any aspect of the nasal system predispose the area to pathologic conditions (see Figure 38-11, *B*).

Maxillary Sinus: Anatomical Variants

Numerous anatomical variants arise that can predispose a patient to postsurgical complications. When these conditions are noted, a pharmacologic discipline may be altered and/or implants may be placed after the sinus graft has matured, rather than predisposing them to an increased risk by inserting them at the same time as the sinus graft. As stated previously, patency of the ostium is paramount to maintain drainage.

WWW.HIGHDENT.IR

همیار دندانسازان

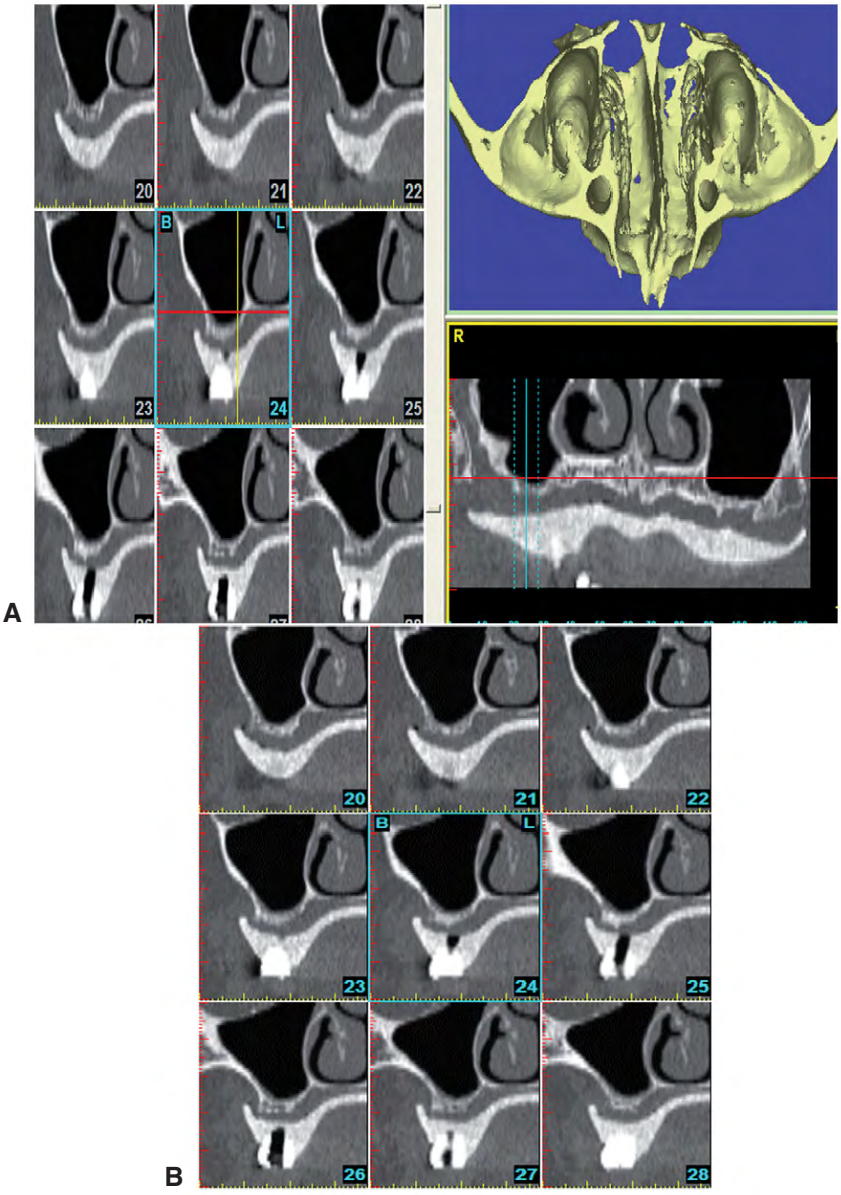


Figure 38-15 **A,** Reformatted transaxial computed tomography images of an edentulous maxilla provide comprehensive evaluation of the sinus cavity and surrounding available bone volumes. A radiopaque template is observed below the bone images and indicates the final teeth position. **B,** Cross-sectional views of a posterior maxilla with a healthy sinus and 1 to 2 mm of bone below the sinus floor. A radiopaque template indicates the soft tissue thickness on the ridge and the ideal tooth position.

Preexisting skeletal and bony abnormalities of the osteomeatal complex may compromise the unit, leading to maxillary sinusitis.

Nasal Septum Deviation

A nasal septum deviation is a very common anatomical variant, occurring in as much as 70% of the population older than 14 years. This bony variant in extremes may cause obstruction of the ostiomeatal unit, which results in inflammation from air turbulence, causing increased mucosal drying and particle deposition. If the deviation is long standing, then atrophy of the middle turbinate may occur, resulting in narrowing of the ostiomeatal complex (Figure 38-16).[75]

Timmenga et al.[76] evaluated 45 patients who received 85 sinus grafts with endoscopy postsurgery. Of the 45 patients, five were found to have sinusitis postsurgery; all five of those patients had a nasal deviation or oversized turbinate. Therefore when these conditions are observed, the implant should not be placed at the same time as the sinus graft, and the recommended preoperative and postoperative pharmacologic protocol is especially warranted.

Middle Turbinate Variants

The middle turbinate plays a significant role in proper drainage of the maxillary sinus. A *concha bullosa* is a pneumatization within the middle turbinate and may

WWW.HIGHDENT.IR

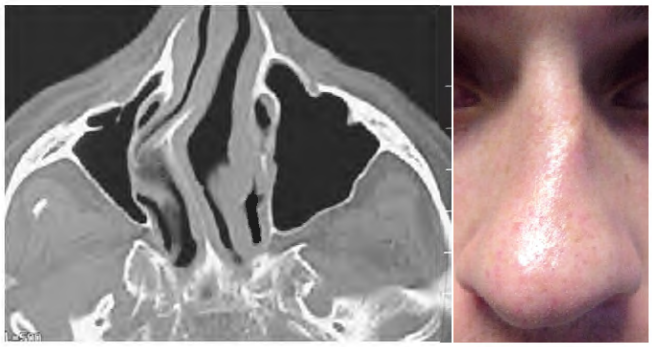


Figure 38-16 A nasal septum deviation is a common variant. Extreme cases may obstruct the ostiomeatal unit and increase the risk of sinusitis after a sinus graft.

occlude the osteomeatal complex, thus compromising adequate drainage. This variant is seen in approximately 4% to 15% of the population (see Figure 38-11).[77] Another variant in this anatomical structure is a paradoxically curved middle turbinate, which presents a concavity toward the septum, thus decreasing the size of the meatus. This also predisposes the patient to sinus disease.

Uncinate Process Variants

A deflected uncinate process (either laterally or medially) can narrow the ethmoid infundibulum, thus affecting the osteomeatal complex. Perforations may also be present within the uncinate process, leading to communication between the nasal cavity and ethmoid infundibulum. In addition, the uncinate process may be pneumatized. Although this is rare, it may compromise adequate clearance and drainage.

Supplemental Ostia

A supplemental ostium or secondary ostia may occur between the maxillary sinus and the middle meatus, which is often found in the posterior fontanelles (PFs). This may be found in approximately 18% to 30% of individuals. Because these secondary openings are usually located posterior and inferior to the natural ostium, they may predispose the patient to sinusitis by the recirculation of infected secretions from the primary meatus back into the sinus cavity. On occasion, these secondary ostia may be encountered during the elevation of the medial wall of the antrum, before placement of the sinus graft. When observed, a piece of collagen is placed over the site to prevent graft material from entering the nasal cavity.

Maxillary Hypoplasia

Hypoplasia of the maxillary sinus may be a direct result from trauma, infection, surgical intervention, or irradiation to the maxilla during the development of the maxillary bone. These conditions interrupt the maxillary growth center, thus producing a smaller-than-normal maxilla. A malformed and positioned uncinate process is associated with this disorder, leading to chronic sinus drainage problems. Most often, these patients have adequate bone height for endosteal implant placement, and a sinus graft is not required to gain vertical height.

Inferior Turbinate and Meatus Pneumatization (Big-Nose Variant)

Misch has observed, on rare occasion, the inferior third of the nasal cavity pneumatizes within the maxilla and resides over the alveolar residual ridge. An evaluation of 550 CT scans of complete or partially edentulous maxillae found this condition in 18 patients (3% incidence). When the patient has this condition, the maxillary sinus is lateral to the edentulous ridge. When inadequate bone height is present below this structure, a sinus graft does not increase available bone height for an implant. This condition is difficult to observe on a panoramic radiograph. If unaware, then the implant can be placed into the nasal cavity above the residual ridge and even penetrate the inferior turbinate. A sinus graft is contraindicated with this patient condition, because the sinus is lateral to the position of the implants. Instead, an onlay graft is required to increase bone height.

MAXILLARY SINUS PATHOLOGY

A preexisting, pathologic, maxillary sinus condition is a contraindication for many procedures that alter the sinus floor before or in conjunction with sinus grafting and/or implant insertion. The risk of postoperative infection is elevated and may compromise the health of the implant and the patient. Therefore pathologic conditions, either preoperative or postoperative, of a maxillary sinus should be evaluated, diagnosed, and treated.

Pathologic conditions of the maxillary sinus may be divided into four categories: (1) inflammation, (2) cystic conditions, (3) neoplasms, and (4) antroliths and foreign bodies. Studies have shown that 20% to 45% of the asymptomatic population has a subclinical pathologic condition in the maxillary sinus. A study completed at the Misch International Implant Institute evaluated 350 consecutive prospective candidates for maxillary sinus augmentations. The results concluded 30.7% of asymptomatic patients had maxillary sinus pathologic condition on CT scan evaluation. Because of this high incidence, it is highly recommended that a thorough radiographic evaluation be completed on all prospective sinus elevation patients.

INFLAMMATION

Inflammatory conditions can affect the maxillary sinus from odontogenic and nonodontogenic causes.

WWW.HIGHDENT.IR

همیار دندانسازان

Odontogenic Sinusitis (Periapical Mucositis)

The close proximity of the roots of the maxillary posterior teeth to the floor of the sinus means that any inflammatory changes in the periodontium or surrounding alveolar bone may cause pathologic conditions in the maxillary sinus.

Etiology

Odontogenic sinusitis is caused by a periapical abscess, cyst, granuloma, or periodontal disease that causes an expansile lesion within the floor of the sinus. Other causes include sinus perforations during extractions and foreign bodies (e.g., gutta-percha, root tips, amalgam). Odontogenic sinusitis is often polymicrobial, with anaerobic streptococci, *Bacteroides* spp., *Proteus* spp., and coliform bacilli being involved. Studies have reported that approximately 25% of chronic sinusitis cases may have some odontogenic origin when teeth are present in the posterior maxilla.[78,79]

Radiographic Appearance

Periodontitis may produce generalized sinus mucosal hyperplasia, which is seen as a radiopaque band that follows the contours of the sinus floor. A localized periapical mucositis reveals a thickening of the mucosal membrane adjacent to the offending tooth and, on occasion, that perforates the floor of the sinus. This radiographic appearance has been termed a *halo effect*. In the author's opinion, smoking may be the influencing factor, causing an increased risk of periodontitis, a decrease in immune response, and a decrease in the healthy flow of the mucus within the antrum.

Differential Diagnosis

This condition may be confused with acute sinusitis or mild mucosal thickening. However, in odontogenic sinusitis, the patient has teeth in the posterior maxilla and will usually exhibit symptoms related to the teeth (i.e., pain from a posterior tooth or a recent extraction, exudate around the existing natural posterior teeth).

Treatment

Before sinus augmentation or implant placement, the tooth or teeth involved should be treated periodontally, endodontically, or extracted. After intraoral soft tissue healing and resolution of the pathologic condition, the sinus graft procedure may be performed with little risk of postoperative complications.

Acute Rhinosinusitis

A nonodontogenic pathologic condition may also result in inflammation in the form of sinusitis. The most common type of sinusitis is acute rhinosinusitis. The signs and symptoms of acute rhinosinusitis are rather nonspecific, making it difficult to differentiate from the common cold, influenza type of symptoms, and allergic rhinitis. However, the most common symptoms include purulent nasal discharge, facial pain and tenderness, nasal congestion, and possible fever.

Acute maxillary rhinosinusitis results in 22 to 25 million patient visits to a physician in the United States each year, with a direct or indirect cost of 6 billion U.S. dollars. Although four paranasal sinuses exist in the skull, the most common involved in sinusitis are the maxillary and frontal sinuses.[70]

Etiology

An inflammatory process that extends from the nasal cavity after a viral upper respiratory infection often causes acute maxillary sinusitis. Microbiological cultures have shown the most common pathogens causing acute rhinosinusitis are *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*. These pathogens include approximately 20% to 27% β-lactamase–resistant bacteria. *Staphylococcus aureus* has also been cited with the microbiology of acute rhinosinusitis. However, this pathogen is usually only seen in nosocomial (hospital induced) sinusitis and is unlikely to be seen in an elective sinus graft patient.

The most important factor in the pathogenesis of acute rhinosinusitis is the patency of the ostium.[80,82] Local predisposing causes of sinusitis include inflammation and edema associated with a viral upper respiratory tract infection or allergic rhinitis. As a consequence, mucous production within the sinus may be abnormal in quality or quantity, along with a compromised mucociliary transport. In an occluded sinus, an accumulation of inflammatory cells, bacteria, and mucus exists. Phagocytosis of the bacteria is impaired with immunoglobulin (Ig)-dependent activities decreased by the low concentration of IgA, IgG, and IgM found in infected secretions.

The oxygen tension inside the maxillary sinus has significant effects on pathologic conditions. When the oxygen tension in the sinus is altered, resultant sinusitis occurs. Growth of anaerobic and facultative organisms proliferate in this environment.[83] Many factors may alter the normal oxygen tension within the sinuses. A direct correlation exists between the ostium size and the oxygen tension in the sinus. In patients with recurrent episodes of sinusitis, oxygen tension is often reduced, even when infection is not present. As a consequence, a history of recurrent acute rhinosinusitis is relevant to determine whether an implant may be at increased risk when inserted at the same time as the sinus graft.

Radiographic Appearance

The radiographic hallmark in acute rhinosinusitis is the appearance of an air-fluid level. A line of demarcation will be present between the fluid and the air within the maxillary sinus. If the patient is supine, then the fluid will accumulate in the posterior area; if the patient is upright during the imaging, the fluid will be seen on the

WWW.HIGHDENT.IR

floor and horizontal in nature. Additional radiographic signs include smooth, thickened mucosa of the sinus, with possible opacification. In severe cases the sinus may fill completely with supportive exudates, which gives the appearance of a completely opacified sinus. With these characteristics, the terms *pyocele* and *empyema* have been applied.

Treatment

Because acute rhinosinusitis is one of the most common health problems today, patients having sinus augmentation procedures should be well screened for a past history and current symptoms. Even though acute rhinosinusitis is a self-limiting disease, a symptomatic patient should be treated and cleared by the physician before grafting procedures. These patients are also more prone to postoperative sinusitis. As a result, a sinus graft is performed and allowed to mature several months before the placement of the implant. In addition, the suggested antibiotic coverage may be altered and extended, both before and after the sinus graft procedure.

Chronic Rhinosinusitis

Chronic rhinosinusitis is a term used for a sinusitis that does not resolve in 6 weeks and also has recurrent episodes. It is the most common chronic disease in the United States, affecting approximately 37 million people. Symptoms of chronic sinusitis are associated with periodic episodes of purulent nasal discharge, nasal congestion, and facial pain.

Etiology

As maxillary rhinosinusitis progresses from acute to chronic, anaerobic bacteria become the predominant pathogens. The microbiology of chronic sinusitis is very difficult to determine because of the inability to achieve accurate cultures. Studies have shown that possible bacteria include *Bacteroides* spp., anaerobic gram-positive cocci, *Fusobacterium* spp., as well as aerobic organisms (*Streptococci* spp., *Haemophilus* spp., *Staphylococcus* spp.).[84] A recent Mayo Clinic study showed that in 96% of patients with chronic sinusitis, active fungal growth was also present.[85]

Radiographic Appearance

Chronic rhinosinusitis may appear radiographically as thickened sinus mucosa, complete opacification of the antrum, and/or sclerotic changes in the sinus walls (which give the appearance of denser cortical bone in the lateral walls).

Treatment

Medical evaluation and clearance by an experienced physician in sinus pathology is highly recommended for patients with chronic maxillary rhinosinusitis before sinus grafting, because significant bacterial resistance and fungal growth is highly probable. Fungal infections may be difficult to treat and control, and serious complications may result in postoperative sinus graft patients. In this manner these patients are evaluated before surgery by the physician most likely to treat them after the sinus graft surgery, should significant complications occur.

Allergic Sinusitis

Etiology

Allergic sinusitis is a local response within the sinus caused by an irritating allergen in the upper respiratory tract. Therefore this allergen may be a cause of acute or chronic rhinosinusitis. This category of sinusitis may be the most common form, with 15% to 56% of patients undergoing endoscopy for sinusitis showing evidence of allergy. This condition often leads to chronic sinusitis in 15% to 60% of patients.[86] The sinus mucosa becomes irregular or lobulated, with resultant polyp formation.

Radiographic Appearance

Polyp formation related to allergic sinusitis is usually characterized by multiple, smooth, rounded, radiopaque shadows on the walls of the maxillary sinus. Most commonly, these polyps are located near the ostium and are easily observed on a CT scan. In advanced cases, ostium occlusion, along with displacement or destruction of the sinus walls, may be present, with a radiographic image of a completely opacified sinus.

Treatment

Special attention must be given to a patent ostium, bacterial resistance, and close postoperative supervision. The polyp, if enlarged, may be removed before the sinus graft. This may be performed through an anterior Caldwell-Luc approach or by an endoscopic procedure through the ostium.

Allergic sinusitis patients often have a greater risk of complications related to an increase in allergen production. Because sinus grafting is an elective procedure, the time of year for the surgery may be altered to decrease the postoperative infection risk. For example, if hay fever or a grass allergy is related to the patient's sinusitis, then the sinus graft surgery should be performed in the season or seasons that have least risk to aggravate the sinus mucosa (i.e., winter or fall).

Fungal Sinusitis (Eosinophilic Fungal Rhinosinusitis)

Granulomatous sinusitis is a very serious (and often overlooked) disorder within the maxillary sinus. Patients who have fungal sinusitis are thought to have had an extensive history of antibiotic use, chronic exposure to mold or fungus in the environment, or being immunocompromised.

WWW.HIGHDENT.IR

همیار دندانسازان

Etiology

Fungal infections are usually caused by aspergillosis, mucormycosis, or histoplasmosis.[87] Chronic sinusitis patients should always be evaluated for granulomatous conditions, because a high percentage of fungal growth exists in this patient population. Of concern in these patients is that eosinophils are activated that release major basic protein (MBP) into the mucus, which attacks and destroys the fungus. However, this results in the membrane being irritated and possibly irreversibly damaged, which allows bacteria to proliferate. Three possible clinical signs may differentiate fungal sinusitis from acute or chronic sinusitis; however, a positive diagnosis require mycological and histological studies.[87]

1. No response to antibiotic therapy
2. Soft tissue changes in sinus associated with thickened reactive bone, with localized areas of osteomyelitis
3. Association of inflammatory sinus disease that involves the nasal fossa and facial soft tissue

Radiographic Appearance

Granulomatous sinusitis may appear radiographically as mild thickening to complete opacification of the sinus.

Treatment

Patients with a history or current knowledge of fungal sinusitis should be referred to their physician or otolaryngologist for treatment and surgical clearance. Treatment usually involves debridement and therapy with an antifungal agent, such as amphotericin B.

CYSTIC LESIONS

Cystic lesions are a common occurrence in the maxillary sinus, and studies have reported a prevalence range of 2.6% to 20%.[88] They may vary from microscopic lesions to large, destructive, expansile pathologic conditions. They include pseudocysts, retention cysts, primary mucoceles, and postoperative maxillary cysts.

Pseudocysts (Mucous Retention Cyst)

The most common cysts in the maxillary sinus are mucous retention cysts. After much controversy, in 1984, Gardner[89] distinguished these cysts into two categories: (1) pseudocysts and (2) retention cysts. Pseudocysts are more common and of much greater concern during sinus graft surgery, compared with retention cysts. Pseudocysts reoccur in approximately 30% of patients and are often unassociated with sinus symptoms. As a consequence, many physicians do not treat this condition. However, when their size is larger than 10 mm in diameter, pseudocysts may occlude the maxillary ostium during a sinus graft procedure and increase the risk of postoperative infections.

Etiology

A pseudocyst is caused by an accumulation of fluid beneath the periosteum of the sinus mucosa. This elevates the mucosa away from the floor of the sinus, giving rise to a dome-shaped lesion. Pseudocysts have also been termed *mucosal cysts*, *serous cysts*, and *nonsecreting cysts*. Pseudocysts are not true cysts because they lack an epithelial lining. The cause of the fluid is from bacterial toxins from the sinus mucosa or from odontogenic causes (see Figure 38-16).

Radiographic Appearance

Pseudocysts are depicted radiographically as smooth, homogenous, dome-shaped, round to ovoid, well-defined radiopacities. Pseudocysts do not have a corticated (radiopaque) marginal perimeter and are usually on the floor of the sinus cavity.

Treatment

Pseudocysts are not a contraindication for sinus graft surgery, unless their approximate size is greater than 10 mm in diameter. However, when in doubt, an evaluation of even smaller "cysts" by an otorhinolaryngologist or ear, nose, and throat (ENT) physician may still be to confirm the diagnosis. If a large pseudocyst is present, then the elevation of the membrane during a sinus graft may raise the cyst to occlude the ostium. In addition, on elevation or placement of the grafting material, the cyst may be perforated, allowing fluid within the cyst to contaminate the graft. Large cysts of this nature should be drained and allowed to heal before or in conjunction with sinus elevation surgery. Most often, an otolaryngologist or ENT physician should evaluate and treat this condition before the sinus graft. If a pseudocyst is less than 10 mm, then less concern is needed and the fluid may be drained in conjunction with sinus grafting, depending on the surgeon's experience in the treatment of this condition. This should be accomplished with care to prevent membrane perforation. A recall evaluation of this area during the follow-up period of the sinus graft surgery is in order, because reoccurrence of pseudocysts are common.

Retention Cysts

Retention cysts may be located on the sinus floor, near the ostium, or within antral polyps. Because they contain an epithelial lining, researchers consider them to be mucous secretory cysts and "true" cysts. Retention cysts are often microscopic in size.

Etiology

Retention cysts result from partial blockage of seromucinous gland ducts located within the connective

WWW.HIGHDENT.IR

tissue underlying the sinus epithelium. As the secretions collect, they expand the duct, producing a cyst that is encompassed by respiratory or cuboidal epithelium. They may be caused by sinus infections, allergies, or odontogenic reasons.

Radiographic Appearance

Retention cysts are usually very small and not seen clinically or radiographically. In rare instances, they may achieve adequate size to be seen in a CT image and may resemble the appearance of a small pseudocyst.

Treatment

No treatment for retention cysts exist before or in conjunction with a sinus graft and/or implant insertion.

Primary Maxillary Sinus Mucocele

A primary mucocele is a cystic, expansile, destructive lesion that may include painful swelling of the cheek, displacement of teeth, nasal obstruction, and possible ocular symptoms.[90]

Etiology

The primary mucocele arises from blockage of the maxillary ostium by fibrous connective tissue. Because of the compromised drainage, the mucosa expands and herniates through the antral walls. This mucocele is classified as a *cyst* because it is lined by antral epithelium, which contains mucin.

Radiographic Appearance

In the early stages, the primary mucocele involves the entire sinus and appears as an opacified sinus. As the cyst enlarges, the walls become thin and eventually perforate. In the late stages, destruction of one or more surrounding sinus walls is evident.

Treatment

Surgical removal of this cyst is indicated before any bone augmentation procedures.

Postoperative Maxillary Cyst

A postoperative maxillary cyst of the maxillary sinus is a cystic lesion that usually develops secondary to a previous trauma or surgical procedure in the sinus cavity. It has also been termed a *surgical ciliated cyst*, *postoperative maxillary sinus mucocele*, or a *secondary mucocele*.[91-93]

Etiology

A postoperative maxillary cyst is a direct result of trauma or past history of surgery within the maxillary sinus. The cyst is derived from the antral epithelium and mucosal remnants that previously were entrapped within the prior surgical site. This separated mucosa results in an epithelium-lined cavity in which mucin is secreted. The antrum becomes divided by a fibrous septum in which one part drains normally, whereas the other part is composed of the mucocele. It is relatively rare in the United States; however, it constitutes approximately 24% of all cysts in Japan. At least three reported cases exist of a postoperative maxillary cyst forming after a sinus graft procedure, including one by the author of this chapter.

Radiographic Appearance

The cyst radiographically presents as a well-defined radiolucency circumscribed by sclerosis. The lesion is usually spherical in the early stages, with no bone destruction. As it progresses, the sinus wall becomes thin and eventually perforates. In later stages, it will appear as two separated anatomical compartments.

Treatment

Surgical ciliated cysts should be enucleated before any bone augmentation procedures. If observed after the sinus graft, then they should be enucleated and regrafted in the site.

NEOPLASMS

Etiology

Primary malignant tumors within the maxillary sinus are usually caused by squamous cell carcinomas or adenocarcinomas. Signs and symptoms of malignant disease are related to the surrounding sinus wall that the tumor invades and includes swelling in the cheek area, pain, anesthesia or paresthesia of the infraorbital nerve (e.g., anterior wall), and visual disturbances (e.g., superior wall). These tumors in the sinus are usually nonspecific and give a variety of consequences, including opacified sinuses, soft tissue masses in the sinus, as well as sclerosis, erosion, or destruction of the walls of the sinus. Sixty percent of squamous cell carcinomas of the paranasal sinuses are located in the maxillary sinus, usually in the lower one half of the antrum. Clinical signs in the oral cavity reflect the expansion of the tumor and an increased mobility of the involved teeth. Invasion of the infratemporal fossa is also possible.[70,94]

Radiographic Appearance

Radiographic signs of neoplasms may include various-sized radiopaque masses, complete opacification, or bony wall changes. A lack of a posterior wall on a panoramic radiograph should be a sign of possible neoplasm (Figure 38-17).

Treatment

Any signs or symptoms of a lesion of this type should be immediately referred for medical consultation. Of course, sinus grafts are contraindicated while this condition exists.

WWW.HIGHDENT.IR

همیار دندانسازان

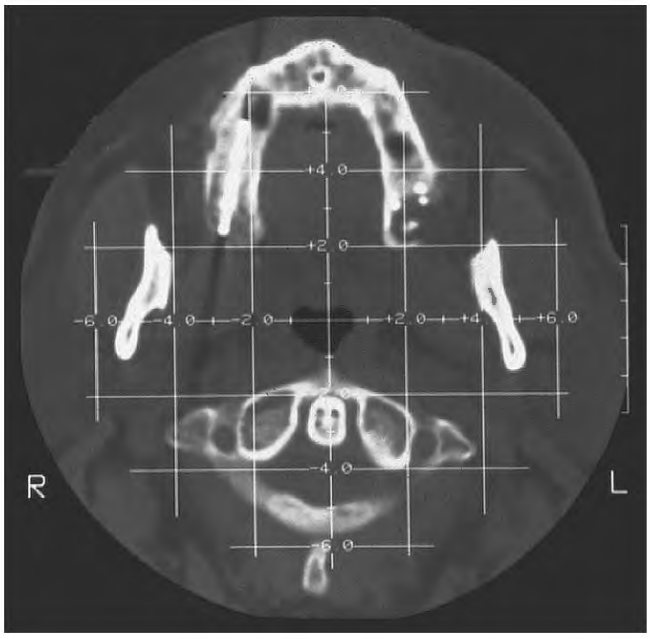


Figure 38-17 A computed tomography scan of a patient with a sinus graft and endosteal implant (blade) on the left. The right posterior maxilla has no bony wall, and the posterior sinus is radiopaque. A keratocyst was found on biopsy.

ANTROLITHS AND FOREIGN BODIES

Maxillary sinus antroliths are the result of complete or partial encrustation of a foreign body. These masses found within the maxillary sinus originate from a central nidus, which can be endogenous or exogenous.[95]

Etiology

The majority of endogenous sources are from dental origin, including retained roots, root canal sealer, fractured dental instruments, and dental implants. Additionally, bone spicules, blood, and mucus have been reported to cause antroliths.[96] Reports in the literature of exogenous sources include paper, cigarettes, snuff, and glue.[97] Although most antroliths are asymptomatic, they often are associated with sinusitis.

Radiographic Appearance

The radiographic appearance of a maxillary antrolith resembles either the central nidus (retained root) or appears as a radiopaque, calcified mass within the maxillary sinus (Figure 38-18).

Differential Diagnosis

Because the calcified antrolith is composed of calcium phosphate ($CaPO_4$), calcium carbonate salts, water, and organic material, it will be considerably more radiopaque than an inflammatory or cystic lesion.[98] The central nidus of the antrolith is similar to its usual radiographic appearance.

Treatment

Before sinus augmentation and implant placement, the antrolith should be surgically removed. If sinusitis exists, then the sinus cavity should be allowed to heal completely before sinus augmentation procedures. A nonsymptomatic condition may have the antrolith removed, sinus membrane opening sealed, and sinus graft performed at the same surgery.

CONSIDERATIONS SPECIFIC TO SINUS GRAFTS

The general contraindications for implant surgery also apply to sinus graft procedures. In addition, specific, local conditions exist that may increase morbidity, render the procedure less predictable, and/or place the patient more at risk.

Several conditions related to the maxillary sinus are a concern but are not necessarily contraindications to the sinus graft procedure. However, they should be treated before or during the sinus graft surgery. For example, root tips in the antrum, pseudocysts, an oral antral opening, extraction of hopeless teeth in the surgical site, and unerupted teeth all may be conditions that may be performed in conjunction with the sinus graft. However, less experienced clinicians should treat these conditions several months before the sinus graft surgery to improve the surgical site before the bone graft procedure. In addition, relative contraindications include patients with a narrowing of the osteomeatal complex (e.g., deviated septum, abnormal large size of middle turbinate [concha bullosa], enlargement of an air cell in the roof of the sinus [Haller cell]). An ENT consultation is warranted for these conditions, and the sinus graft surgery should be separate from implant placement surgery because a higher risk of postoperative infection exists.

Smoking

Among the factors that may negatively interfere with the final outcome of sinus grafting procedures, smoking may play a relevant role. Overall, smokers have a 7% greater failure rate in the posterior maxilla than nonsmokers.[99] Smoking is known to be associated with an increased susceptibility to allergy and infections, because it interferes with ciliary function and secretory immunity of the nasorespiratory tract. In the maxillary sinus, this may have effects on both immune exclusion and suppression, because IgA and IgM responses are reduced, whereas IgE responses are increased. Smoking is believed to disturb bone graft healing because it reduces local blood flow by increasing peripheral resistance and causing an increased platelet aggregation.[35,100] By-product chemicals of smoking, such as hydrogen cyanide and carbon monoxide, have been shown to

WWW.HIGHDENT.IR

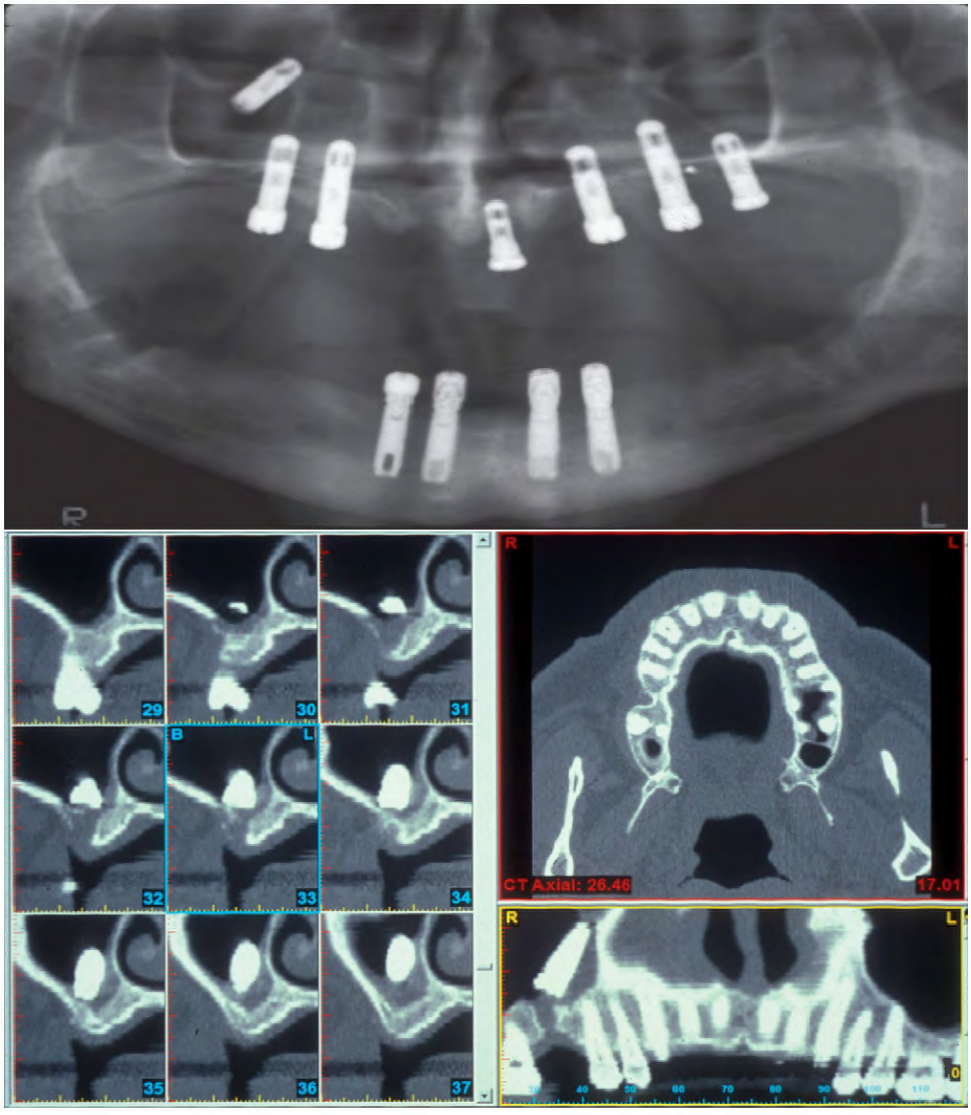


Figure 38-18 **A,** A panoramic radiograph of a patient with a history of implant stage I and stage II surgery. The patient was unaware of the implant in the right maxillary sinus (*left side of the picture*). **B,** A computed tomography scan of a patient with a sinus lift of 6 mm within the implant osteotomy and immediate implant insertion. The implant became mobile and was displaced into the sinus.

inhibit wound healing, as does nicotine, which inhibits cellular proliferation. Tobacco may interfere directly with osteoblastic function, and strong evidence exists of decreased bone formation in smokers. In addition, smokers have a significant reduction of bone mineral content. Bone mineral density can be reduced two to six times in a chronic smoker. Overall, smoking may contribute to poor available bone quality and poor healing capacity resulting from vascular and osteoblastic dysfunction.[35,100]

Although smokers have several concerns related to sinus grafting, most of these patients may be treated with sinus grafting procedures.[36,101] In a study by Dietsh-Misch et al., 50 consecutive sinus grafts were performed on 31 patients ranging from 21 to 68 years of age.[102] One third of these patients (10 patients and 15 sinus grafts) were nonsmokers, and two thirds (21 patients and 35 sinuses) smoked at least 20 cigarettes each day. The sinus graft procedures observed one perforation of sinus mucosa in nonsmokers and three perforations in smokers. Postoperative infections were zero for nonsmokers, and two sinuses became infected within 2 months of the sinus graft in smokers. However, all 50 posterior maxillae grew adequate bone height and received implants (116 implants), and all were successful. After 6 years of follow-up, zero implants and zero prostheses were lost. Therefore it appears cigarette smoking is not a strict contraindication to sinus grafting.

However, this is not to say smokers have no risk. Smoking may represent a relative contraindication because of the risk of wound dehiscence, graft infection and/or resorption, and a reduced probability of osseointegration. It is recommended, however, that if a decision to proceed with surgery has been made, then

WWW.HIGHDENT.IR

همیار دندانسازان

patients refrain from smoking at least 15 days before surgery (the time it takes for nicotine to clear systemically) and 4 to 6 weeks after surgery. Moreover, smokers should sign a detailed informed-consent form in which risks connected to smoking are clearly explained.

Chronic Maxillary Rhinosinusitis

Patients that have chronic rhinosinusitis and have a history of long-term antibiotic use are a relative contraindication for sinus graft surgery. These patients have altered sinus flora and are at greater risk of containing fungi that can lead to severe fungal-related infections. Patients at a higher risk should have a thorough preoperative examination with a CT evaluation and possible medical clearance. Furthermore, the sinus graft procedure should be performed before and separate from the implant placement surgery. In addition, when a patient is more at risk of a sinus infection during a specific time period (e.g., grass allergy in the spring), it is prudent to perform the sinus graft procedure at the time of the year with the least risk. In addition, pre- and postoperative antibiotic drugs may be extended to cover these patients around the sinus graft procedure.

In addition to relative contraindications for sinus grafting, absolute local contraindications to sinus grafting include the following (Box 38-2):

1. The patient has an active sinus infection on the day of surgery. (The patient should blow the nose, and the physician should evaluate the sample for color and thickness of mucus.)
2. The patient has a significant recurrent history of chronic sinusitis despite a history of maxillary sinus corrective surgery.
3. The patient has a significant recurrent history of fungal sinusitis.
4. The patient has uncontrolled, late-stage diabetes. (These patients are very susceptible to fungal infections.)
5. The patient has cystic fibrosis (CF), a genetic disease with a 92% to 100% chronic sinusitis rate. (In addition, patients with CF exhibit significant rates of polyp formation and fungal infections.)
6. The patient has maxillary sinus hypoplasia (MSH). (The sinus drainage system is chronically compromised and is associated with a malformed uncinate process. If implants are to be placed in patients with MSH, then the sinus cavity should not be used and SA-1 procedures are recommended.)
7. The patient has neoplasms (i.e., primary or secondary malignant tumors within the maxillary sinus).
8. The patient has inferior turbinate and/or meatus pneumatization (i.e., big nose variant).

Box 38-2 Absolute Contraindications to Sinus Grafting

- Active sinus infection day of surgery
- Significant recurrent history of chronic sinusitis
- Significant recurrent history of fungal sinusitis
- Uncontrolled, late-stage diabetes
- Cystic fibrosis (CF)
- Maxillary sinus hypoplasia (MSH)
- Neoplasms
- Inferior turbinate and/or meatus pneumatization (big-nose variant)

SURGICAL ENVIRONMENT

In a previous chapter the difference between an aseptic and "clean" surgery is addressed. Although studies have reported little (if any) difference between the two approaches for the placement of implants in existing bone volumes, care should be taken related to the surgical environment for a sinus graft.

A sinus graft surgery has a higher risk of infection than implant insertion surgery, because the maxillary sinus is often predisposed to this complication. In fact, a patient may obtain a sinus infection after the surgery that is unrelated to the sinus graft operation. However, the surgical manipulation of the antral mucosa causes an increased release of histamine, which increases the thickness of the sinus mucosa and therefore increases the risk that the osteomeatal unit will become compromised. As such, a surgical environment that includes intraoral and extraoral scrubbing with chlorhexidine, scrubbing and draping the patient, as well as gowning the doctor and assistant should be considered in addition to sterile gloves and sterile instruments. In addition, a filter to circulate and clean the air of the operating room may be advantageous to decrease the risk of allergens. The risk of postoperative sinus infection is generally less than 5% when these procedures and a preoperative and postoperative pharmacologic regimen are used.[25,103,104] However, postoperative infection occurrence has been reported to be more than 20% in conditions more prone to contribute to sinus infection.[7,105-108]

PREMEDICATIONS

Antimicrobial Medications

The risk of bacterial contamination during and after sinus graft procedures are much different than regular implant surgical procedures. Therefore the pharmacologic protocol should be effective against the organisms in this surgical site. The pharmacologic regimen includes an antibiotic drug, anti-inflammatory medications, antimicrobial rinse, and analgesic medications.

Compared with routine dental implant surgery, sinus augmentation has a greater chance of morbidity because of the possible additional routes of infection.

WWW.HIGHDENT.IR

Bacterial invasion may originate from different sources: (1) transoral surgery, (2) bone graft material, and (3) bacteria from the sinus cavity. Additionally, it has been documented that the inclusion of foreign bodies (e.g., implants, alloplasts, allografts) increases infection rates.[109,110] Because a greater chance of infection and morbidity exists with this type of surgical procedure, a strict antibiotic protocol is of benefit. Antibiotic medications have been shown to significantly reduce the number of sinus graft or implant failures caused by infection.[111]

Following the principles of prophylactic antibiotic administration, the antibiotic should be effective against the bacteria most likely to cause infection. The most likely contaminating organisms after transoral surgery are primarily streptococci, anaerobic gram-positive cocci, and anaerobic gram-negative rods. *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis* are the three most common pathogens found with acute sinus infections.[109,112] *Staphylococcus aureus* along with anaerobic bacteria have a significant role in causing chronic rhinosinusitis disease. The organisms associated with infection of allografts such as demineralized bone in oral surgery include α-hemolytic streptococci and *S. viridans*.[113,114] *S. aureus*, *Bacteroides* spp., and endogenous bacteria cause the vast majority of postoperative infections. Therefore a pharmacologic protocol effective against these organisms is appropriate.

When evaluating various classes of antibiotic medications used for treatment of maxillary sinus infections, the antibiotic class of choice is the β-lactam antibiotic drugs. However, bacterial resistance has become a significant problem in the treatment of these pathogens. With the wide range of possible routes of bacterial invasion and types of bacteria, the antibiotic drug must be broad spectrum to account for all these possibilities. Bacterial resistance is initiated by two common mechanisms: (1) production of antibiotic-inactivating enzymes (*S. aureus, H. influenzae*, and *M. catarrhalis*) and (2) alteration in target site *(S. pneumoniae)*. Studies have shown the following resistance results[115]:

- *H. influenzae*—36.8%
- *M. catarrhalis*—98%
- *S. pneumoniae*—28.6%

Because of the high rate of bacterial resistance, amoxicillin (the drug of choice for many years) is no longer used for antibiotic prophylaxis for the sinus graft surgery. Instead, amoxicillin-clavulanate (Augmentin) is used, because the addition of clavulanic acid enhances amoxicillin's activity against the β-lactamase–producing strains of bacteria.

The patient with a history of nonanaphylactic allergic reaction to penicillin may take cefuroxime axetil (Ceftin) as an alternative.[116] Ceftin possesses good potency, efficiency, and strong activity against resistant *S. pneumoniae* and *H. influenzae.*

If a patient has a history of anaphylactic reaction to penicillin, recurrent sinus infections, or a recent history of antibiotic use, then levofloxacin (Levaquin) may be indicated. This newer type of quinolone antibiotic exhibits superior activity against most types of involved bacteria, along with resistant strains.

Maximum effectiveness of prophylactic antibiotic drugs occurs when the antibiotic is in adequate concentrations in the tissue before bacterial invasion. Because the sinus mucosa has limited blood supply to combat possible bacterial invasion from the sinus surgery, antibiotic medications should be administered at least 1 full day before surgery and extended for 5 days after surgery. Recommended systemic antibiotic drugs are shown in Box 38-3.

Local Antibiotic Medications

The antibiotic concentration within a blood clot of the sinus graft depends on the systemic blood titer. After the clot stabilizes, further antibiotic drugs do not enter the area until revascularization.[117] The bone graft is a dead space with minimum blood supply and absence of protection by the host's cellular defense mechanisms.

Box 38-3 Recommended Prophylactic Antibiotic Drugs for Sinus Grafting Procedures

Systemic Antibiotic Prophylaxis

No History of Sinus Pathologic Condition/No Antibiotic Use in Last 6 Weeks

1. Augmentin (amoxicillin-clavulanic acid) (825 mg/125 mg)
   One tablet twice a day (bid) starting 1 day before surgery and 5 days after surgery
2. Ceftin (cefuroxime axetil) (500 mg), nonanaphylactic allergy to penicillin
   One tablet bid starting 1 day before surgery and 5 days after surgery

History of Sinus Pathologic Condition/Antibiotic Use in Last 6 Weeks/Anaphylactic Reaction to Penicillin

1. Levofloxacin (Levaquin 500 mg)
   One tablet 1 day before surgery and 5 days after surgery

Antibiotic in Graft

Ancef (1 g)
Dilute with 2 mL Saline (500 mg/mL)
0.2 mL or 100 mg: add to collagen membrane
0.8 mL or 400 mg: add to graft material

Clindamycin 150 mg/1 mL
0.2 mL or 30 mg: add to collagen membrane
0.8 mL or 120 mg: add to graft material

WWW.HIGHDENT.IR

همیار دندانسازان

This leaves the graft prone to infections that would normally be eliminated by either the host defenses or the antibiotic. The osteogenic induction of autografts and allografts is greatly retarded when contaminated with infectious bacteria.[118] To ensure adequate antibiotic levels in a SA graft, it is recommended to add antibiotic to the graft mixture.[69,119,120] This local antibiotic may protect the graft from early contamination and infection. Numerous studies have shown that an antibiotic added to graft material has no deleterious effects on bone growth. Antibiotic drugs such as penicillin, cephalosporin, and clindamycin, even in high concentrations, have not been found to be destructive to bone-inductive proteins.[119-121]

The locally delivered antibiotic should have efficacy against the most likely organisms encountered. Because the incidence of allergy is so high with β-lactam antibiotic drugs, the parenteral form of cefazolin (Ancef) or clindamycin is selected. Orally administered capsules and tablets should not be used within the graft, because they contain fillers that are not conducive to osteogenesis. When the parenteral form of antibiotic is a liquid, the volume of liquid added to the graft should be minimized to allow adequate handling of the graft mixture. (See Box 38-3 for the recommended amount of antibiotic that should be added to sinus graft material).

Clinical experience indicates that less risk of infection exists when preoperative and postoperative antibiotic drugs are used both orally and in the graft. Because infection considerably impairs bone formation for patients undergoing sinus graft procedures, oral antibiotic coverage is continued for 5 days after the surgery.

Oral Antimicrobial Rinse

An additional antimicrobial medication is chlorhexidine gluconate. This category of mouth rinse has been shown to successfully decrease infectious episodes and minimizes postoperative complications from the incision line. Gentle oral rinses of chlorhexidine gluconate 0.12% should be used twice daily for 2 weeks after surgery.

Glucocorticoid Medications

The pharmacodynamics of glucocorticoid medications are discussed in Chapter 21. The decrease in inflammation of the soft tissue decreases postoperative pain, swelling, and incision line opening. In addition, the clinical manifestations of surgery on the sinus mucosa can also be decreased by use of a steroid.[122] Therefore the usual surgical protocol for most implant surgeries, including sinus grafts, includes a short-term dose of dexamethasone (Decadron) (Box 38-4). To ensure patency of the ostium and minimize inflammation in the sinus before surgery, steroid medications are initiated 1 full day before surgery. This medication should also be extended 2 days postoperatively because edema peaks at 2 days.

Decongestant Medications

Sympathomimetic drugs that influence α-adrenergic receptors have been used as therapeutic agents for the decongestion of mucous membranes. Both systemic and topical decongestant medications are useful in reopening a blocked sinus ostium and facilitating drainage. Oxymetazoline 0.05% (Afrin or Vicks Nasal Spray) and phenylephrine 1% are useful topical decongestant medications. The vasoconstrictor action of oxymetazoline lasts approximately 5 to 8 hours, which is preferred in comparison with 1 hour for phenylephrine. However, decongestant drugs have many disadvantages. Topical decongestant drugs can cause a rebound phenomenon and the development of rhinitis medicamentosa if used more than 3 to 4 days. The effectiveness of the topical decongestant is markedly enhanced by proper position of the patient's head during administration of the drug. It should also be noted that the pulse amplitude and blood flow in the sinus mucosa is reduced with decongestant drugs, such as oxymetazoline. This may, in turn, decrease the defense mechanism of the tissues.[123]

Caution is advised in pseudoephedrine administration to patients with high blood pressure, heart disease, diabetes, thyroid disease, and/or prostatic enlargement. The use of this decongestant should be avoided in patients taking antihypertension or antidepressant drugs containing a monoamine oxidase inhibitor because of a potentiation of the action of the sympathomimetic amine. Patients receiving digitalis may experience an increase in ectopic pacemaker activity. Side effects of this systemic decongestant also include nervousness, dizziness, or sleeplessness.

As a consequence of the medical and local risks of decongestant medications, the modified sinus graft pharmacologic protocol extended the use of corticosteroid medications (1 day prior) and discontinued the preoperative or postoperative dose of oxymetazoline. Decongestant medications should only be used for 1 to 3 days if signs and symptoms of postoperative sinusitis are present and the ostiomeatal complex is obtunded from the surrounding mucosa.

Box 38-4 Glucocorticoid Protocol

Dexamethasone (4 mg)

- Two tablets (8 mg) in the morning, the day before surgery
- Two tablets (8 mg) the morning of surgery
- One tablet (4 mg) in the morning, the day after surgery
- One half to one tablet (2 to 4 mg) in the morning, the second day after surgery

WWW.HIGHDENT.IR

Analgesic Medications

Any analgesic combination containing codeine, such as Tylenol 3, is prescribed postoperatively because codeine is a potent antitussive, and coughing may place additional pressure on the sinus membrane and introduce bacteria into the graft. The patient is instructed to cough (if necessary) with the mouth open so that no air pressure through the ostium occurs.

Cryotherapy

With sinus elevation procedures, postoperative inflammation in the posterior maxilla is very common. Because postoperative swelling can adversely affect the incision line, measures should be taken to minimize this condition. Application of cold dressings and cold oral liquids, along with elevation of the head and limited activity for 2 to 3 days, will help minimize the swelling. The applied cold dressing and liquids will cause vasoconstriction of the capillary vessels, thus reducing the flow of blood and lymph, resulting in a lower degree of swelling. Ice or cold dressings should only be used for the first 24 to 48 hours. After 2 to 3 days, heat may be applied to the region to increase blood and lymph flow, to help clear the area of the inflammatory consequences. This also helps reduce any ecchymosis that may have occurred from the tissue reflection.

SURGICAL TECHNIQUE

Preparation and Antisepsis

Because of the extent of tissue reflection, technique sensitivity of the surgery, and need for antisepsis, oral or conscious sedation is recommended for sinus graft procedures. After sedation and adequate infiltration anesthesia (i.e., posterior and middle alveolar nerve, greater palatine nerve) are obtained, the patient is prepared for surgery. Preparation of the surgical site is important in sinus manipulation surgery to reduce contamination by the patient's own normal flora. The mouth cannot become a sterile environment for surgery. However, intraoral preparation before surgery may significantly reduce the bacterial count in the mouth. Studies reveal a significant reduction in bacteremia during extractions and implant surgery complications after preparation with antiseptic mouth rinse.[124,125]

Iodophor compounds (Betadine) are a most effective antiseptic. However, because the iodine is complexed with organic surface-active agents, it has been shown to inhibit the osteoinduction of demineralized bone. Therefore care is taken to avoid contamination of the graft. As a consequence, intraoral preparation of the surgical site requiring a bone graft, a 0.12% chlorhexidine gluconate (Peridex) scrub, and rinse is most often used. Extraoral presurgical scrubbing of the skin should also be performed with chlorhexidine antiseptics.

SINUS SURGERY: HISTORY

In the early 1970s, Tatum[3,4] began to augment the posterior maxilla with autogenous rib bone to produce adequate vertical bone for implant support. He found that onlay grafts below the existing alveolar crest would decrease the posterior intradental height significantly, yet very little bone for endosteal implants would be gained. Therefore in 1974, Tatum developed a modified Caldwell-Luc procedure for SA grafting. The crest of the maxilla was infractured to elevate the maxillary sinus membrane. Autogenous bone was then added in the area previously occupied by the inferior third of the sinus. Endosteal implants were inserted in this grafted bone after approximately 6 months. Implants were then loaded with final prostheses after an additional 6 months.

In 1975, Tatum developed a lateral-approach surgical technique to elevate the sinus membrane and place the implant simultaneously. The implant system used was a one-piece ceramic implant, and a permucosal post was required during the healing period. Early ceramic implants were not designed adequately for this procedure, and results with the technique were unpredictable. In 1981, Tatum[126,127] developed a submerged Ti implant for use in the posterior maxilla and achieved predictable results (Figure 38-19).

From 1974 to 1979, the primary graft material for sinus grafts was autologous bone. In 1980, Tatum[126,127] further expanded the application of the SA augmentation technique with a lateral maxillary approach, with the use of synthetic bone. The same year, Boyne and James[5] first reported on the sinus graft technique using autogenous bone for SA grafts. Most of the publications in the 1980s were anecdotal or based on very small sample sizes.

TREATMENT CLASSIFICATIONS FOR THE POSTERIOR MAXILLA

In 1984, Misch[69] organized a treatment approach to the posterior maxilla based on the amount of bone below the antrum, and in 1986 he expanded the treatment approach to include the available bone width that was related to implant design (see Figure 38-12). In 1987, Misch[69] included the technique of the sinus floor elevation through the implant osteotomy before implant placement. He reported on 170 sinus graft cases, with two complications and an uneventful resolution.

In the Misch SA classification, the treatment modality is dependent on the available bone height between the

WWW.HIGHDENT.IR

همیار دندانسازان

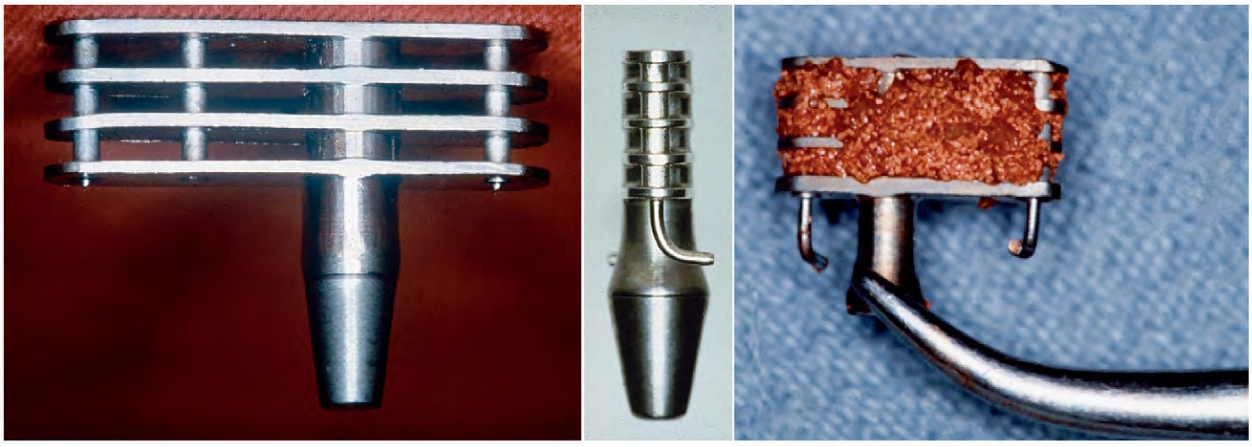


Figure 38-19 **A,** A Tatum S titanium (Ti) implant had a removable abutment for submerged healing and was 10 to 25 mm long (circa 1981). **B,** The S implant was 3 to 5 mm in width and had crestal fingers to prevent the implant from being displaced in the antrum during surgery. **C,** The implant would be filled with autograft and synthetic bone substitutes before insertion.

floor of the antrum and the crest of the residual ridge in the region of the ideal implant locations. The SA protocol also suggested a surgical approach, bone graft material, and a time table for healing before prosthetic reconstruction. In 1995, Misch[41] modified his 1987 classifications to include the lateral dimension of the sinus cavity; this dimension was used to modify the healing period protocol, because smaller-width sinuses (0 to 10 mm) form bone faster than larger-width (>15 mm) sinuses. The Division A–width ridge was also increased to 6 mm to permit more bone to flank the implant on each side. Jensen[128] and Chiapasco[129] and Misch et al.[130] proposed other classifications of the sinus graft procedure.

SURGICAL TECHNIQUE

Subantral Option One: Conventional Implant Placement

The first Misch SA treatment option, SA-1, occurs when sufficient bone height is available to permit the placement of endosteal implants following a usual surgical protocol. Because the quality of bone in the posterior maxilla often is D3 or D4 bone, bone compaction to prepare the implant site is common. This permits a more rigid initial insertion of the implant and also increases the BIC after initial healing.

In the abundant bone volume (Division A), root form implants are used for prosthetic support. The minimum ideal bone height is related to implant design and bone density; however, at least a 12-mm implant in height is suggested for a 4-mm-diameter threaded implant (Figure 38-20).

The goal of the sinus graft in the posterior maxilla is to augment the antral floor and gain additional bone in height. As a result, the sinus graft procedure results in an SA-1 condition after bone formation. Therefore after successful sinus grafts, the patient is placed in the SA-1 category and surgical approach. Often the softer bone type after sinus graft indicates bone compression rather than bone extraction techniques (Figure 38-21).

Because the maxillary sinus is not invaded during an SA-1 approach, it is less critical to evaluate the sinus before implant insertion. Therefore the contraindications for sinus graft surgery do not apply for implant insertion when adequate bone is present below the sinus for implants of adequate size to support the load of the prosthesis. Although a common axiom in implant dentistry is to remain 2 mm or more from an opposing landmark, this is not necessary in the SA region.

Narrower bone volume patients (Division B) in SA-1 may be treated with osteoplasty or augmentation to increase the width of bone. The insertion of smaller surface area implants (as small-diameter root form implants) is not suggested because the forces are greater in the posterior regions of the mouth, and the bone density is less than in most regions. In addition, the narrow ridge is often more medial than the central fossa of the mandibular teeth and will result in an offset load on the restoration, which will increase the strain to the bone.

Osteoplasty in the SA-1 posterior maxilla may change the SA category if the height of the remaining bone is less than 12 mm after the bone modification is completed. Augmentation for width may be accomplished with bone spreading, autogenous onlay, and/or appositional grafts. Larger-diameter implants are often required in the molar region, and bone spreading to place wider implants is the most common approach when the bone density is poor. If less than 2.5 mm of width is available in the posterior edentulous region (C–w), then the most predictable treatment option is to increase width using onlay autogenous bone grafts. After graft maturation the

WWW.HIGHDENT.IR

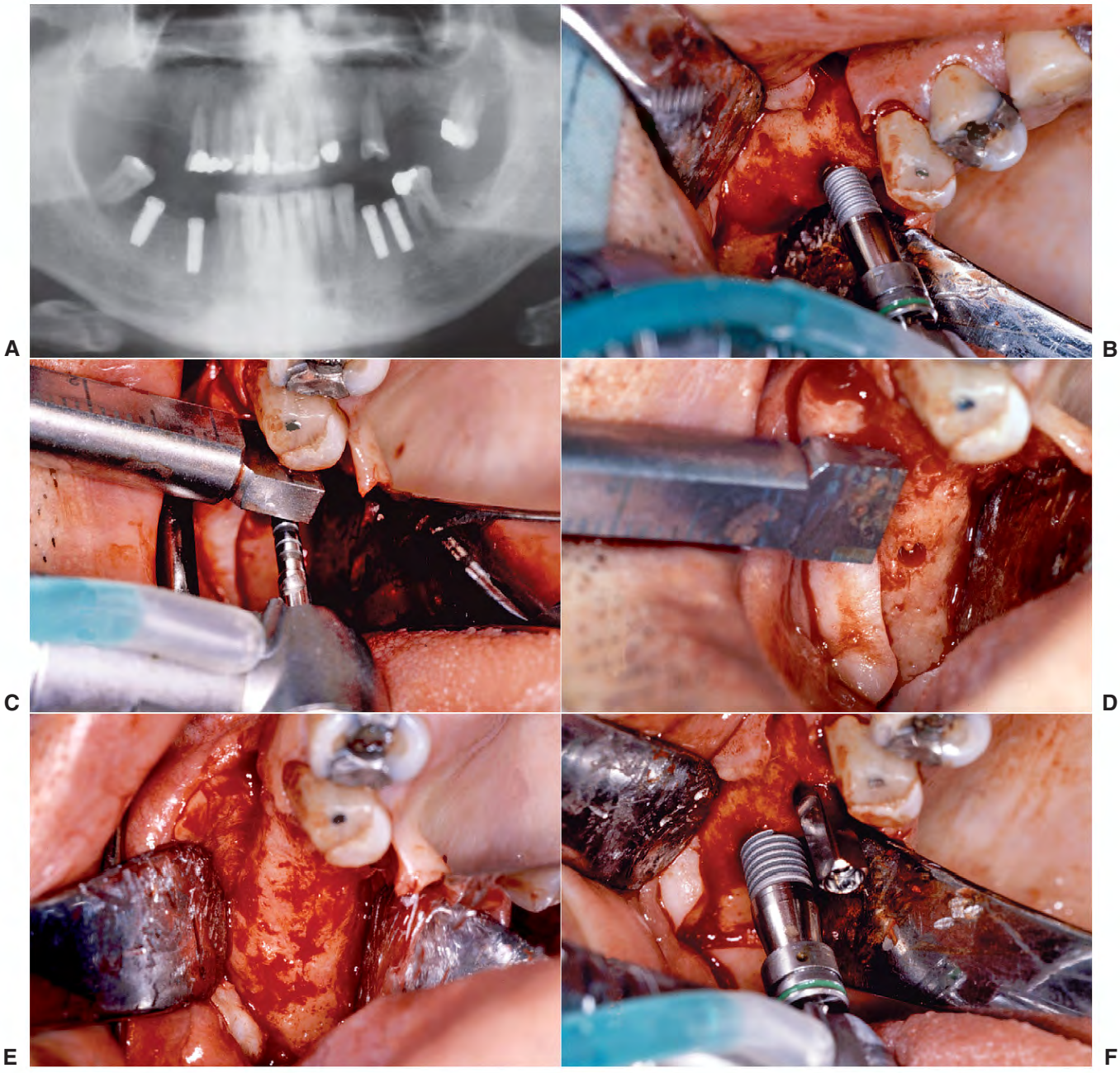


Figure 38-20 **A,** A panoramic radiograph of a posterior maxilla with abundant bone height in the right and left side. A subantral (SA)-1 surgical approach is indicated. **B,** The maxillary right posterior quadrant is incised and reflected. Abundant bone width is available for endosteal root form implants. **C,** The pilot drill maps out the position of the molar implants. A guide is used to place the anterior implant 2 mm from the adjacent tooth. **D,** The interimplant spacing guide is used to ensure the implants are at least 3 mm apart. **E,** After the implant osteotomy preparation, the first molar implant is threaded into position. **F,** A 5-mm-diameter second molar implant is threaded into position. These implants are 12 mm in length. *Continued*

area is reevaluated to determine the proper treatment plan classification.

Endosteal implants in the SA-1 category are left to heal in a nonfunctional environment for approximately 4 to 8 months (depending on bone density) before the abutment post or posts are added for prosthodontic reconstruction. Care is taken to ensure that the implants are not traumatized during the initial healing period. Progressive loading during the prosthetic phases of the treatment is suggested in D3 or D4 bone.

Subantral Option Two: Sinus Lift and Simultaneous Implant Placement

The second SA option in the Misch SA classification, SA-2, is selected when 10 to 12 mm of vertical bone

WWW.HIGHDENT.IR

همیار دندانسازان

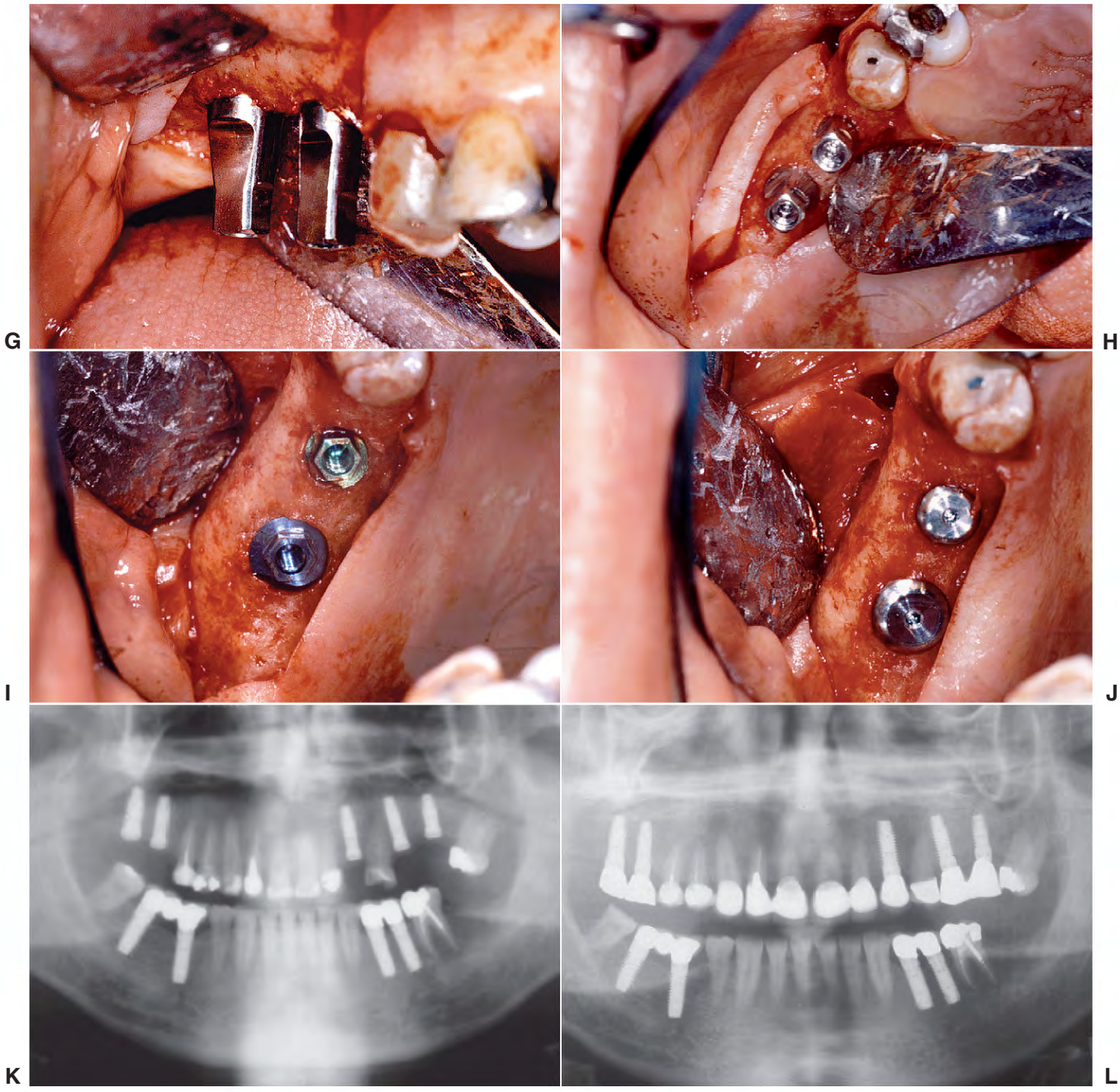


Figure 38-20, cont'd **G,** The molar implants are parallel to each other and perpendicular to the occlusal plane. **H,** Adequate bone exists on the buccal aspect of the implants. **I,** The implant mounts and abutments are removed. **J,** First-stage healing caps are inserted into the implant bodies. **K,** A postoperative panoramic radiograph demonstrates the maxillary posterior implants after stage I surgery. **L,** A 3-year postprosthesis panoramic radiograph demonstrates the restored implants supporting splinted crowns.

is present (2 mm less than the minimum height in SA-1) (Figure 38-22). To obtain the 12 mm of vertical bone necessary for improved implant survival in ridges of adequate width (Division A), the antral floor is elevated through the implant osteotomy 0 to 2 mm. Tatum[3,4,126] originally developed this technique in 1970, and Misch[69] first published it in 1987. Summers[131] published a similar procedure in 1994, 24 years after Tatum's first presentation.

The SA-2 surgical approach modifies the floor of the maxillary sinus. Therefore a preexisting pathologic condition of the sinus should not be present, because it may affect the implant site by retrograde infection.

Incision and Reflection

In an edentulous posterior maxilla, a full-thickness incision is made on the crest of the edentulous ridge from the tuberosity to the distal of the canine region.

WWW.HIGHDENT.IR

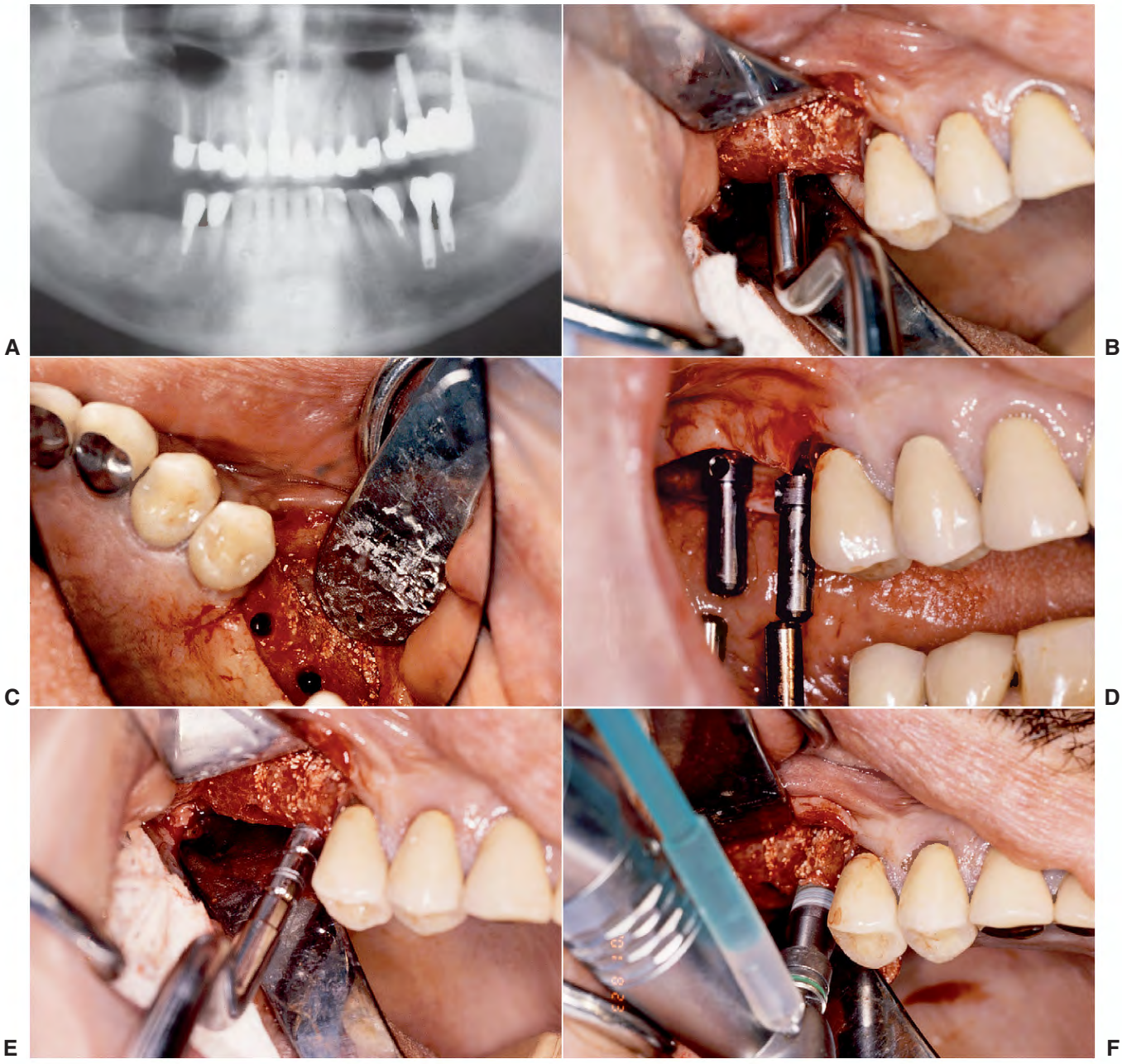


Figure 38-21 **A,** A panoramic radiograph of a maxillary right posterior region after a sinus graft has healed 6 months. **B,** A bone compression technique is used to prepare the osteotomy. **C,** The initial osteotomies with adequate final bone for implant insertion. The bone compaction technique is used to compress the bone, not expand the facial plate. **D,** Direction indicators indicate the osteotomies are parallel to each other and perpendicular to the occlusal plane. They are also over the direction indicators of the implant osteotomies in the mandibular arch. **E,** A larger-diameter osteotome prepares the mesial site. **F,** A threaded implant is inserted with a slow-speed hand piece at 30 rpm. *Continued*

A vertical, lateral relief incision is made at its distal and anterior extension of the crestal incision for approximately 5 mm. If minimal attached tissue exists on the crest of the ridge, which is more often observed in the premolar region, then the primary incision is made more palatal to place more keratinized tissue on the facial aspect. When teeth are present in the region, the crestal incision extends at least one tooth beyond the edentulous site. When one tooth is missing, the reflection is similar to a single-tooth replacement option, and even a direct (flapless technique) may be used.

A full-thickness palatal flap is first reflected because the palatal dense cortical plate facilitates soft tissue reflection. Special attention is given to avoid the pathway of the greater palatine artery or to remain completely subperiosteal so that this structure remains in the soft tissue. The labial mucosa is pulled off the edentulous ridge, rather than elevating the tissue from the bone.

WWW.HIGHDENT.IR

همیار دندانسازان

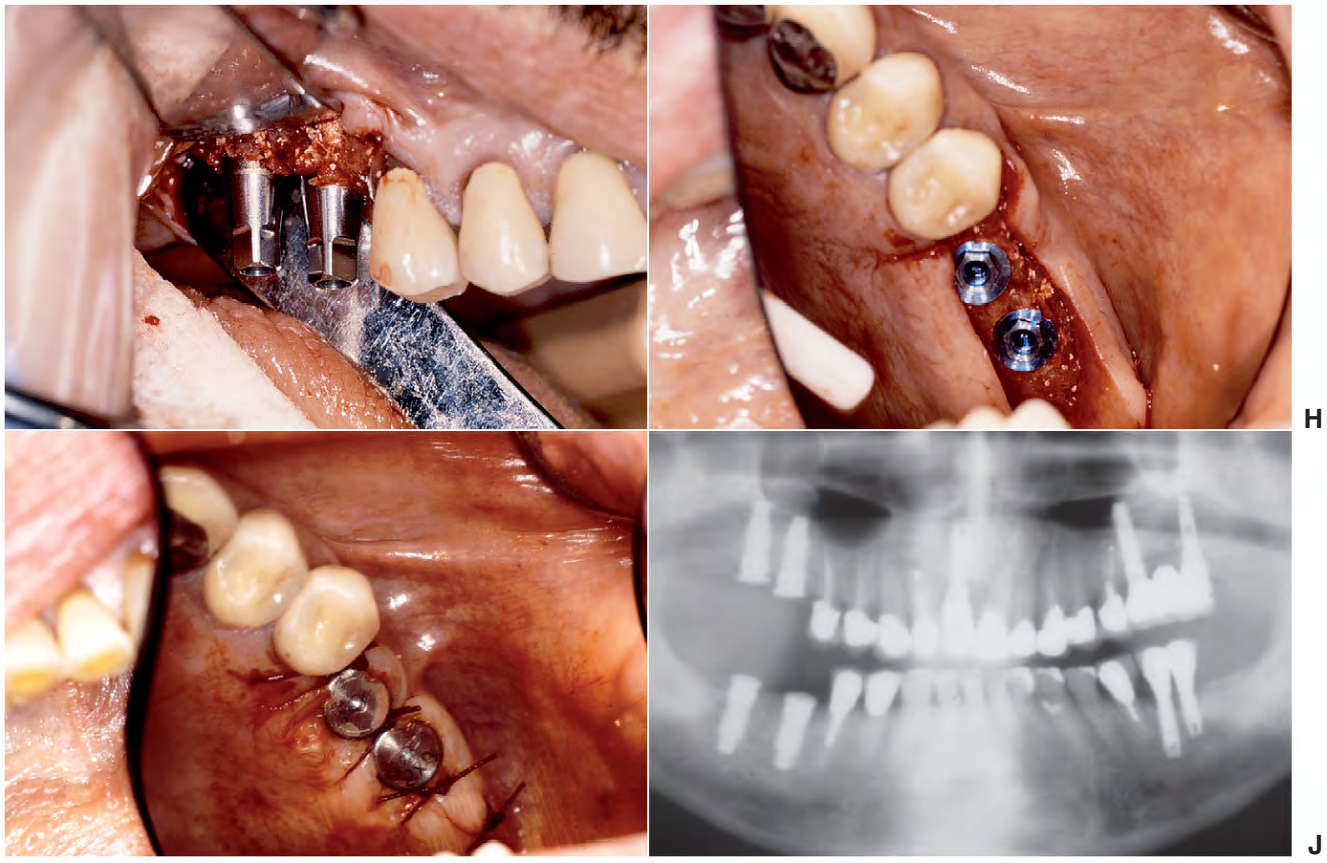


Figure 38-21, cont'd **G,** Two 5-mm-diameter, 12-mm-long molar implants are inserted into the posterior maxilla. **H,** The two implant mounts and abutments are removed. The two endosteal implants in position have adequate bone on the buccal aspect. **I,** Two permucosal extensions are inserted into the implant bodies, and the tissues are approximated for a one-stage surgical procedure. **J,** The postoperative radiograph of the two maxillary molar implants opposing two mandibular molar implants.

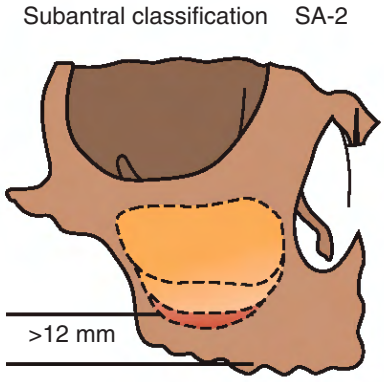


Figure 38-22 A subantral (SA)-2 option is indicated when 10 to 12 mm of bone is present below the maxillary sinus. The goal is to elevate the antral floor 0 to 2 mm within the osteotomy and simultaneously place an endosteal implant.

The crest is not used to leverage the tissue, because the ridge may have minimal cortical bone. This could result in damage to the residual ridge or possibly even penetrate the sinus cavity. Once the tissue is reflected, the width of the available bone is evaluated to ensure that it is greater than 6 mm wide and allows the placement of Division A root form implants.

Osteotomy and Sinus Lift (SA-2)

The endosteal implant osteotomy is prepared as determined by the density of bone protocol, which is usually D3 bone. The depth of the osteotomy is approximately 1 to 2 mm short of the floor of the antrum (Figure 38-23). When in doubt of the height dimension, the osteotomy should err on a shorter length. The implant osteotomy is prepared to the appropriate final diameter, short of the antral floor, following the established protocol for bone density.

A flat-end or cupped-shape osteotome of the same diameter as the final osteotomy is selected. It is of a different end shape than osteotomes used for bone spreading. The osteotome is inserted and tapped firmly in 0.5- to 1.0-mm increments beyond the osteotomy until reaching its final vertical position, up to 2 mm beyond the prepared implant osteotomy (Figure 38-24). A slow elevation of the sinus floor is less likely to tear the sinus mucosa. This surgical approach compresses the bone below the antrum, causes a greenstick-type fracture in the antral floor, and slowly elevates the unprepared bone and sinus membrane over the broad-based osteotome. If the osteotome cannot proceed to the desired osteotomy depth after tapping, then it is

WWW.HIGHDENT.IR

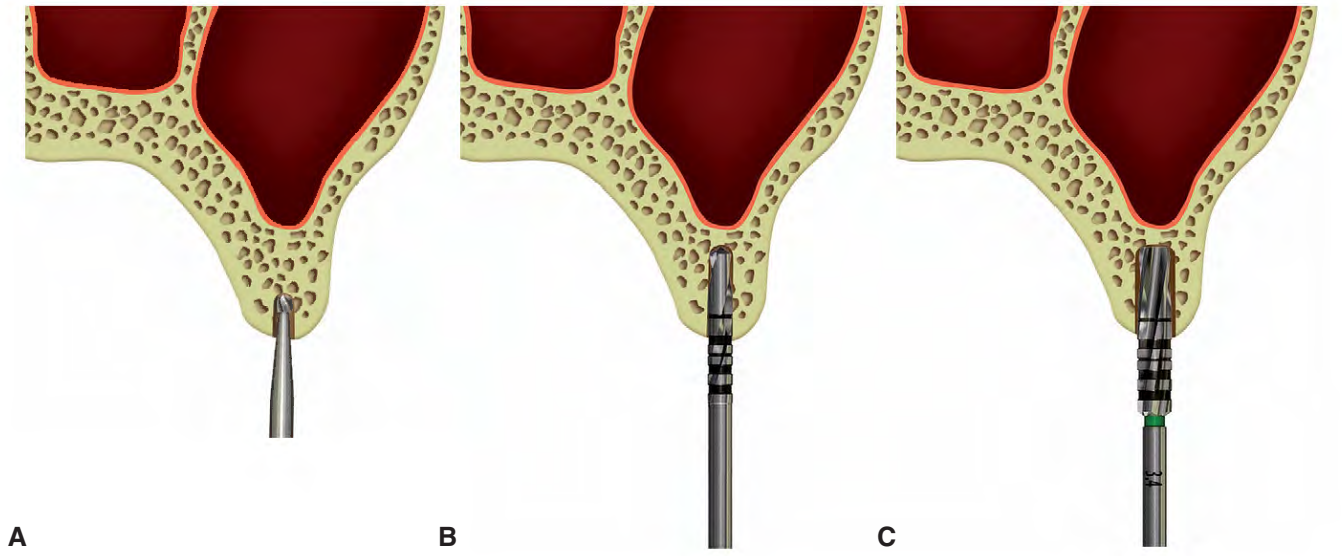


Figure 38-23 **A,** The subantral (SA)-2 technique begins with a pilot bur to mark the implant site. **B,** A 2-mm twist drill prepares the osteotomy 1 to 2 mm short of the sinus floor **C,** A final osteotomy width is sequentially obtained, remaining 1 to 2 mm short of the antral floor.

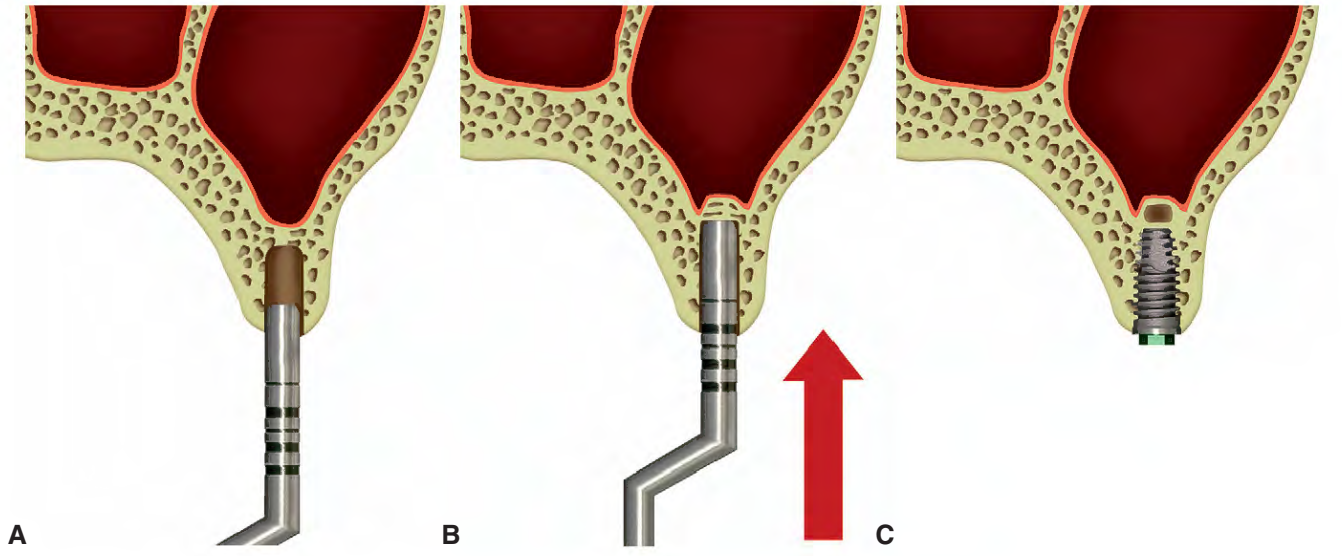


Figure 38-24 **A,** A flat-ended osteotome, the same diameter as the final osteotomy size, is used within the site. **B,** A surgical mallet is used to gently and slowly tap the osteotome 1 to 2 mm through the sinus floor. The osteotome raises the floor and 1 to 2 mm of bone with the sinus mucosa over the implant site. **C,** An implant is inserted into the osteotomy and extends 0 to 2 mm above the surgical sinus floor.

removed and the osteotomy is prepared again with rotary drills an additional 1 mm in depth. The osteotome is then reinserted to attempt the greenstick fracture of the antral floor. Extraction forceps may be used to rotate and remove the osteotome from the osteotomy. The osteotome is not luxated, because this will increase the width of the final osteotomy.

Once the osteotome prepares the implant site, the implant may then be threaded into the osteotomy and extend up to 2 mm above the floor of the sinus. The implant is slowly threaded into position so that the membrane is less likely to tear as it is elevated. The apical portion of the implant engages the more dense bone on the cortical floor, ideally with bone over the apex, and an intact sinus membrane. The implant may extend 0 to 2 mm beyond the sinus floor, and the 1 mm of compressed bone covering over the implant apex results in as much as a 3-mm elevation of the sinus mucosa (Figure 38-25).

Some authors have used the SA-2 sinus lift procedure to gain more than 2 mm of implant vertical height and/or place bone graft materials in the osteotomy site before implant insertion. These blind surgical techniques increase the risk of sinus membrane perforation. When the sinus mucosa is perforated, the graft material may extrude into the atrum and increase the risk of postoperative infection, because it may occlude the ostium and alter the environment of the

WWW.HIGHDENT.IR

همیار دندانسازان

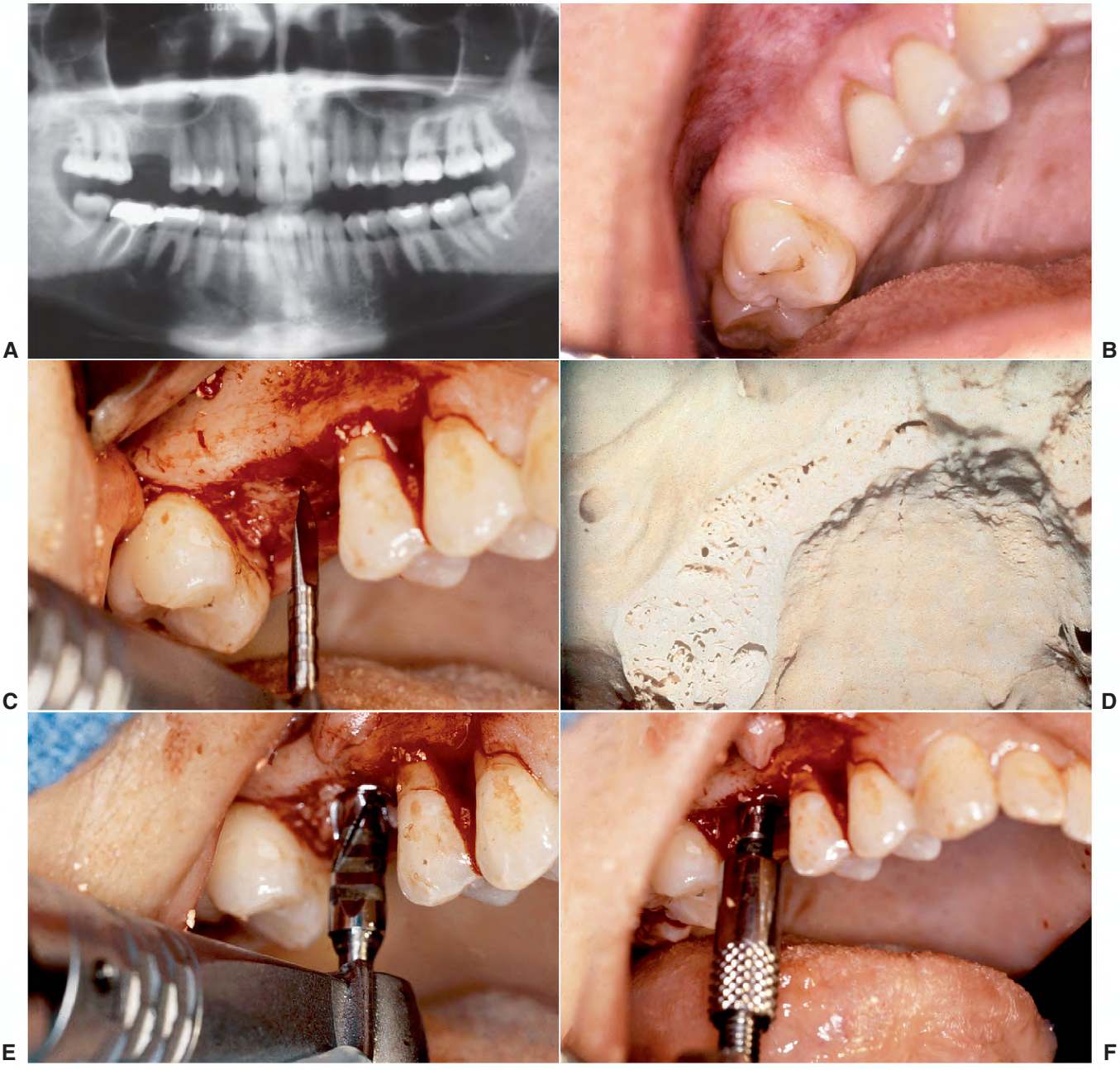


Figure 38-25 **A,** A panoramic radiograph of a patient with a missing maxillary right first molar; 10 mm of bone exists below the sinus, and the ideal implant length is 12 mm long. Therefore this is a subantral (SA)-2 surgical option. **B,** The intraoral view of the missing first molar appears to have adequate bone width for an endosteal root form implant. **C,** The soft tissues are reflected, and a pilot drill is used to prepare the initial osteotomy 1 to 2 mm short of the sinus floor. **D,** The floor of the sinus may have a thin margin of bone that is more dense then the bone below this structure. This bone will help support the apical portion of the implants, once inserted. **E,** The final osteotomy diameter is prepared, also 1 to 2 mm short of the sinus floor. **F,** The flat-ended (or cup-shaped) osteotome, the same diameter as the final osteotomy, is placed into the site. The position of the stop is at a 12-mm depth.

Continued

sinus proper. If a sinus infection occurs, then a bacterial smear layer may accumulate on the implant apex. This prevents future BIC and may contribute to future sinus infections.

The success of the intact sinus membrane lift cannot be confirmed before or at the time of implant placement. Attempts to feel the elevation of the membrane from within an 8-mm-deep implant osteotomy that is approximately 3 mm in diameter may easily cause tearing of the sinus lining. Four to 6 months after the surgical procedure, a radiograph that demonstrates bone over the implant apex may be used to indicate the success of the 0- to 2-mm increased vertical height. The patient's prosthodontic treatment may proceed similar to that in the SA-1 category.

It is not unusual for the maxillary sinus to drape over the apices of a maxillary molar. When the tooth is extracted, the sinus often resides too close to the crest

WWW.HIGHDENT.IR

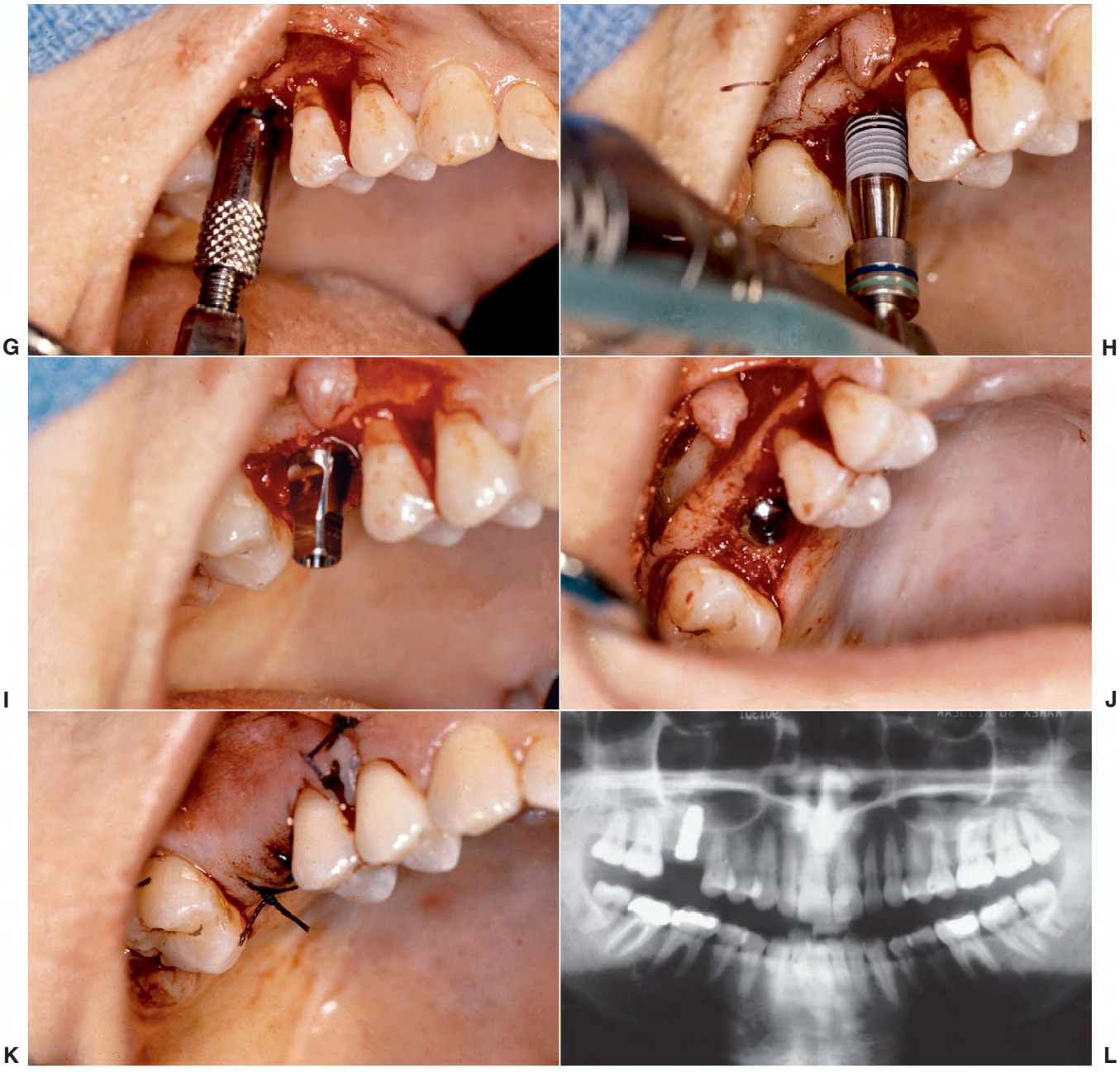


Figure 38-25, cont'd **G,** A mallet gently and slowly taps the flat-ended osteotome through the sinus floor, for up to 2 mm. **H,** An implant is slowly threaded into the implant site and proceeds up to 2 mm beyond the sinus floor. **I,** The 5-mm-diameter, 12-mm-long molar implant is in place. **J,** A first-stage cover screw is inserted into the implant body. **K,** The tissues are approximated for primary closure. **L,** A postoperative radiograph after 4 months indicates bone has formed over the apex of the implant.

Continued

for implant insertion, without grafting the sinus floor. Rosen et al.[132] developed a modification to the SA-2 treatment approach for use at the time of an extraction of a maxillary molar.

The technique is indicated when the maxillary molar is extracted, the surrounding walls of bone are intact, and no periapical pathologic condition extends into the antrum. The crest of the ridge to the antral floor should be 7 mm or more in height. Once the tooth is extracted and the surrounding boney walls confirmed, a modification of the SA-2 technique is in order. A 5- to 6-mm trephine bur is used in the center of the extraction site and prepares the bone 1 to 2 mm below the antral floor. A 5- to 6-mm-diameter, flat-ended or cup-shaped osteotome and mallet intrudes the core of bone 2 mm above the sinus floor, creating 9 mm or more of vertical bone. A socket graft may be used within the extraction socket but is not pushed into the surgical space of the

WWW.HIGHDENT.IR

همیار دندانسازان

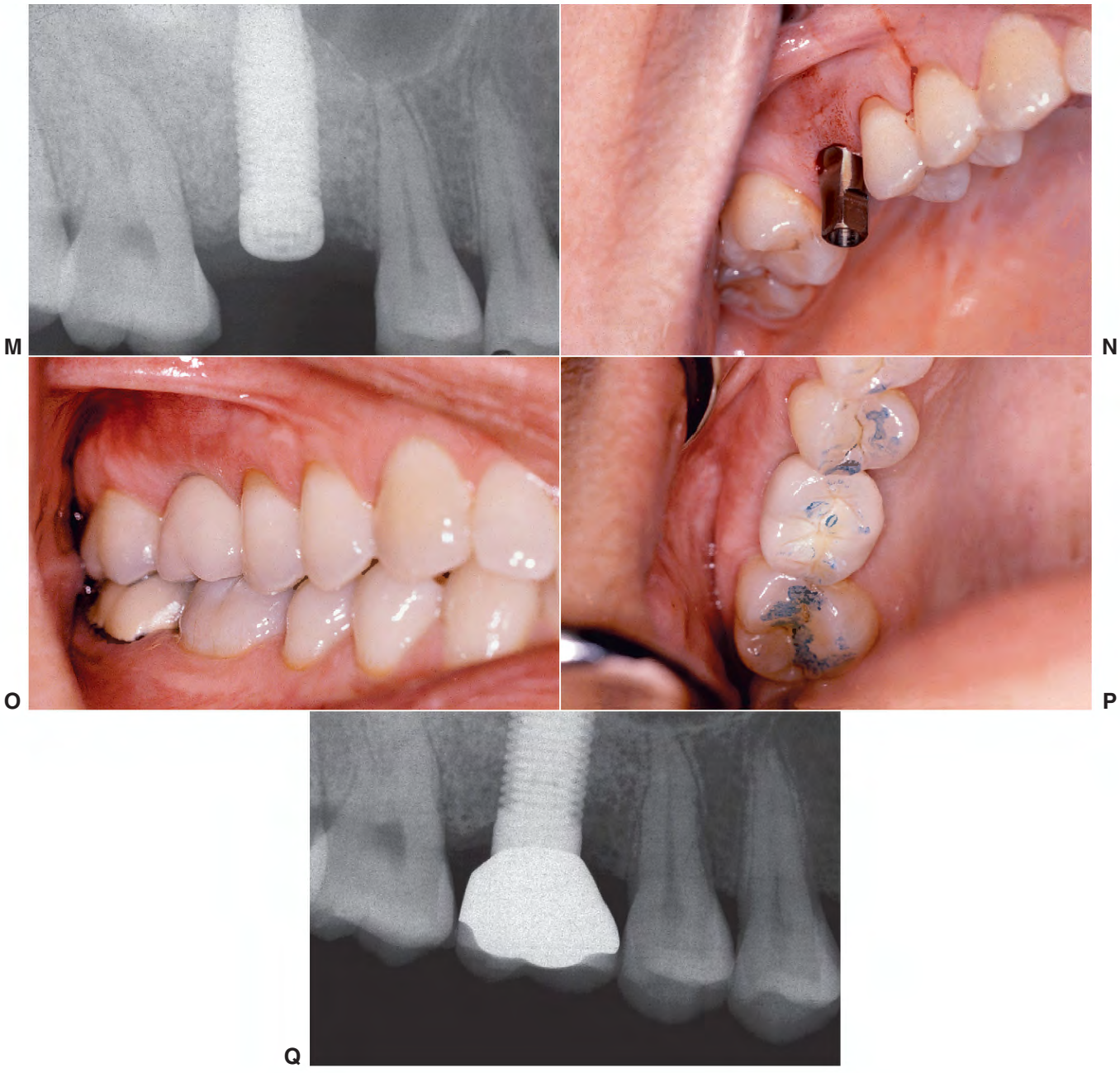


Figure 38-25, cont'd **M,** A periapical (PA) radiograph after 5 months confirms bone has formed over the apex of the implant. **N,** The molar implant is uncovered, and an abutment is positioned for the restoration. **O,** The final crown is cemented onto the abutment to restore the maxillary first molar. **P,** The occlusion on the crown follows the guidelines of implant-protective occlusion. **Q,** A periapical radiograph at a subsequent recare appointment demonstrates the marginal bone has been maintained around the implant.

sinus, because it may perforate the sinus mucosa. After 4 months, an implant may be inserted, often with a SA-1 or SA-2 approach.

If sinus membrane perforation occurred during the initial implant placement procedure, then increased bone height is not likely. This is the primary reason why only 0 to 2 mm of additional bone height is attempted with this technique. However, even when membrane perforation occurs and/or no bone grows around the apical end of the implant, the SA-2 technique is of benefit, because the apical end of the implant is surrounded by denser bone. This enhances rigid fixation during healing and increases BIC, leading to improved loading conditions. As a result of the less predictable outcome, one additional implant may be placed or a larger-diameter implant is suggested. If inadequate bone is formed around the apical portion of a implant, then a progressive-loading protocol for D4 bone is suggested during prosthetic reconstruction.

Worth and Stoneman[133] have reported a comparable phenomenon of bone growth under an elevated sinus membrane called *halo formation*. They observed the

WWW.HIGHDENT.IR

natural elevation of the sinus membrane around teeth with periapical disease. The elevation of the membrane resulted in new bone formation once the tooth infection was eliminated. In an article by Palma et al.,[134] the elevation of the sinus membrane in implant insertion, with or without a graft material below the mucosa, gave similar results in primates regarding implant stability or BIC after healing. As a result of the autologous bone present above the apical portion of the implant with an SA-2 technique, and the sinus floor fracture (which increases the regional accelerated phenomenon of bone repair and formation), new bone formation over the implant apex is predictable.

When more than 2 mm of bone is desired above the sinus floor, a lateral-access opening to the antrum, with direct vision and access to elevate the sinus mucosa on the floor, is suggested. Because the lateral-access opening to the sinus is often 10 × 10 mm or larger and the lateral maxilla over the sinus is only 1 mm thick, the sinus mucosa is more readily elevated. If the mucosa is torn, then it may also be sealed with collagen before the sinus floor augmentation. Attempting to elevate the sinus mucosa more than 2 mm through an implant osteotomy 3 to 4 mm wide and 8 mm deep is not predictable. Reiser et al.[135] reported that when the sinus elevation was 4 to 8 mm in cadavers, almost 25% resulted in sinus perforation. The implant osteotomy sinus floor augmentation technique is often attempted because of the perceived ease of surgery of an SA-2 technique versus an SA-3 lateral-access procedure. However, the few extra minutes needed to gain access from the maxillary lateral wall and direct vision to the antral floor present obvious benefits.

Subantral Option Three: Sinus Graft with Immediate or Delayed Endosteal Implant Placement

The third approach to the maxillary posterior edentulous region, SA-3, is indicated when at least 5 mm of vertical bone and sufficient width are present between the antral floor and the crest of the residual ridge in the area of a needed prosthodontic abutment (Figure 38-26). A Tatum lateral maxillary wall approach is performed just superior to the residual alveolar bone. After the lateral-access window and membrane are rotated in and upward to a superior position, a mixture of autogenous bone, alloplast, and/or allograft material is placed in the space previously occupied by the sinus. When the original ridge is greater than 5 mm in width, the implant may be inserted at the same time as the sinus augmentation or delayed 2 or more months before implant insertion.[53,55-60] The short delay between graft placement and implant insertion ensures the graft is more stable and is healing without compromise related to postoperative sinus infection. When the original ridge width is Division B or C–w, an onlay graft in conjunction with the sinus augmentation is a possible treatment option (Table 38-2).

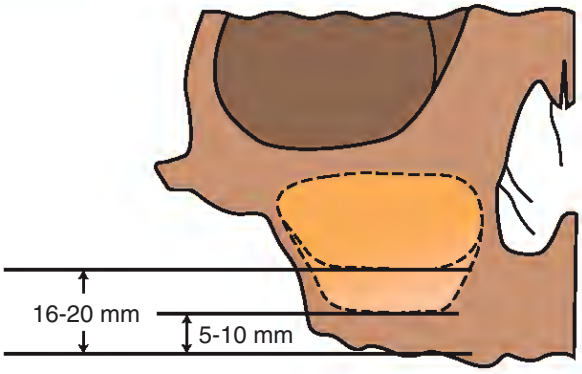


Figure 38-26 A subantral (SA)-3 sinus category exists when 5 to 10 mm of bone exists below the antrum. A Tatum lateral-wall sinus membrane elevation and sinus graft to obtain 16 to 20 mm of bone height is indicated.

The author has chosen a residual height of 5 mm for the SA-3 category for two main reasons: (1) this height (in adequate bone width and quality) can be considered sufficient to allow primary stability of implants placed at the same time of the sinus graft procedure; (2) this height may allow the use of alloplastic materials, because

Table 38-2 Healing Times for Treatment Categories

TREATMENT	HEIGHT (mm)	PROCEDURE	HEALING TIME (MONTHS) GRAFT	HEALING TIME (MONTHS) IMPLANT
Subantral (SA)-1	>12	Division A root form placement	—	4-6
SA-2	10-12	Sinus lift, simultaneous Division A root form placement	—	6-8
SA-3	5-10	Lateral-wall approach sinus graft, delayed Division A root form placement	2-4	4-8*
SA-4	<5	Lateral-wall approach sinus graft, delayed Division A root form placement	6-10	4-10*

*Evaluate at implant insertion.

WWW.HIGHDENT.IR

همیار دندانسازان

Figure 38-27 The second division block of the fifth cranial nerve (V2; deep in the pterygomaxillary space) is used for anesthesia.

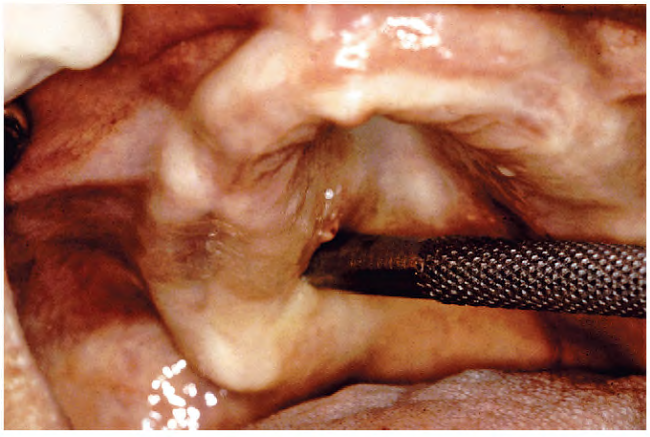


Figure 38-28 An injection deep within the greater palatine foramen may obtain V2 block anesthesia.

adequate amounts of bone may be harvested from the tuberosity to augment the alloplastic component of the graft.

Anesthesia

Infiltration anesthesia has been used with success for sinus graft surgeries in the past; however, more profound regional anesthesia is achieved by blocking the secondary division of the maxillary nerve (V2). The sinus graft surgery often requires the reflection of the soft tissue to the zygomatic process. In addition, several branches of the maxillary branch of the fifth cranial nerve innervate the sinus mucosa. As such, a V2 block is advantageous for patient comfort, and this achieves anesthesia of the hemimaxilla, side of the nose, cheek, lip, and sinus area.

Two options exist for V2 block anesthesia: (1) high and within the pterygomaxillary tissue behind the posterior wall of the maxilla (Figure 38-27) or (2) at the depth of approximately 1 inch with a long-gauge needle within the greater palatine foramen (Figure 38-28). The first method is easier to perform but may injure the pterygoid plexus or the maxillary artery and result in hematoma, or it may fail to reach the proper landmark. With the second option, it is more difficult to find the foramen and negotiate up the canal.[136] It may also injure the greater palatine artery or nerve. Too deep an administration with a greater palatine approach may result in the penetration of the orbit floor. Possible sequelae include periorbital swelling and proptosis, diplopia, retrobulbar block with dilated pupil, corneal anesthesia, motionless eye, retrobulbar hemorrhage, and optic nerve block with transient loss of vision. However, the success rate is greater, and the clinical risks appear minimal. Therefore most often, the first attempt for block anesthesia is within the greater palatine foramen; if unsuccessful, then the high posterior approach is used. Prevention of these complications is ensured by reduction of the needle depth measurement for smaller patients and the strict application of the technique. Proper angulation during penetration prevents the penetration into the nasal cavity through the medial wall of the pterygopalatal fossa.

Infiltration anesthesia is first administered to the posterior and middle alveolar nerve and greater palatine nerve. Scrubbing, gowning, and draping of the patient is next. Then after the infiltration is effective, the V2 block is administered. A long-acting anesthetic such as bupivacaine 0.5% (Marcaine) or etidocaine 1.5% (Duranest) with epinephrine 1:200,000 is preferred. Block anesthesia with these agents is longer acting than infiltration in the maxilla.[136]

Of benefit to find the greater palatine foramen is an open-bore instrument, (i.e., the handle of a mouth mirror with the mirror portion removed). Pressure is applied with this instrument along the palatal tissue, at the union of the residual ridge and hard palate, in the region of the second molar (Figure 38-29). Most often,

Figure 38-29 A blunt open bore instrument determines the greater palatal foramen.

WWW.HIGHDENT.IR

the open-bore handle will feel and recede into the foramen. Slight pressure for a few seconds then marks the tissue over the opening of the foramen. A long, 1 1/2-inch needle is introduced into the foramen from the opposite side of the mouth and negotiates the canal for approximately 1 inch.

Incision Line and Reflection

A crestal incision is made on the palatal aspect of the maxillary posterior edentulous ridge from the tuberosity to one tooth anterior to the anterior wall of the maxillary sinus, leaving at least 3 mm of attached tissue on the facial aspect of the incision. Because ridge resorption occurs toward the midline at the expense of the buccal dimension, the incision is made with awareness of the greater palatal artery, which proceeds close to the crest of the ridge in the severely atrophic maxilla. If bleeding from the palatal flap occurs, a hemostat may be used to constrict the blood vessels distal to the bleeding, pressure may be applied over the greater palatine foramen with a blunt instrument, or electrocoagulation at the bleeding site may be used.

A vertical relief incision is made on the distal of the incision to enhance surgical access to the maxillary tuberosity. A broad-base anterior vertical relief incision is also made at least 10 mm anterior to the anterior vertical wall of the antrum. This may mean that the incision is made on the distal aspect of the first bicuspid or canine when the anterior wall of the antrum is very close to the roots of the anterior teeth. The facial soft tissue flap is designed, following general principles, with a base wider than the crest to ensure proper blood supply. The palatal portion of the flap is first reflected, followed by the facial crestal tissue, which is lifted off the crest.

The facial full-thickness mucoperiosteal flap is reflected to expose the complete lateral wall of the maxilla and a portion of the zygoma. The facial flap should be reflected to provide complete vision and access to maxillary lateral wall. The reflection is usually excessive if the infraorbital nerve (fan-shaped appearance) emerging from the infraorbital foramen is visualized. Aggressive reflection of the facial flap may cause damage to this exposed nerve structure. The reflected labial tissue can be tied to the cheek mucosa with 2-0 silk sutures, carefully avoiding the parotid duct. All fibrous tissue should be removed from the future lateral wall access site to avoid soft tissue contamination of the bone graft. A wet surgical sponge can be used for this purpose (Figure 38-30).

Access Window

The overall design of the lateral-access window is determined after the review of the CT scan, which helps determine the thickness of the lateral wall of the antrum, the position of the antral floor from the crest of the ridge, the posterior of the anterior wall in relationship to the teeth (if present), and the presence of septa on the floor and/or walls of the sinus.

The outline of the Tatum lateral-access window is scored on the bone with a rotary hand piece under copious cooled sterile saline. It is often easier to perform this step at 50,000 rpm, but it is possible even at 2000 rpm, depending on the lateral-wall bone thickness. With experience, the first bur is usually a No. 6 round carbide, which scratches the bone and designs the overall window dimension. This bur is followed with a No. 4 round diamond, which "polishes" away the bone within the groove made by the carbide bur. A No. 6 round diamond bur for the entire process is of benefit for an early learning curve, because carbide burs more readily tear the sinus membrane if the bur inadvertently comes in contact with it. The inferior score line of the rectangular access window on the lateral maxilla is placed approximately 2 to 5 mm above the level of the antral floor (which is 5 to 10 mm from the crest). If the inferior score line is made at or below the level of the antral floor, then infracture of the lateral wall will be very difficult, leading to possible membrane perforation. If the inferior score line is made too high (>5 mm) above the sinus floor, then a ledge above the sinus floor will result in a blind dissection of the membrane on the floor. When available bone height is almost 10 mm, the sinus graft requires only 5 to 6 mm of additional bone. Under these conditions, the lateral access is more limited, and the inferior score line may be 1 to 2 mm above the antral floor.

The most superior aspect of the lateral-access window should be approximately 8 to 10 mm above the inferior score line. A soft tissue retractor placed above the superior margin of the lateral-access window helps retract the facial flap and prevents the retractor's inadvertent slip into the access window, which may damage the underlying membrane of the sinus.

The anterior vertical line of the access window is scored approximately 5 mm distal to the anterior vertical wall of the antrum. If the sinus access window outline is difficult to determine in relation to the sinus cavity, then it should err over the antrum rather than over the bone around this structure. The distal vertical line on the lateral maxilla is approximately 15 mm in the edentulous posterior maxilla from the anterior limit of the window and is usually in the region of the first molar, which is within direct vision of the operator. A larger access window offers many advantages, including less stress on the membrane during initial elevation and ease of additional membrane elevation with instruments because of the direct access that facilitates graft placement. Excessive size of the lateral-access window is not indicated, because the outer bony wall of the maxilla helps bone grow into the sinus graft material.

The corners of the access window are usually round, rather than at right or acute angles. If the corner angles are too sharp, then membrane perforation may occur

WWW.HIGHDENT.IR

همیار دندانسازان

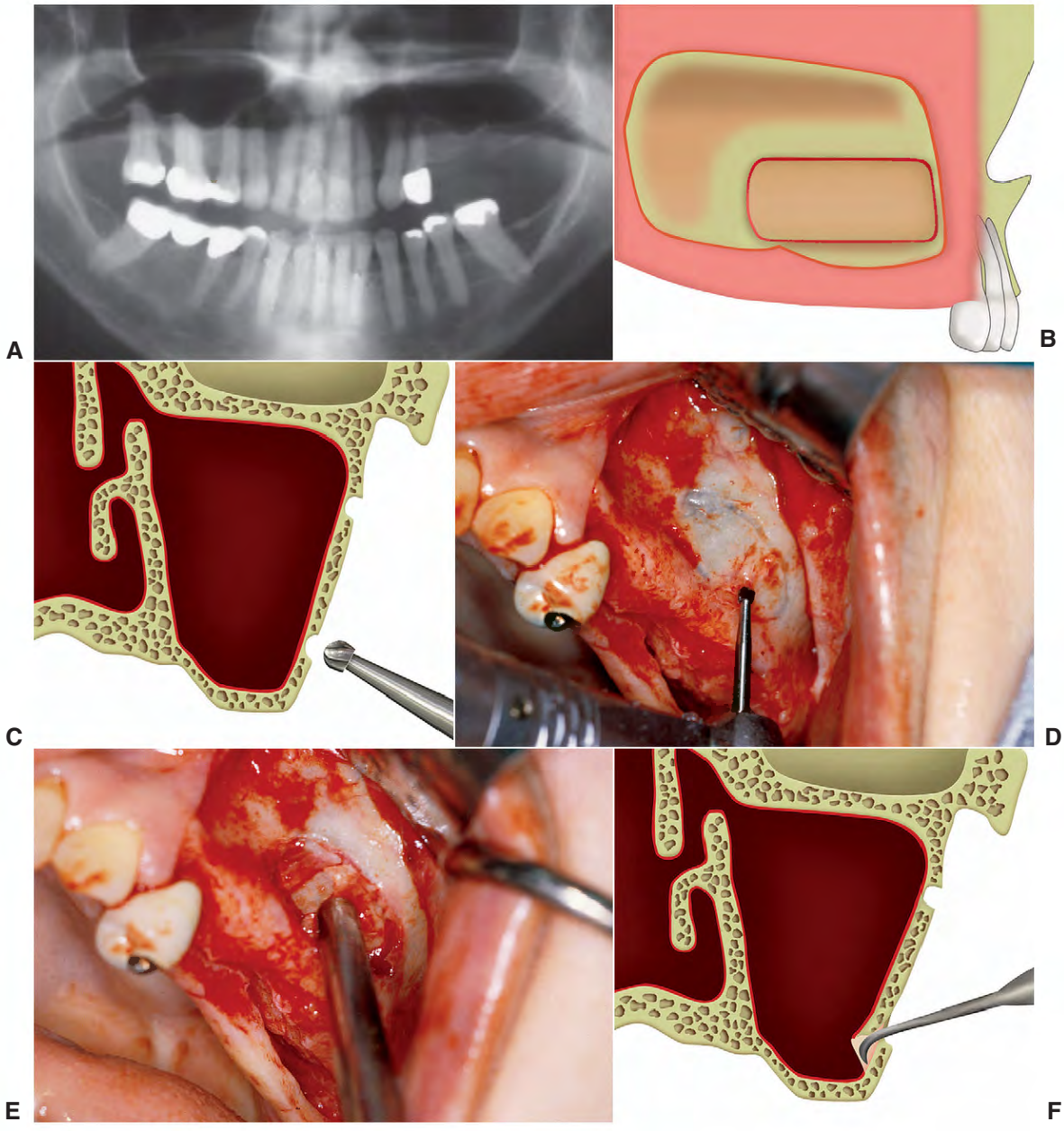


Figure 38-30 **A,** A panoramic radiograph of missing posterior teeth in the maxillary left region; 5 to 10 mm of bone exists below the maxillary sinus floor. **B,** The lateral maxilla is reflected and reveals the zygomatic process, tuberosity, and lateral maxilla. The Tatum access window is 2 to 5 mm above the antral floor, 2 to 5 mm from the anterior wall, 15 mm long, and 10 mm in height. **C,** The carbide drill should not perforate the lateral maxilla, which will tear the sinus membrane. A paintbrush stroke is used to outline the access window. **D,** A No. 4 to 6 diamond is used in a straight or contraangle hand piece and deepens the score line of the access window until a bluish hue or bleeding is observed. **E,** A flat-ended metal punch is placed over the lateral-access window. A mallet is used to gently tap the instrument and greenstick fracture the lateral-access window from the lateral wall of the maxilla. **F,** The back, rounded portion of the curette is placed against the lateral-access window and gently pushes it medial. The sharp blade of the curette is placed against the inner wall of bone and scrapes the sinus membrane off the bone. *Continued*

from the use of a surgical curette at the corner or during the infracture of the lateral wall. Once the lateral-access window is delineated, the rotary bur continues to scratch the outline with a paintbrush stroke approach under cooled sterile saline irrigation, until a bluish hue is observed below the bur or hemorrhage from the site is observed. The expansion of the maxillary sinus after tooth loss pushes the arteries of the membrane to the outside of the structure and just below the surrounding bone. Therefore either the bluish hue of the membrane or bleeding in the area are signs of approaching the sinus membrane. This observation should be achieved circumferentially around the access window. The access window should not be overprepared in depth, because touching the membrane with rotary burs may cause it to tear (see Figure 38-30, *C, D*).

WWW.HIGHDENT.IR

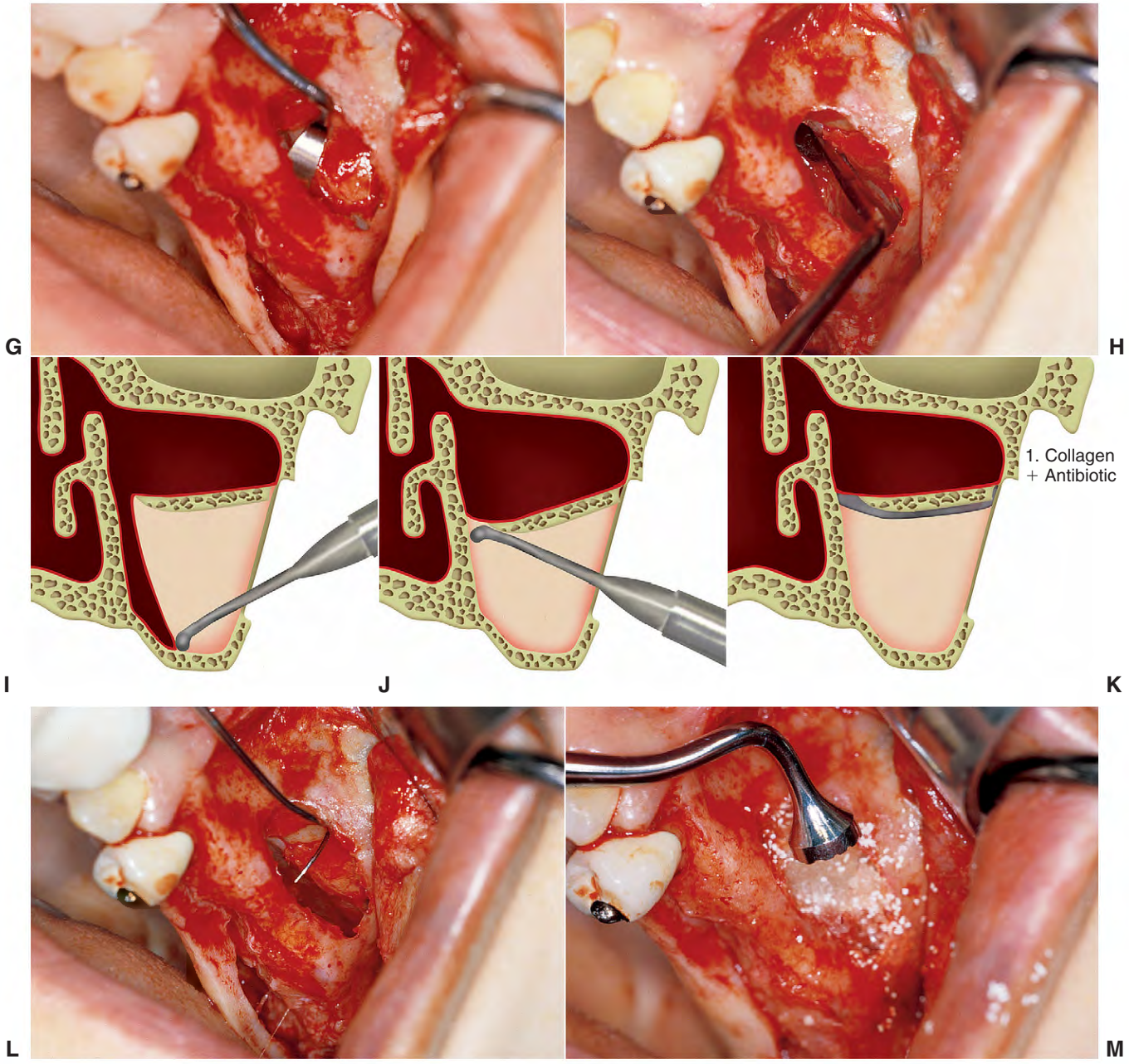


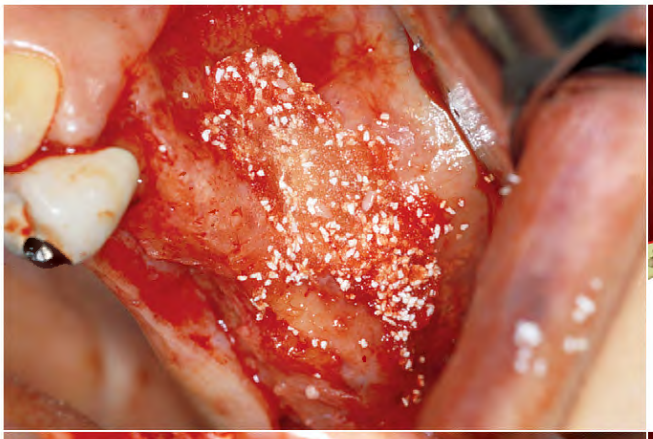
Figure 38-30, cont'd **G,** The first curette is used to scratch the membrane off the floor of the sinus. **H,** The second curette scrapes the floor and medial wall of the antrum. **I,** The sinus curette scratches the floor of the antrum to the medial wall. **J,** An intraoral view of the elevated sinus membrane along the medial wall of the antrum. The distance from the crest of the ridge to the elevated sinus is at least 16 mm. **K,** A bovine collagen (CollaTape) soaked with 0.2 mL of antibiotic is placed along the ceiling of the sinus graft as the first layer of the sinus graft. **L,** A dental explorer is used to scratch the floor and inner walls of the sinus graft region, to develop a regional acceleratory phenomenon for the bone graft. **M,** The sinus graft mixture is positioned on the original sinus floor.

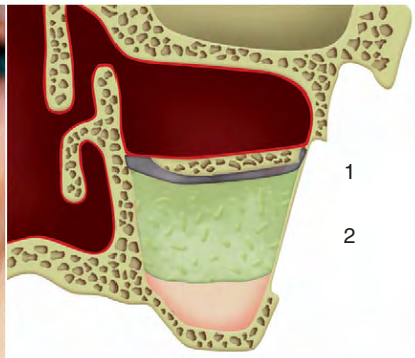
Continued

It should be noted that the largest blood vessel in the lateral wall is from an endosseous anastomosis from the posterior superior alveolar and the infraorbital artery that is approximately 1.5 mm in diameter. However, when the lateral wall is very thin in the edentulous patient, this blood vessel atrophies and often is not present. Thus excessive bleeding is rare. This vessel proceeds in the lateral wall of the maxilla 15 to 20 mm from the dentate crest. The horizontal lines of the access window should not be positioned directly over this structure. The vertical lines of the access window often cut through the artery. Because the blood supply may be from either direction, both vertical access lines may have bleeding. This is rarely a concern for vision or blood loss during the procedure. If intraosseous bleeding is a problem, the high-speed diamond used to score the window may be used without irrigation and polish the bleeding site, which cauterizes the vessel

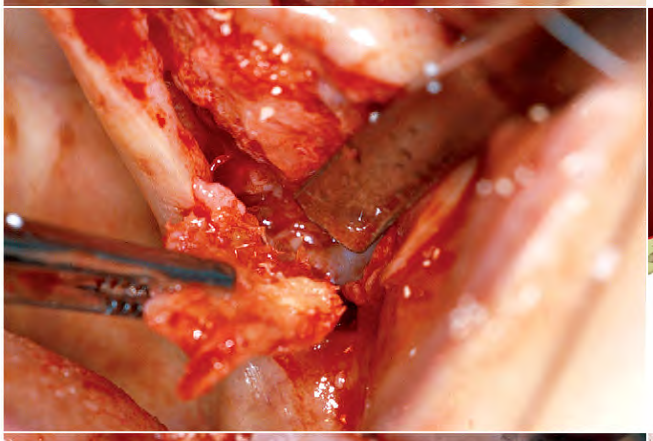
WWW.HIGHDENT.IR

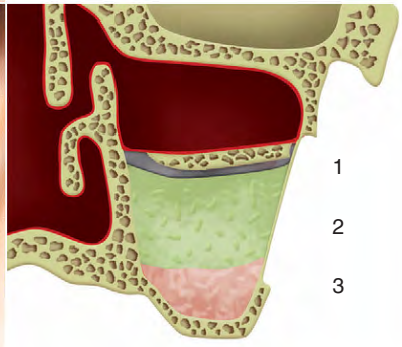
همیار دندانسازان


N

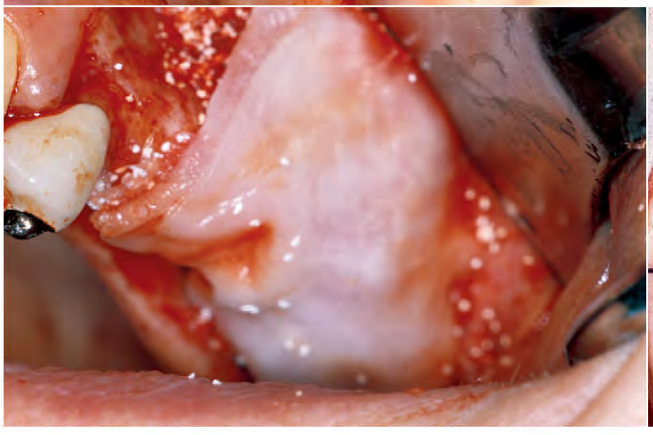


1. Collagen + Antibiotic
2. Microporous HA + DFDB (intermediate) + Antibiotic
O


P



1. Collagen + Antibiotic
2. Microporous HA + DFDB (intermediate)
3. Autogenous bone (inferior)
Q


R

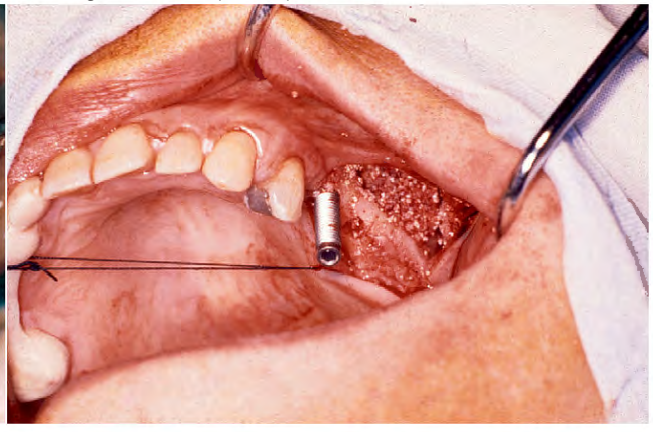

S

Figure 38-30, cont'd **N, O,** The top layer (collagen and antibiotic) and the second layer of the sinus graft. The second layer of the sinus graft is the largest component of the graft. **P,** The maxillary tuberosity is harvested with rongeurs, chisels, or trephine bur. **Q,** The sinus graft has three primary layers to fill the inferior third of the maxillary sinus. The first layer is collagen and antibiotic. The second layer is a combination of microporous hydroxyapatite (HA) (as freeze-dried bone [FDB]), demineralized freeze-dried bone (DFDB), antibiotic, and platelet-rich plasma (PRP). The third layer is autologous bone. **R,** A collagen layer or barrier membrane, soaked in PRP or platelet-poor plasma (ppp) is placed over the lateral wall of the maxilla. **S,** When conditions are satisfactory, endosteal implants may be inserted into the 5 to 10 mm of available bone below the antrum and extend into the graft site (different patient).

Continued

from the heat on the bony wall. Electrocautery may also be used on this vessel, if necessary. A hemostat on the artery may be less effective, because it may fracture the lateral wall and/or perforate the sinus mucosa. Elevating the head and a surgical sponge applied to the site for a few minutes is also sufficient to control this hemorrhage in many cases.

Sinus Membrane Elevation

A flat-ended metal punch (or mirror handle) and mallet are used to gently infracture the lateral-access window from the surrounding bone, while still attached to the thin sinus membrane. The flat-ended punch is first positioned in the center of the window. If light tapping does not greenstick fracture the bone, the flat-ended

WWW.HIGHDENT.IR

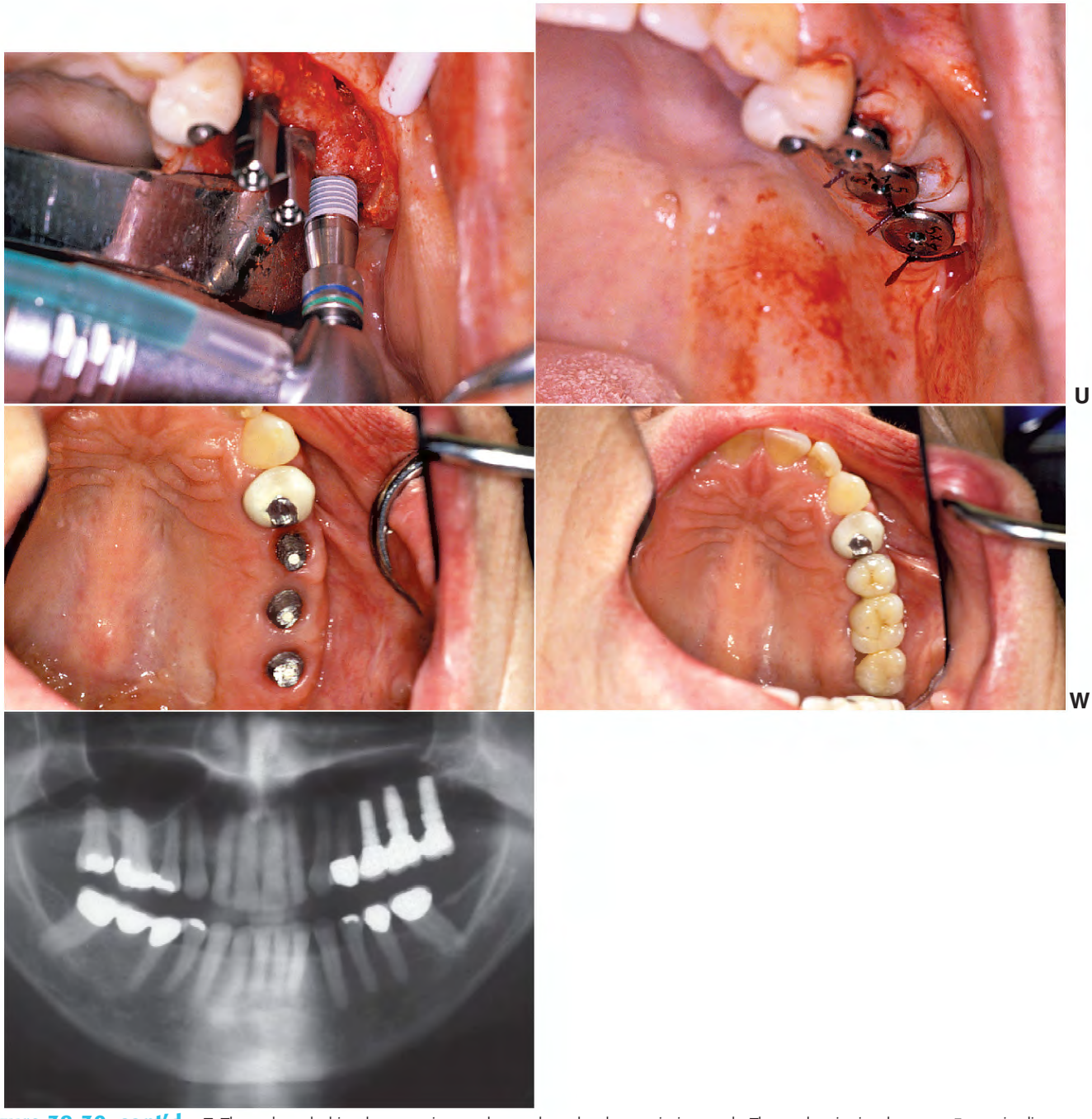


Figure 38-30, cont'd **T,** Three threaded implants are inserted to replace the three missing teeth. The molar site implants are 5 mm in diameter and 12 mm long. **U,** Permucosal abutments are inserted into the implant bodies, and the tissues are approximated. **V,** Final abutments are inserted into the implant bodies after bone maturation. **W,** Three splinted crowns are supported by the three implants. **X,** A panoramic radiograph of the sinus graft, three posterior implants, and the implant prosthesis.

punch is placed along the periphery of the access window and tapped again. If the window does not separate easily, the punch is rotated so that only an edge comes in contact with the scored line. This decreases the surface area of the punch against the score line of the window and increases the stress against the bone. Another light tap with the mallet will most likely cause greenstick fracture of the bone along the scored line. If this still does not free the window, then further scoring of the bone with the hand piece and diamond bur is indicated, and the tapping procedure is repeated (see Figure 38-30, *E*).

A short-bladed soft tissue curette designed with two right-angle bends is introduced along the margin of the window (i.e., BioHorizons Sinus Currette No. 1). The curved portion is placed against the window, whereas the sharp edge is placed between the sinus membrane and the margin of the inner wall of the antrum for a

WWW.HIGHDENT.IR

همیار دندانسازان

depth of 2 to 4 mm. If any sharp edges of bone remain on the bone's margin, then they may be flicked off with the curette. The curette is slid along the bone margin, 360 degrees around the access window. This ensures the release of the membrane from the surrounding walls of the sinus without tearing from the sharp bony access margins. The sinus membrane may be elevated from the antral walls easily, because it has few elastic fibers and is not attached to the cortical wall. Specially designed and shaped curettes are available to facilitate this maneuver (see Figure 38-30, *F*).

A larger curved periosteal or sinus membrane elevator is then introduced through the lateral-access window along the inferior border (i.e., BioHorizons Sinus Currette No. 2). Once again, the curved portion is placed against the window, and the sharp margin of the curette is dragged along the floor of the atrum, while elevating the sinus membrane. The curette should always be maintained on the bony floor to avoid a membrane perforation. The curette is never blindly placed into the access window. The surgeon should see and/or feel the curette against the antral floor or sinus walls at all times. Once the mucosa on the antral floor is elevated, the lateral, distal, and medial wall of the sinus is addressed. The curette is pushed against the bone that easily reflects the membrane. The sinus membrane is inspected for perforations or openings into the antrum proper (see Figure 38-30, *G* to *J*).

It is easier to gain direct vision and access to the distal portions of the antrum than the anterior portions when the sinus area expands beyond the access window. Therefore whenever the periosteal elevator or curette cannot stay against the bone with good access in the anterior area, the access window should be increased in size toward the anterior. A Kerrison rongeur or a preparation similar to the initial score-and-fracture technique may be used to expand the size of the access window.

The periosteal elevators and curettes further reflect the membrane off the anterior vertical wall, floor, and medial vertical wall to a height of at least 16 mm from the crest of the ridge, or approximately 8 to 11 mm in an SA-3 procedure. It is better to err on the high side to ensure that ideal implant height may be placed without compromise (always making sure the ostium remains unobstructed). The lateral-access window is positioned as part of the superior wall of the graft site, once in final position. The SA space has the original sinus floor as the base; the posterior antral wall, medial antral wall, and anterior antral wall as its sides; and the lateral-access window and elevated sinus mucosa as its superior wall.

Sinus Graft: Layered Approach

The Top Layer: Collagen and Antibiotic. A resorbable collagen membrane (CollaTape) soaked with a parental form of antibiotic (Ancef 0.2 mL or Cleocin 0.2 mL) is then prepared. The collagen and antibiotic is placed onto the elevated antral floor region and attaches to the sinus mucosa on the superior part of the graft site.

Box 38-5 Sinus Graft Layers

Superior

Collagen membrane
Antibiotic medication

Intermediate

Calcium phosphate (70% by volume) (cortical freeze dried bone [FDBA], MinerOss) or xenograft (microporous OsteoGraf N-300, or Bio-Oss)
Demineralized freeze-dried bone (DFDB) (30% by volume), i.e., cortical fiber (Grafton)
Platelet-rich plasma (PRP) from 10 to 20 cc of whole blood
Antibiotic, i.e., Ancef 500 mg/mL, 0.8 mL, or Cleocin 150 mg/mL, 0.8 mL

Inferior

Regional acceleratory phenomenon
Autogenous bone

Ideally the lateral-access window, which now is part of the superior limit, is not covered with the collagen. The collagen is a carrier for the antibiotic to decrease the risk of postoperative infection. In addition, in case of membrane tearing or separation of the sinus mucosa (with or without the awareness of the surgeon) the collagen membrane seals the opening (see Figure 38-30, *K*; Box 38-5).

The Second Layer: Sinus Graft Materials. The author and several others have investigated the components of the sinus graft using animal research clinical biopsies, case series studies, and reentry observations. Several graft materials have been proposed in single use or mixes of different combinations, which include autogenous bone,* demineralized freeze-dried bone (DFDB) powder or cortical fibers,[14,149-153] freeze-dried bone (FDBA),[33,34,154,155] β-tricalcium phosphate (β-TCP),[6,32,64,81] xenograft hydroxyapatite (HA) (bovine anorganic bone),† calcium carbonates (bioactive glass),[161-162] and all combinations of these.‡ Recently, additional research has been focusing on combining "traditional" bone substitutes with bone growth factors.[28,155,171-174] Each graft material presents a similar, yet distinct, biological approach to the sinus graft.

DFDB has minute amounts of osteoinductive material capable of inducing some undifferentiated mesenchymal cells to form osteoblasts. The mechanism for this process appears to relate to the bone morphogenic protein found primarily in cortical bone. The ideal size and shape appears to be fibers of cortical bone (Grafton). In animal and human studies, demineralized freeze-dried

*References 5, 8, 10, 18, 19, 22, 137-148.
†References 14, 18, 20, 30, 142, 156-160.
‡References 6, 8, 9, 28, 163, 164-170.

WWW.HIGHDENT.IR

bone allograft (DFDBA) powder used alone in sinus grafts did not provide a satisfactory result, and the author does not use it currently. Bone was present, but not in as much volume as the graft material originally placed. Speculation exists that the material resorbes more rapidly than the bone formation process, resulting in lesser volume of bone. In addition, when DFDB is placed into an area of low-oxygen tension (hypoxic or hypocellular tissue), the material results in fibrous or cartilage tissue rather than bone.[175] Other authors have observed similar conclusions on the poor performance of DFDB used alone in animal and human studies.[152,165,167,176,177] At the Sinus Graft Consensus Conference,[13] high success rates were reported for all materials and combinations, with the exception of DFDB when used alone.

The DFDB portion of the graft appears primarily to be a source of type I collagen and a small secondary response of BMP for undifferentiated cells. It may resorb and permit blood vessels to invade the graft. Its combination with autogenous bone allows bone formation from phase I or phase II simultaneously. Researchers have postulated that this may produce more bone than when either material is used alone.[164,178-180] In the graft technique the author uses, the DFDB cortical fibers are mixed with another graft material (e.g., microporous HA or a mineralized bone allograft) and platelet-rich plasma (PRP) (not with whole blood or anesthetic solution). The toxic by-products of blood catabolism[181] and the acidic pH of anesthetic may decrease bone formation. Whole blood is drawn (10 to 20 mL) from the patient and placed into a centrifuge for approximately 10 minutes at 3400 to 5600 rpm. The blood is separated into three layers: (1) red blood cells, (2) a buffy coat of platelets and white blood cells, and (3) serum. The buffy coat and serum is then centrifuged again to increase the concentration of platelets. Platelets contain platelet-derived growth factors (PDGFs), which are involved in the cascade of bone mineralization.[28,155,171] The PRP is added to the DFDB cortical fibers and osteoconductive ceramic graft material in the intermediate layer of the sinus graft. An antibiotic is also added to the graft material when used for sinus grafts.

Osteoconduction describes the ability of a material to permit bone to grow in its presence. An osteoconductive material does not grow bone in the absence of bone or differentiated mesenchymal cells. Therefore these materials act more as fillers and may help form a future bone matrix or maintain volume and consistency for surgical placement. Microporous HA may include mixtures of calcified or mineralized cortical or trabecular bone, β-TCP, $CaPO_4$, and xenographic bone and are all osteoconductive bone graft materials.

A histomorphometric study by Froum et al.[182] at 26 to 32 weeks after grafting evaluated mineralized cancellous bone allograft (MCBA) and anorganic bovine bone material (ABBM) for sinus augmentation. Bilateral sinus grafts, one filled with MCBA and the other with ABBM, were compared. The average vital bone content of the MCBA was 28.25%; the ABBM was only 12.44%. This report is similar to studies by the author; however, when a layered approach to sinus grafting includes autogenous bone at the most inferior layer, a greater vital bone percent is obtained.[179]

The osteoconductive portion of the graft is mixed with an antibiotic for sinus grafts, because the risk of infection is greater than for most bone-grafting procedures. A parental form of antibiotic is used rather than a tablet form, because oral antibiotic drugs often have fillers in the product that are not osteoconductive. The most common antibiotic is Ancef 500 mg/mL, and 0.8 mL of solution is added to the graft.

In sinus grafts the second layer of the graft consists of either microporous HA, inorganic bovine bone, or mineralized FDBA (70%) mixed with 30% DFDB (cortical fibers), the PRP obtained from 10 cc of whole blood, and a parental form of antibiotic. The mixture forms the intermediate layer of the sinus graft, and it is placed below the collagen and antibiotic (which is on the top) and the autogenous bone on the original sinus floor on the bottom. These materials are mixed together, placed in an open 3-mL syringe that has its end removed, and placed in the sinus graft site with a forward and inferior packing motion. Because the membrane has been elevated, the sinus graft mixture can be introduced below it and condensed into position. Packing is firm but not excessive, because perforation of the medial wall is possible and spaces for blood vessels must be present to grow to form new bone (see Figure 38-30, *N* and *O*).

The Bottom Layer: Regional Acceleratory Phenomenon. Once the sinus membrane has been elevated and collagen membrane plus antibiotic has been inserted into the superior aspect of the graft site, the original antral floor and anterior wall are scratched with a sharp hand instrument. This trauma sets up a regional acceleratory phenomenon (RAP) that introduces more growth factors into the site and helps blood vessels from the bony walls to grow into the graft. These vessels allow migration of osteoclasts and osteoblasts that resorb and replace the graft with living bone. In addition, the blood vessels provide blood supply to the autologous bone portion of the graft, required for initial osteogenesis.

The medial wall and posterior wall are not aggressively scratched; because they are so thin, perforation may occur (see Figure 38-30, *L*).

The size of the antrum in the lower one third is evaluated once the membrane has been elevated. The lateral wall to medial nasal wall may be less than 10 mm (small), 10 to 20 mm (average), or greater than 20 mm (large) and noted in the surgical report. This can be correlated with the rate of graft transformation because

WWW.HIGHDENT.IR

همیار دندانسازان

the new bone forms from the surrounding walls of bone around the graft site. In general, the bone graft maturation time varies from 4 to 10 months, depending on type of graft material and size of the graft site mediolateral and anteroposterior (A-P) dimension.

Autologous Bone. An osteogenic material is capable of producing bone, even in the absence of local undifferentiated mesenchymal cells. Autologous bone predictably exhibits this activity in the sinus graft. Tatum[3,4] first developed and reported the use of autogenous bone for sinus grafts in the 1970s, and Boyne and James[5] first published the information in 1980. In primates *(Macaca fascicularis)*, Misch[178,179] found the use of iliac crest or tail bone in sinus grafts produced bone slightly denser than typical in the region, as evidenced from histology sections harvested at the reentry procedure. Similar findings have been observed during case series studies with patients undergoing sinus grafts with autologous bone from the iliac crest or intraoral donor sites.[148,152,165]

It is interesting to note that sinus grafts in the literature that have used all autogenous bone have lower success rates than sinus grafts with synthetic substitutes (e.g., Del Fabbro et al.[29] reported 87.70% versus 95.98%).[8,29,36,139,137] However, these reports are misleading. The sinus graft success is linked to the success rate of the implants inserted. Those articles that used autologous bone as the sinus graft material most often used a machine surface condition implant, whereas those articles with only bone substitutes most often used a rough implant surface condition. When success rates of implants in a sinus graft relative to implant surface condition are evaluated, smooth-surface implants were 85.64% successful versus rough surfaces at 95.98%. Therefore autogenous bone is no worse than bone substitutes as a graft material in the sinus; however, it cannot be said to be better.

All the grafts studied by the author have included at least some autogenous bone. The Tatum surgical approach, using a portion of the lateral maxilla attached to the sinus membrane to gain access to the graft region, results in at least some autogenous cortical bone in the graft. Whether the lateral-wall cortical bone is an actual medium for bone growth has not been determined, but this is a reasonable speculation. In addition, an autograft is harvested and placed on the original sinus floor. The author has performed reentry of more than 1500 sinus grafts (at implant placement) accompanied by more than 50 human histologic sections and 18 primate sinus grafts and histology. A consistent histologic and clinical finding is that bone grows into the SA region from the surrounding walls of the maxillary antrum where the sinus membrane was elevated.[178,179,183] In other words, the bone growth came from the surrounding walls of bone, similar to an extraction socket. The last regions to form bone are usually the center of the lateral-access window and the region under the elevated sinus membrane. In fact, no new bone at time intervals up to 12 months was found to grow immediately under the sinus membrane.

As a result of these observations, Misch altered the surgical approach and alloplastic and/or autologous graft placement position after 1987. Since then, the sinus membrane of the medial wall of the antrum is consistently elevated to the height of the SA augmentation. This provides an additional wall for host bone to help new bone formation. The sinus membrane of the anterior wall, and usually the posterior wall, are also elevated to the expected height of the sinus graft.

In addition to the lateral-access window, as much autogenous bone as practical is harvested and used in the graft. The most common harvest site for the SA-3 approach is the tuberosity on the same side of the patient that the sinus is being augmented. In this way, an additional surgical site is not required. Additional sources of bone to be added to the graft site may be any debris from implant osteotomies, bone cores over the roots of anterior teeth, sinus exostoses, and cores from the mandibular symphysis or ramus region. The autogenous bone is placed on the original bony floor in the area most indicated for implant insertion. A blood supply from the host bone can be established earlier to this grafted bone and maintains the viability of the transplanted bone cells and the osteogenic potential of the transplanted bone growth factors. Cancellous bone is best used after compaction of the graft to increase the cell volume.[181,183] Autogenous bone represents an important component of the sinus graft, and harvesting of at least some autogenous bone is highly encouraged.

It should be noted that the autograft layer is not mixed with any bone substitute material. When mixed with other materials, it is isolated farther from any initial source of blood vessels; as a consequence, it may not remain vital. Although the release of growth factors, space maintenance, and a calcium source are still of benefit, the primary advantage of osteogenesis is lost when the autogenous bone is mixed with other graft materials.

Once the middle layer of the graft has been placed, the maxillary tuberosity is completely exposed. The SA-3 posterior anatomy most always provides substantial amounts of autogenous bone to harvest. A rotary bur separates the tuberosity from the palatine process and sections the crestal bone distal to the second molar. A curved osteotome and mallet then separate the intact tuberosity. Another option is to harvest the tuberosity with a rongeur or trephine bur. The tuberosity bone is usually soft and therefore is compressed to form more cells per volume. Then it is used in the most inferior portion of the sinus graft, on top of the original antral floor. Additional autogenous bone may be harvested intra- or extraorally as indicated on a case-by-case basis (see Figure 38-30, *P*).

A multilayered graft is therefore placed into the antrum (see Figure 38-30, *Q*). The first material intro-

WWW.HIGHDENT.IR

Box 38-6 Sinus Graft: Immediate and Delayed Implant Placement

Immediate Implant Placement (SA-3I)

When the endosteal implants are inserted at the same time as the sinus graft, the following conditions should exist:

1. Greater than 5 mm bone height
2. Greater than 6 mm bone width
3. D3 bone quality or better
4. No sinus pathologic condition
5. No history of recurrent sinusitis
6. No relative contraindications
7. No or small sinus membrane tear during surgery, completely sealed with collagen
8. No parafunction on removable soft tissue–borne prosthesis

Delayed Implant Placement (SA-3)

Implants should not be inserted at the same time as the sinus graft when the following conditions exist:

1. Less than 6 mm bone width
2. D4 bone quality
3. Treated sinus pathologic condition within last few months
4. History of recurrent sinusitis (especially when treated with recurrent antibiotic medications)
5. Relative contraindications (smoking, medically compromised patient)
6. Medium to large tear in the sinus membrane during the graft surgery
7. Parafunction on overlying removable prosthesis

duced is a collagen membrane and antibiotic, which forms the superior layer of the graft. The next layer (and often the largest) is a 70% mixture of $CaPO_4$ (usually cortical FDBA (MinerOss) or a xenograft microporous HA (OsteoGraft N-300 or Bio-Oss), a 30% volume of DFDB cortical fiber (Grafton), PRP from 10 to 20 cc of whole blood, and antibiotic (Ancef 500 mg/mL, 0.8 mL, or Cleocin 150 mg/mL, 0.8 mL), which acts to form bone through a combination of osteoinduction and osteoconduction pathways. The last material introduced into the antrum in its most inferior position is autogenous bone, usually harvested from the tuberosity. This osteogenic portion of the graft is typically placed in the more anterior regions or in critical future implant sites.

Access Window. A resorbable membrane may be placed over the lateral-access window (see Figure 38-30, *R*). This delays the invasion of fibrous tissue into the graft and enhances the repair of the lateral bony wall. A nonresorbable membrane is not used because it may complicate the treatment of a postoperative sinus infection. A bacterial smear layer may accumulate in the nonresorbable material and contribute to the infection process. An infection with a resorbable membrane is less of an issue, because the lower pH of the infection resorbs the membrane rapidly. PRP is placed over the lateral collagen membrane to increase the amount of growth factors for bone formation and to increase the growth factors for tissue healing. If inadequate PRP is available because it was used in the second layer of the graft, then PPP may be used.

Froum et al.[182] evaluated sinus grafts with barrier membranes over the lateral-access wall compared with no barrier membrane. All sinus graft combinations in the study demonstrated higher vital bone percent on the cores when a barrier membrane was used. Misch observed a higher vital bone percent even when collagen was used over the lateral-access site compared with no collagen. Therefore either a collagen material (CollaTape), when autograft is used, or collagen barrier membrane (BioMend), when no or little autograft is available, should be placed over the lateral maxillary access window site.

Implant Insertion. A review of the literature by Del Fabbro et al.[29] notes success rates of implants placed at the same time as the graft have a survival rate of 92.17%, whereas a delayed implant insertion has a survival rate of 92.93%. The 5 to 10 mm of initial bone height in a SA-3 posterior maxilla, the cortical bone on the residual crest, and the cortical-like bone on the original antral floor may stabilize an implant that is inserted at the time of the graft and permit its rigid fixation. Therefore an endosteal implant may be inserted at this appointment and has been advocated for some years in the past by this author[69] and others.* Therefore when the conditions are ideal for the SA-3 sinus graft, the implant may be inserted (see Figure 38-30, *S*; Box 38-6).

The ideal conditions for implant insertion at the same time as the sinus graft include the following: (1) greater than 5 mm of bone below the antrum, (2) greater than 6 mm width of crestal bone, (3) D2 or D3 bone in the region, (4) no pathologic condition of the sinus is present, (5) no history of recurrent sinusitis is given by the patient, (6) no relative contraindications exist (e.g., smoking, ASA 3 patients with lowered immune responses), (7) no or only a small tear in the sinus membrane during the procedure, and (8) no parafunction on an overlying soft tissue–borne prosthesis.

Although the implant may be inserted at the same time as the sinus graft, several advantages exist to delaying implant placement for approximately 2 to 4 months:

1. The individual rate of healing of the graft may be assessed after 2 to 4 months while the implant osteotomy is being prepared and the implant

*References 10, 13, 36, 127, 128, 176.

WWW.HIGHDENT.IR

همیار دندانسازان

inserted. The healing time for the implant is no longer arbitrary, but it is more patient specific.

2. Under ideal conditions, postoperative sinus graft infections occur in approximately 3% to 5% of patients, which is greater than the percentage for implant placement surgery or intraoral onlay bone grafts. If the sinus graft becomes infected with an implant in place, then a bacterial smear layer may develop on the implant and make future bone contact with the implant less predictable. The infection is also more difficult to treat when the implants are in place and may result in greater resorption of the graft as a consequence. If the infection cannot be adequately treated, then the graft and implant must be removed. Therefore a decreased risk of losing the graft and implant exists if a postoperative infection occurs with a delayed implant insertion. Some reports in the literature indicate a slightly higher failure rate of implants when inserted simultaneously compared with a delayed approach.
3. Blood vessels within the graft are required to form and remodel bone. An implant in the middle of the sinus graft does not provide a source of blood vessels. It may even impair the vascular supply.
4. Bone width augmentation may be indicated in conjunction with sinus grafts to restore proper maxillomandibular ridge relationships and/or increase the implant diameter in the molar region. Augmentation may be performed simultaneously with the sinus graft. As a result, larger-diameter implants may be placed with the delayed technique.
5. The bone in the sinus graft is denser with the delayed implant placement. As such, implant angulation and position may be improved because it is not dictated by existing anatomical limitations at the time of the sinus graft.
6. The surgeon may access the sinus graft before implant insertion. On occasion, the sinus graft underfills a region, and the lack of awareness of the condition during implant insertion at the same time results in an implant placed in the sinus proper, rather than the graft site.
7. On reentry to a sinus graft, it is not unusual to observe a craterlike formation in the center of the lateral-access window, with soft tissue invagination. If the implant is already in place, then it may be difficult to remove the soft tissue and assess its precise extent. When soft tissue is present at a delayed implant insertion, the region is curetted and replaced with a bone graft before implant placement. The healing time for the implant is related to the developing bone assessed at the delayed surgery, not an arbitrary period that may be, on occasion, too brief.

The primary disadvantage of delaying the implant placement is the need for an additional surgery. If overall treatment time is a significant factor for the patient, then the implant may be inserted after 2 months, which considerably reduces the risk of infection after implant insertions. Otherwise, 2 to 4 months or more is typical for the reentry and implant insertion after a SA-3 technique (see Table 38-2).

Soft Tissue Closure. The soft tissues and periosteum should be reapproximated for primary closure without tension, with care to eliminate graft particles in the incision line. However, because a collagen membrane is placed over the lateral-access window, the tissues often will not reapproximate without tension. Therefore the facial flap must often be expanded. A tissue pickup holds the facial flap to the height of the mucogingival tissues junction. The flap is then elevated, and a No. 15 blade is used to incise the tissue 1 mm deep through the periosteum above the mucoperiosteum. Tissue scissors are then introduced into the incision parallel to the facial flap at a depth of 3 to 5 mm. A blunt dissection under the flap releases the periosteum and muscle attachments to the base of the facial flap. The flap may then be advanced over the graft site to the palatal tissues.

It should be noted that horizontal vascular anastomoses occur lateral to the maxilla, within the soft tissue, and approximately 20 mm above the crest of the ridge. A blunt dissection does not violate these vessels. No tension should exist on the facial flap with primary closure of the site. Interrupted horizontal mattress or a continuous suture (3-0 polyglycolic acid [PGA]) may be placed. Suturing is more critical with this procedure than with many other implant placements. Incision line opening may contribute to infection, contamination, or loss of graft materials. The borders and flange of an overlaying soft tissue–borne denture or partial denture are aggressively relieved to eliminate pressure against the lateral wall of the maxilla.

Subantral Option Four: Sinus Graft Healing and Extended Delay of Implant Insertion

In the fourth option for implant treatment of the posterior maxilla, SA-4, the SA region for future endosteal implant insertion is first augmented. This option is indicated when less than 5 mm remains between the residual crest of bone and the floor of the maxillary sinus (Figure 38-31). The SA-4 corresponds to a larger antrum and minimal host bone on the lateral, anterior, and distal regions of the graft, because the antrum generally has expanded more aggressively into these regions. The inadequate vertical bone in these conditions decreases the predictable placement of an implant at the same time as the sinus graft, and less recipient bone exists to act as a vascular bed for the graft (see Figure 30-8). In addition, less autologous bone exists in the tuberosity for harvesting, and fewer septa or webs usually exist in the sinus (and typically exhibit longer mediodistal and

WWW.HIGHDENT.IR

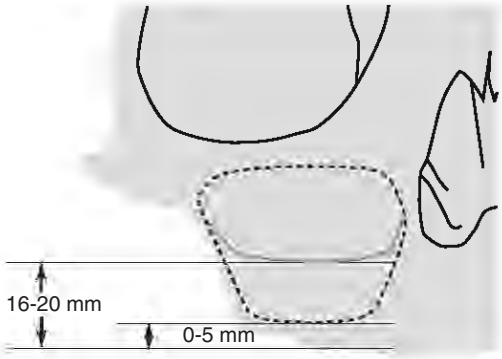


Figure 38-31 The subantral (SA)-4 classification has 0 to 5 mm of bone below the maxillary sinus floor. A sinus graft before an extended delay and before implant placement is in order.

wider lateromedial dimensions). Therefore the fewer bony walls, less favorable vascular bed, minimal local autologous bone, and larger graft volume all mandate a longer healing period and slightly altered surgical approach.

The Tatum lateral-wall approach for sinus graft is performed as in the previous SA-3 procedure (Figure 38-32). Most SA-4 regions provide better surgical access than the SA-3 counterparts because the antrum floor is closer to the crest, compared with the SA-3 maxilla. However, in Division D maxillae it is usually necessary to expose the lateral maxilla and the zygomatic arch. The access window in the severely atrophic maxilla may even be designed in the zygomatic arch. The medial wall of the sinus membrane is elevated at least 16 mm from the crest so that adequate height is available for future endosteal implant placement. This means the elevation is 11 to 16 mm from the sinus floor. The combination of graft materials used and their placement are similar to those for the SA-3 technique. However, because less autogenous bone is often harvested from the tuberosity, an additional harvest site may be required, most often above the roots of the maxillary premolars or from the mandible (i.e., from the ascending ramus).

The width of the host site for most edentulous posterior maxillae is Division A. However, when Division C–w to B exists, an onlay graft for width is indicated. When the graft cannot be secured to the host bone, it is often better to perform the sinus graft 6 to 9 months before the autogenous graft for width. Then after the onlay graft maturation, the implants may be inserted.

VASCULAR HEALING OF THE GRAFT

Healing of the sinus graft takes place by several vascular routes, including the endosseous vascular anastamosis and the vasculature of the sinus membrane from the sphenopalatine artery. In mildly resorbed ridges, the host bone receives its blood supply from both centromedullary and mucoperisosteal vessels. However, as age and the resorption process increases, the bone gradually becomes totally dependant on the mucoperiosteum for the blood supply. The periphery of the graft is mainly supplied by vessels of the sinus membrane and by intraosseous vascular bundles. The central portions of the graft receive blood from collateral branches of the endosseous anastomosis. The extraosseous vascular anastomosis may enter the graft from the lateral-access window.

Many local variables are related to sinus graft maturation, including healing time, the volume of the SA graft, the distance from the lateral to medial wall (small, average, or large), and the amount of autologous bone in the multilayered approach, all of which relate to the speed and amount of new bone formation.

The time of evaluation of the sinus graft is perhaps the greatest variable of all. When the sinus graft is reentered at 2 months, the amount of new bone is minimal. At 4 to 6 months, more new bone in the sinus graft is observed; at 8 to 10 months, more vital bone is evident.* Froum et al.[182] evaluated a sinus graft from the same patient at 4 months, 6 months, 12 months, and 20 months. The amount of new bone continuously increased, compared with the amount of graft material in the antrum. In addition, the additional time allowed the graft to mature into a type of bone typical of the region. It is clear that the more time that elapsed from sinus graft to implant loading, the more vital bone was available to support the occlusal load. Unfortunately, no study is available to date to determine when the ideal bone volume and quality is obtained in a sinus graft or lift relative to elapsed time from initial surgery.

The materials in the sinus graft affect the rate of bone formation. Bone formation is fastest and most complete within the first 4 to 6 months with autogenous bone, followed by the combination of autogenous bone, porous HA, and DFDB (6 to 10 months); alloplasts only (i.e., TCP) may take 24 months to form bone.† In 2003, Merkx et al.[185] reported more new bone formed in a shorter time frame with autogenous bone, whereas HA and bone allograft gave lower vital bone percent values.

A sinus graft site, which is the inferior third of the maxillary sinus, may be 5 to 30 mm in mediolateral depth and 5 to 40 mm in mesiodistal length. The time required to form new bone in this region is related to the volume of the graft. Therefore the time required before implant insertion for SA-4 or implant uncovery in SA-3 is dependent on the volume of the sinus graft. Delayed implant placement for 4 months or more in a small sinus permits the evaluation of the graft and indicates the further time required for direct bone fixation of the implant. If the sinus graft volume is moderate in size, then 6 months before reentry is

*References 27, 160, 169, 170, 182, 183.
†References 77-78, 80, 85, 89, 160, 165, 166, 170, 177, 182-184.

WWW.HIGHDENT.IR

همیار دندانسازان

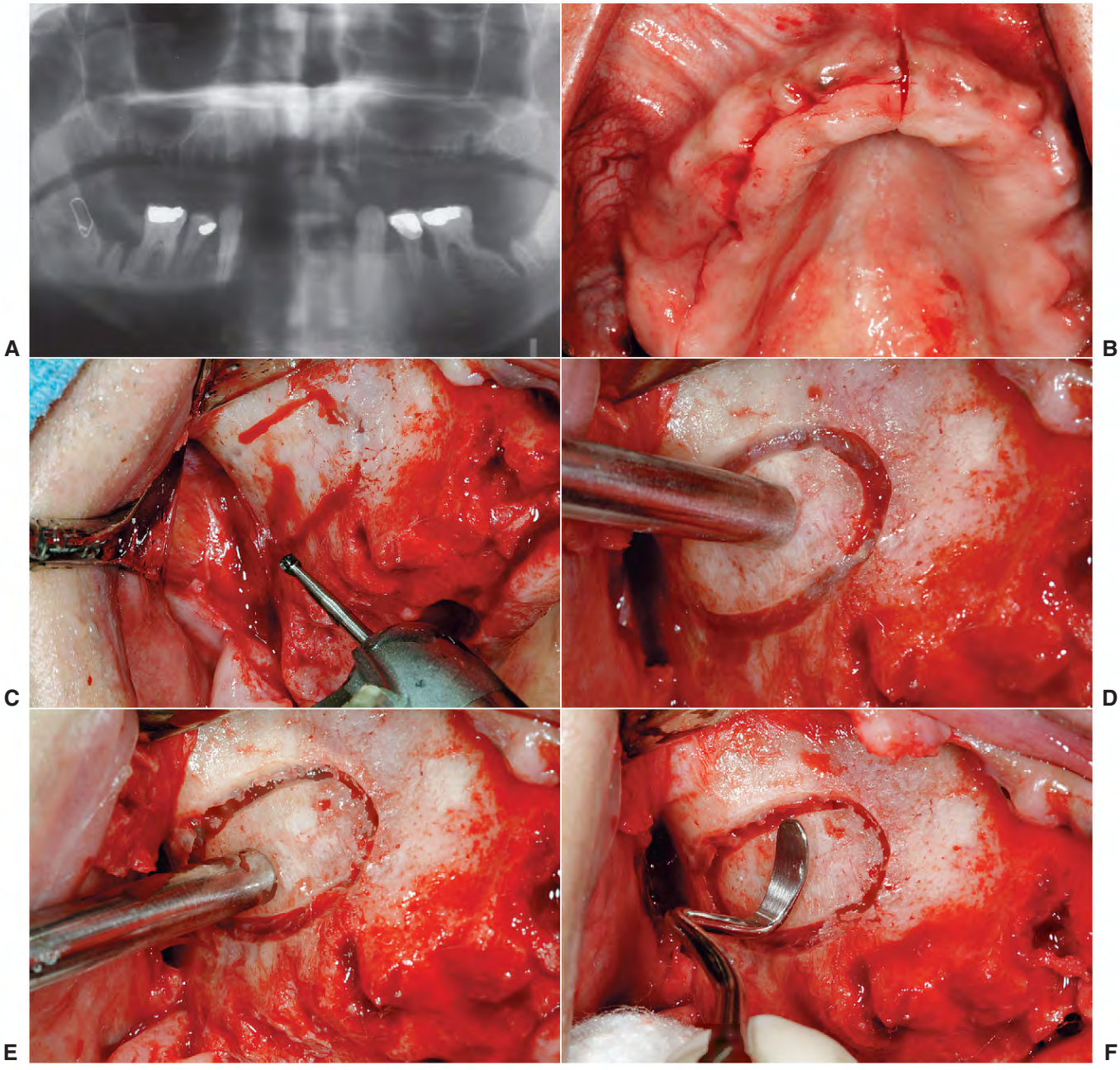


Figure 38-32 **A,** A panoramic radiograph of a completely edentulous maxilla with less than 5 mm of available bone height below the maxillary sinuses (subantral [SA]-4 category). **B,** A crestal incision is made slightly toward the palate and extends from the maxillary right tuberosity region to the midline. Vertical-release incisions are made at both ends of the crestal incision. **C,** A No. 6 round carbide bur with an extended shank in a 5:1 hand piece is used to score the lateral maxilla and represents the initial outline of the Tatum lateral-access window to the maxillary sinus. **D,** A blunt instrument is placed on the lateral-access window and gently tapped with a mallet to greenstick fracture the access window from the lateral maxilla. **E,** The window separates from the lateral maxilla and may be depressed 1 to 2 mm toward the antrum. Because the window is large, a reduced amount of stress is applied to the sinus membrane. Because the sinus membrane has few elastic fibers connected to bone, the membrane is elevated from the rim of the access window. **F,** The round back of the double-action sinus curette is placed against and depresses the access window 1 to 2 mm. *Continued*

suggested. A large antrum (>20 mm wide and >30 mm A-P) may require 8 months before the implant insertion or uncovery. If the bone density was D4 at implant placement, the graft site was large, and/or little autologous bone was used in the graft, then the time before uncovery or placement of the implants may be as long as 10 months.

POSTOPERATIVE INSTRUCTIONS

The postoperative instructions are similar to those for most oral surgery procedures that include sinus manipulation. Rest, ice, pressure, and elevation of the head are particularly important. Strict adherence to the pharmacologic protocol is of major importance.

WWW.HIGHDENT.IR

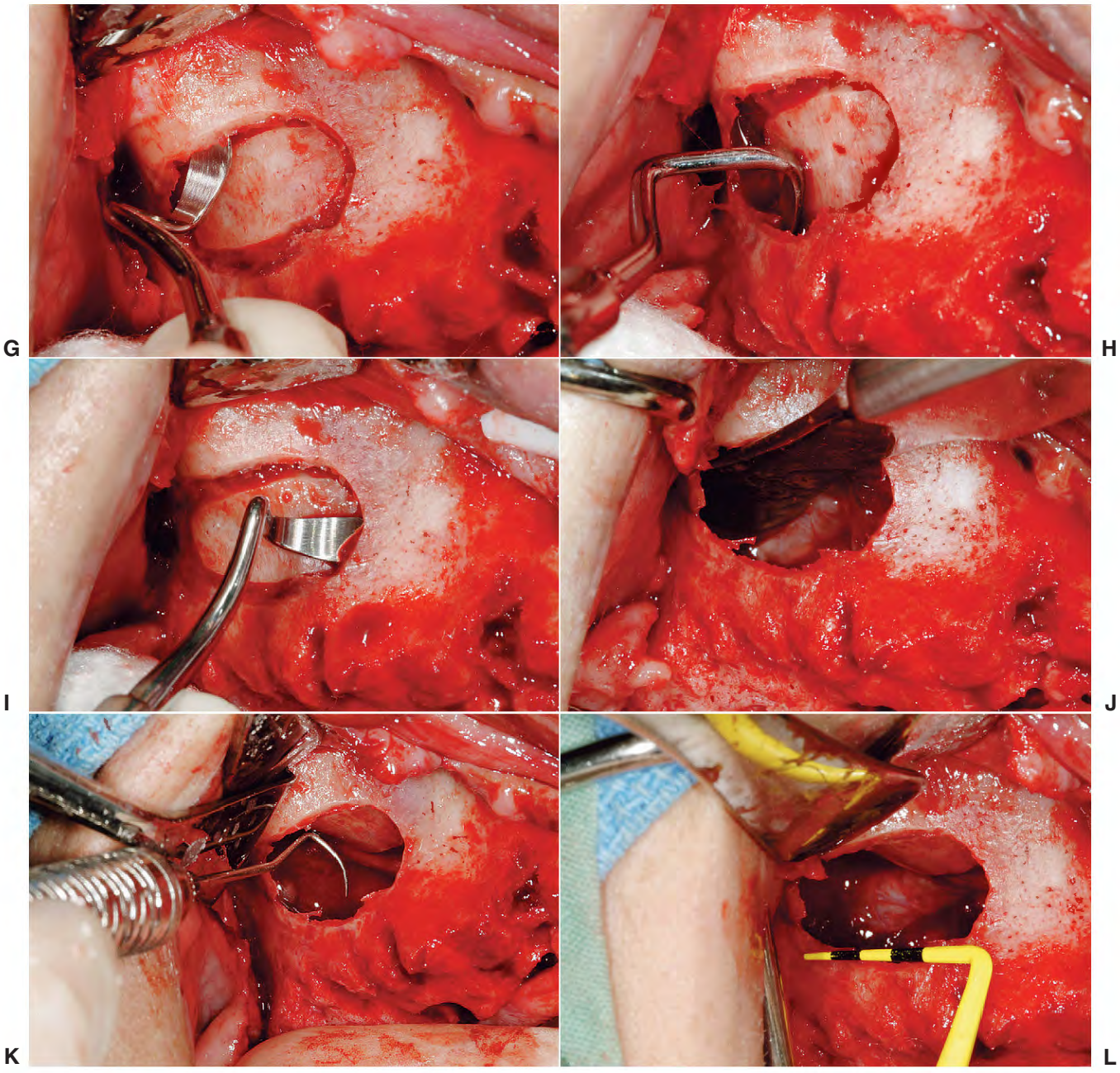


Figure 38-32, cont'd **G,** The blade of the sinus curette is slid along the inner bony wall a few millimeters and scrapes the sinus membrane along the top of the access window from the lateral wall of bone and around the sinus. **H,** The sinus curette slides along the floor of the antrum approximately 5 mm from the access window. **I,** The anterior portion of the access window is engaged. The curette must remain on bone. If it does not scratch the bony wall, then the membrane will be separated. The most common location of a sinus membrane tear is anterior to the access window, because direct vision of the membrane is not possible and the hand position to remain along the bone depends on the double bend of the sinus curette. **J,** The sinus mucosa is gently elevated off the medial wall of the maxillary sinus so that 16 mm of bony walls exist above the crest of the residual ridge. **K,** A sharp explorer is used to scratch the inner sinus bony walls below the elevated sinus mucosa. This increases the regional acceleratory phenomenon and helps to encourage blood vessels from the walls of bone to grow into the sinus graft. **L,** The length of the Tatum lateral-access window approximates 15 mm. The corners are round, rather than square or acute.

Continued

In addition, several other instructions are significant (Box 38-7). Although smoking is not an absolute contraindication for sinus grafting, smoking during the days immediately after the procedure is contraindicated because it may compromise the healing from both the intraoral and SA graft region.

In studies by Dietsh-Misch et al.[102] and Levin and Schwartz-Arad,[100] the risk of complications related to smoking and sinus grafts were reported. The complications of nonsmokers and smokers related to sinus graft procedures were similar (two infections out of 35 sinus grafts[102] in nonsmokers versus zero

WWW.HIGHDENT.IR

همیار دندانسازان

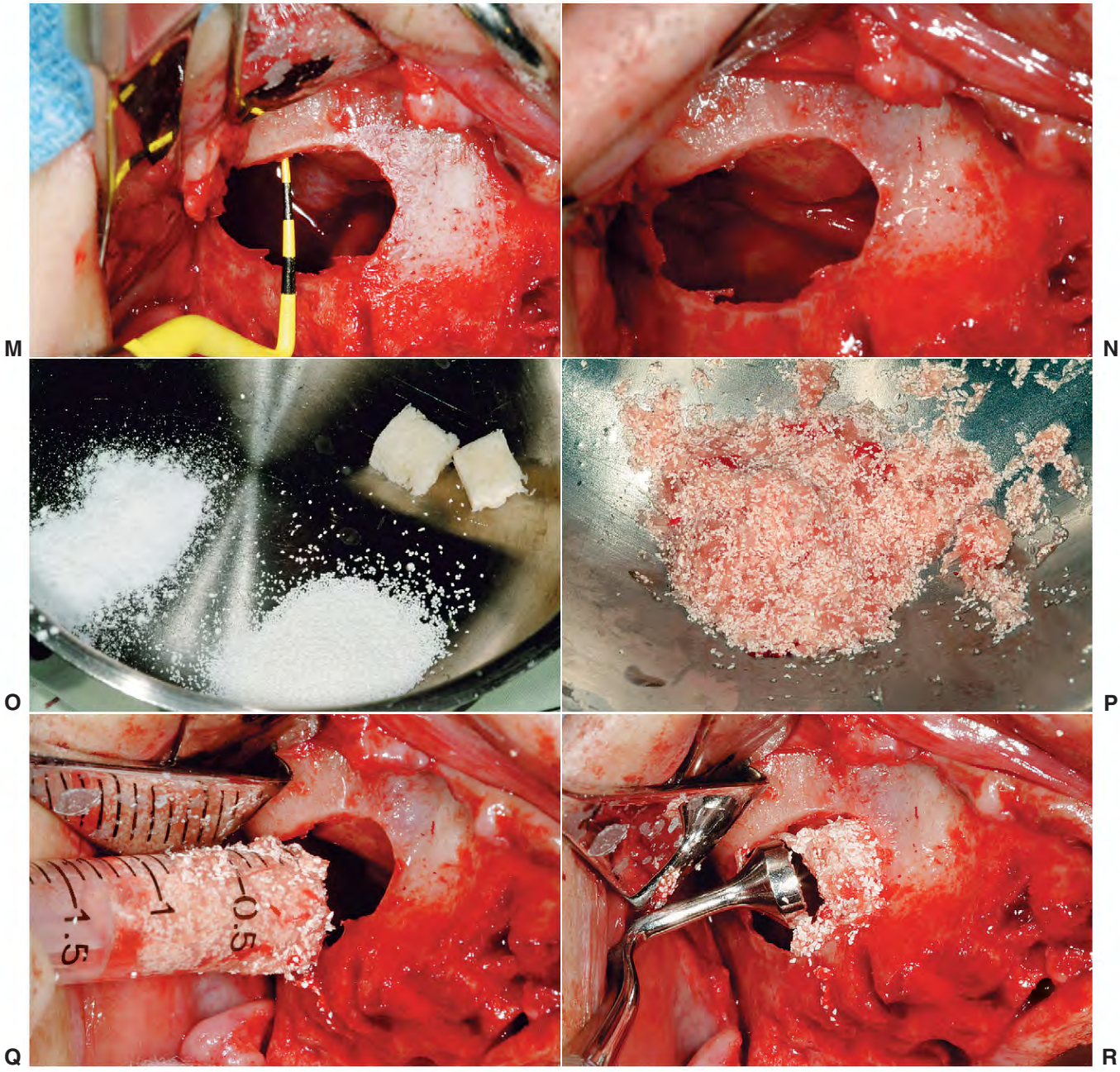


Figure 38-32, cont'd **M,** The lateral-access window starts a few millimeters above the antral floor, and the sinus mucosa is elevated so that 16 mm of height exists between the crest of the ridge and the height of the sinus elevation. **N,** The collagen and antibiotic is placed on the elevated sinus mucosa on the superior aspect of the graft site (layer 1). **O,** Demineralized freeze-dried bone fibers (Grafton plugs), mineralized bone (MinerOss, OsteoGraf/N, or Bio-Oss), and antibiotic are placed in a surgical tray. **P,** The platelet-rich plasma is mixed into the sinus graft mixture. **Q,** The graft material is introduced through the lateral-access window and into the space created for the sinus graft. **R,** A bone plugger pushes the sinus graft material distal and anterior to fill the space below the elevated sinus mucosa.

Continued

infections in 25 sinus grafts in smokers; 55.7% versus 63.3%, respectively).[100] A similar report by Peleg et al.[36] also found no statistical difference in implant failure rates in sinus grafts between smokers and nonsmokers up to a 9-year postoperative period. This is not to say smoking is not a concern. For the health of the patient and the potential risk of sinus infection, the patient is encouraged to cease smoking before, during, and after the sinus graft and implant insertion.

Blowing the nose and/or creating negative pressure while sucking through a straw or cigarette should also be eliminated for the week after surgery. Block and Kent[176] reported on a patient who lost the entire sinus graft 2 days after surgery from blowing the nose. Sneezing, if it occurs, should be done with the mouth open to relieve pressure within the sinus. Swelling of the region is common, but pain is usually less severe than after anterior implants in an edentulous mandible.

WWW.HIGHDENT.IR

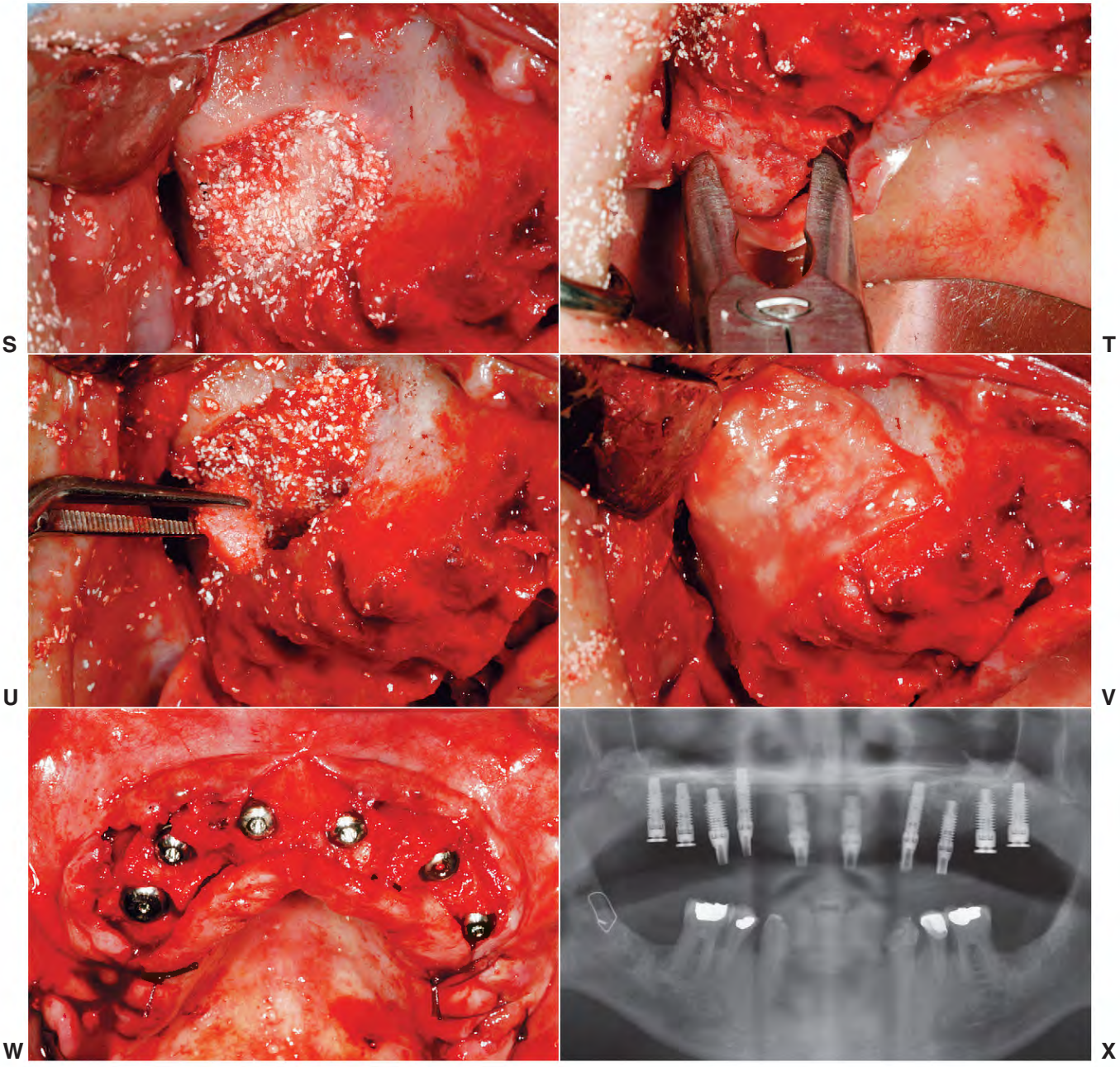


Figure 38-32, cont'd **S,** The sinus graft mixture fills the space of the sinus. This is the second layer of the sinus graft. **T,** The tuberosity on the same side as the sinus graft is harvested with a rongeur. **U,** The autograft from the tuberosity is placed in the bottom aspect of the graft and directly on the host bone. **V,** A piece of collagen, coated with platelet-poor plasma, covers the graft site and the lateral maxilla. **W,** First-stage cover screws are placed into the implants and inserted anterior to the sinus graft for a two-stage surgery. **X,** A panoramic radiograph after the bilateral sinus grafts matured for 6 months and four posterior implants were inserted.

Continued

In addition, the patient should be warned against lifting and pulling on the lip to observe the surgical site or during oral hygiene procedures, to reduce the risk of incision line opening. The patient should be notified that small bone particles or synthetic bone found in the mouth or expelled from the nose with bleeding is not unusual (see Box 38-7).

IMPLANT INSERTION

The implant surgery at reentry after successful sinus grafts is similar to SA-1, with a few exceptions. The periosteal flap on the lateral side is elevated to directly allow inspection of the previous access window of the sinus graft. The previous access window may appear

WWW.HIGHDENT.IR

همیار دندانسازان

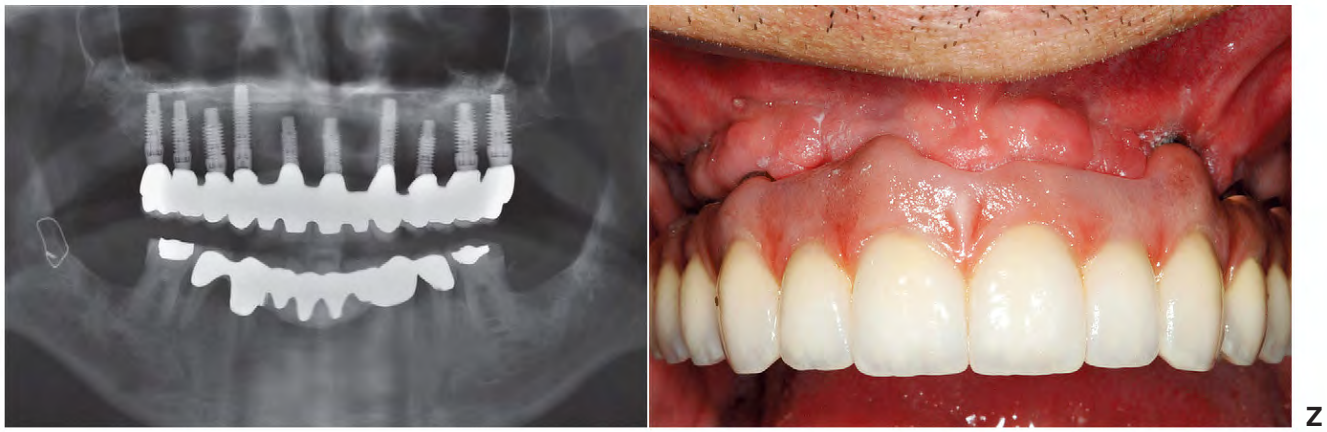


Figure 38-32, cont'd **Y,** A panoramic radiograph after the posterior implants healed 6 months and final restoration was fabricated. **Z,** An intraoral view of the final FP-3 porcelain-metal fixed prosthesis in the maxilla.

Box 38-7 Sinus Graft Postoperative Instructions

Sinus Elevation

1. *Do not* blow your nose.
2. *Do not* smoke or use smokeless tobacco.
3. *Do not* take in liquids through a straw.
4. *Do not* lift or pull on lip to look at sutures (stitches).
5. If you must sneeze, then do so with your mouth *open* to avoid *any* unnecessary pressure on the sinus area.
6. Take your medication as directed.
7. You may be aware of small granules in your mouth for the next few days. This is *not* unusual. Notify the office if the following occur:
   You feel granules in your nose.
   Your medications *do not* relieve your discomfort.

completely healed with bone, soft and filled with loose graft material, or with cone-shaped fibrous tissue ingrowth (with the base of the cone toward the lateral wall).

If the graft site on the lateral-access wall appears clinically as bone, then the implant osteotomy and placement follow the approach designated by the bone density. If soft tissue has proliferated into the access window from the lateral-tissue region, then it is curetted and removed. The region is again packed to a firm consistency with autologous bone from the previously augmented tuberosity, with DFDB cortical fibers (Grafton), and with resorbable HA (similar to the original graft). The implant osteotomy may then be prepared and the implant placed following the D4 bone protocol. An HA-coated, threaded implant offers an advantage when the bone density is D4 at implant insertion. Additional time (6 months or more) is allowed until the stage II implant uncovery is performed and progressive bone loading is used during prosthetic reconstruction.

The time interval for stage II uncovery and prosthetic procedures after implant insertion of a sinus graft is dependent on the density of bone at the reentry of implant placement (see Box 38-5). The crest of the ridge and the original antral floor may be the only cortical bone in the region for implant fixation. The most common bone density observed for a sinus graft reentry is D3 or D4. Most often, mineralized bone graft material in the sinus graft has not converted to bone. The tactile sense and the CT evaluation interprets the mineralized graft material as a more dense bone type. Therefore a tactile or radiographic D3 bone may actually be D4-like bone. Therefore it is prudent to wait longer (rather than shorter) for implant uncovery. As a result, the total time for SA-3 is 0 to 4 months before implant insertion and 4 to 6 months for uncovery, and an SA-4 sinus graft waits 4 to 6 months for implant insertion and another 4 to 8 for implant uncovery. Therefore the overall graft maturity time is 4 to 10 months for SA-3, and SA-4 healing time is 8 to 14 months before prosthetic reconstruction. Progressive loading after uncovery is most important when the bone is particularly soft and less dense. Inadequate bone formation after the sinus graft healing period of SA-4 surgery is a possible, but uncommon, complication. When observed, the SA-3 technique may be used to place additional SA graft before the implant placement surgery 4 months later.

INTRAOPERATIVE COMPLICATIONS RELATED TO SURGERY

Membrane Perforations

The most common complication during sinus graft surgery is tearing or creation of an opening in the sinus membrane (Box 38-8). This has several causes, which include a preexisting perforation, tearing during scoring of the lateral window, existing or previous pathologic condition, and elevation of the membrane from the

WWW.HIGHDENT.IR

Box 38-8 Perioperative Complications

Window

Too small—make another window
Too big—redesign
Septum—make two windows separated by septum

Membrane

Perforation—repair after elevation
- Small—collagen membrane (CollaTape)
- Large—slow resorbable membrane (BioMend)

Large polyp—curette out
Mucus retention cyst—drain, delay implant placement

Bleeding

Soft tissue of facial flap
Lateral bony wall of maxilla
Medial wall of sinus
Nares
Elevate head
Pressure against fourth cervical vertebra (C4) in neck
Collagen (Avitene)
Cauterization

bony walls. This complication occurs about 10% to 34% of the time. It has been reported with a higher frequency in smokers. If membrane perforation occurs more often than this, then the surgeon should give consideration to alter or reevaluate the surgical technique used in sinus grafting.

Sinus membrane perforation usually does not affect the sinus graft. However, in a report of the Sinus Consensus Conference of 1996, analysis of failed sinus grafts found 48% (79 of 164 failures) were attributed to perioperative complications, of which 48% (38) were found in patients that had sinus membrane perforations.[13] In an endoscopic evaluation after sinus grafts, macrolaceration of the sinus membrane resulted in a typical sinusitis appearance, even when clinical conditions of infection were not present.[186] Once the tear or perforation is identified, the continuation of the sinus elevation procedure is modified. The sinus membrane should be elevated off the bony walls of the antrum, despite the mucosal tear. If a portion of the membrane is not elevated away from a sinus wall, then the graft material will be placed on top of the membrane, thus preventing the bone graft from incorporating with the bony wall.

The perforation of the sinus membrane should be sealed to prevent contamination of the graft from the mucus and contents of the sinus proper and to prevent the graft material from extruding into the sinus proper. When graft materials enter the sinus proper, they may become sources for infection or may migrate and close off the ostium to the nose and create an environment for an infection.

However, numerous studies have shown a very low probability of sinus infections after perforations in the sinus membrane. Jensen et al.[10] reported that graft maturation occurred and no sinus infections were observed despite a 35% incidence of sinus perforation during the procedure in 98 patients.

The surgical correction of a perforation is initiated by elevating the sinus mucosal regions distal from the opening. Once the tissues are elevated away from the opening, the membrane elevation with a sinus curette should approach the tear from all sides so that the torn region may be elevated without increasing the opening size. The antral membrane elevation technique decreases the overall size of the antrum, thus "folding" the membrane over on itself and resulting in closure of the perforation. A piece of resorbable collagen membrane (e.g., CollaTape) is placed over the opening to ensure continuity of the sinus mucosa before the sinus bone graft is placed. The collagen sticks to the membrane and seals the SA space from the sinus proper (Figure 38-33).

If the sinus membrane tear is larger than 6 mm and cannot be closed off with the circumelevation approach, then a resorbable collagen membrane, but of a longer resorption cycle (BioMend), may be used to seal the opening. The remaining sinus mucosa is first elevated as described previously. A piece of collagen matrix is cut to cover the sinus tear opening and overlap the margins more than 5 mm. It should be noted that when a sinus tear occurs, it is sealed with a dry collagen membrane so that it may be rotated into the lateral-access opening, gently lifted to the mucosal tissue around the opening, and allowed to stick to the mucosa. Because no antibiotic is used on the collagen to make this procedure easier to perform, additional antibiotic is added to the graft material. Therefore 1 mL of Ancef 500 mg/mL or Cleocin 150 mg/mL is added to the graft material. Once the opening is sealed, the sinus graft procedure may be completed in routine fashion. However, care should be taken when packing the sinus with graft material. After a perforation, the graft is easily pushed through the collagen-sealed opening and into the sinus proper, and a periosteal elevator may be placed at the top of the graft site and under the perforation. The graft material is then gently inserted and pushed toward the sinus floor and sides but not toward the top of the graft (Figure 38-34).

A sinus perforation may cause an increased risk of short-term complications. A greater bacterial penetration risk exists into the graft material through the torn membrane. Therefore additional antibiotic is added to the particulate graft material. In addition, mucus may invade the graft and affect the amount of bone formation. Graft material may leak through the tear into the sinus proper, migrate to and through the ostium, and be eliminated through the nose or obstruct the ostium and prevent the normal sinus drainage.

WWW.HIGHDENT.IR

همیار دندانسازان

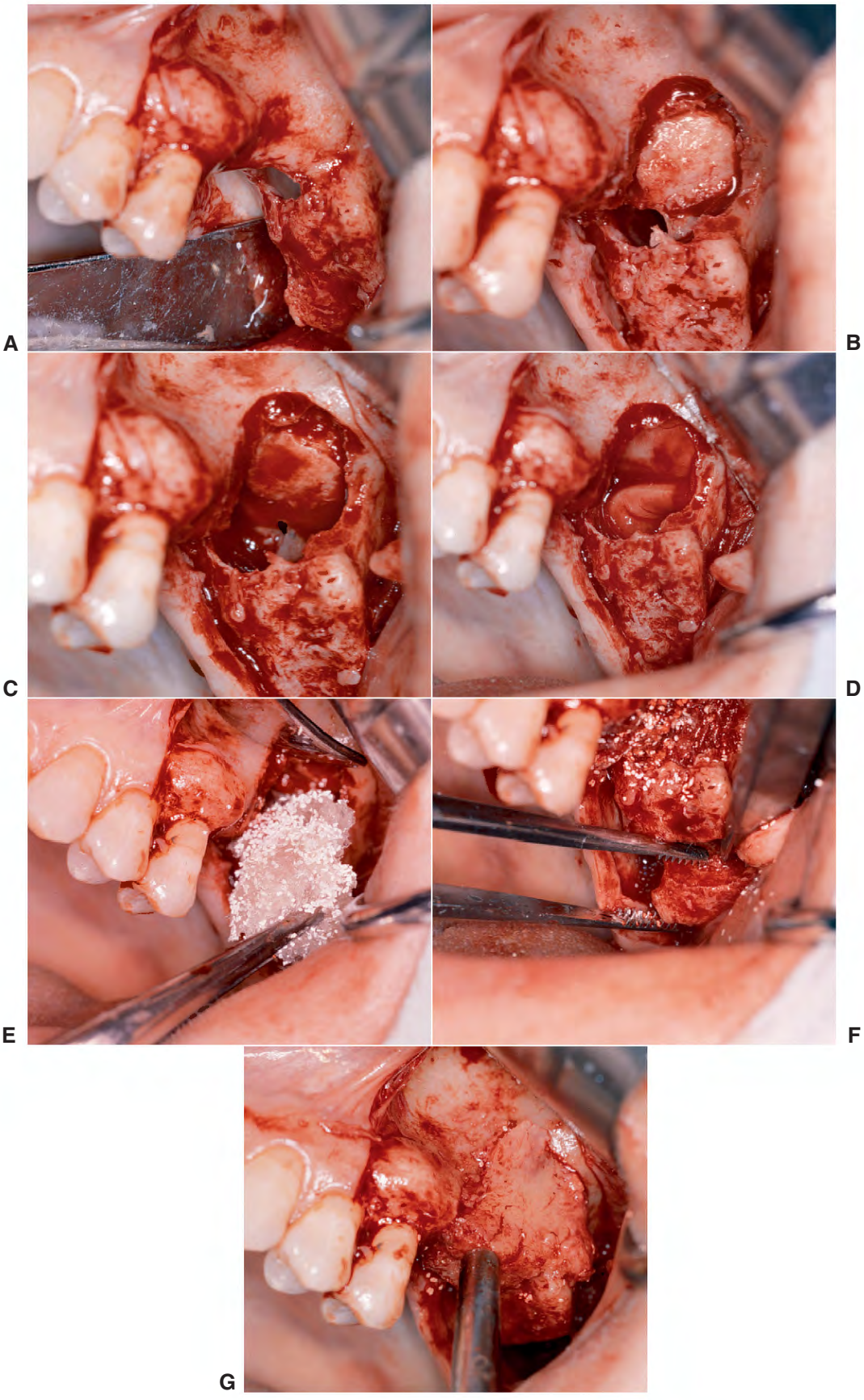


Figure 38-33 **A,** The bony opening in the lateral maxilla contributes to a maxillary sinus membrane tear on reflection of the soft tissue. **B,** The lateral-access window is designed and elevated distal to the mucosal opening. **C,** The sinus membrane elevation around the tear makes the opening smaller. **D,** A piece of collagen is placed over the membrane. **E,** The sinus graft procedure is completed. Care is taken to prevent overfilling the sinus. **F,** The tuberosity is harvested. **G,** The tuberosity is placed on the sinus floor and lateral wall.

WWW.HIGHDENT.IR

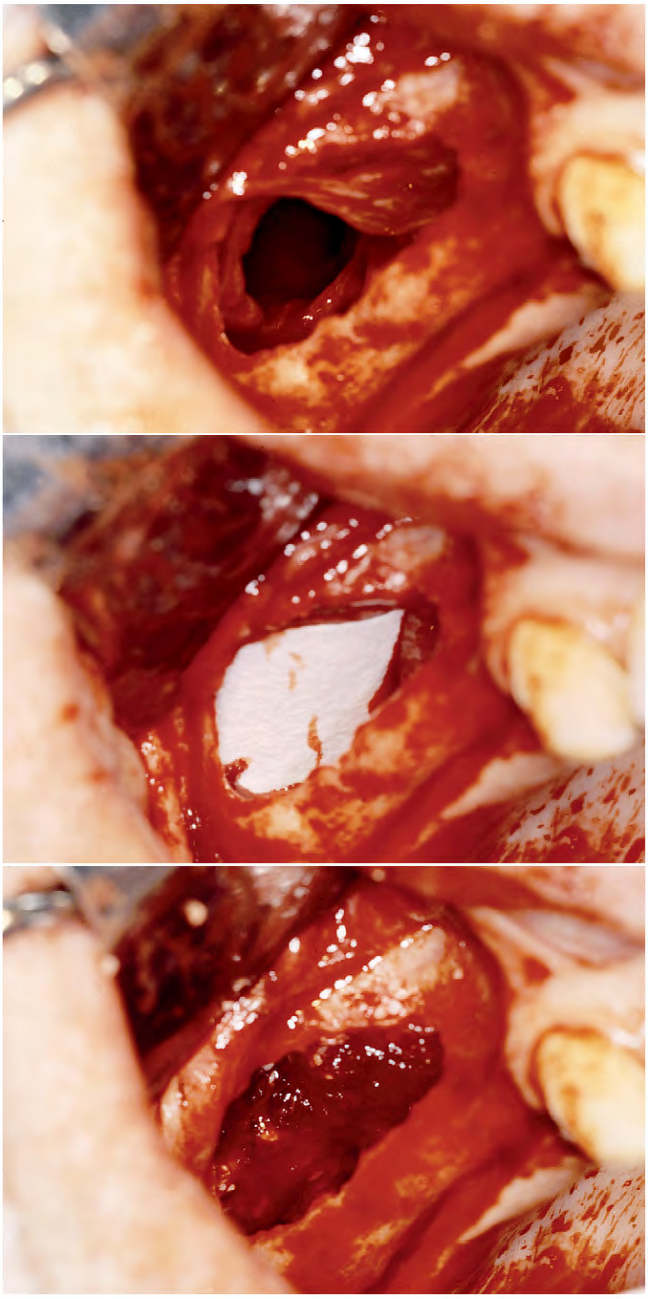


Figure 38-34 **A,** When the opening in the sinus mucosa is greater than 6 mm, the tissues distal are elevated, but often the remaining mucosal margins do not approximate each other. **B,** A dry piece of collagen (BioMend) is placed over the sinus membrane opening. **C,** A parental form of antibiotic moistens the collagen barrier once in place, or extra antibiotic is mixed into the graft material.

Ostium obstruction is also possible from swelling of the membrane related to the surgery. These conditions increase the risk of infection. However, despite these potential complications, the risk of the infection is low (less than 5%); therefore the sinus graft surgery should continue, and the patient should be monitored postoperatively for appropriate treatment.

When a larger sinus membrane perforation has occurred during a SA-3 approach, it is prudent to delay implant insertion. Placement of implants is deferred for at least 2 months (in a SA-3 option) to allow for healing of the membrane and the gingival tissues on the edentulous crest. Risks of the sinus graft entering the sinus proper increase when implants are pushed into the grafted site. Therefore the waiting period before implant insertion permits assessment of postsurgical complications and graft consolidation before the implants are inserted.

Antral Septa

Antral septa (of buttresses, webs, and struts) are the most common osseous anatomical variant seen in the maxillary sinus. Underwood, an anatomist, first described maxillary sinus septa in 1910. He postulated that the cause of these bony projections derived from three different periods of tooth development and eruption. Krennmair et al.[187] further classified these structures into two groups: primary, which are a result of the development of the maxilla, and secondary, which arise from the pneumatization of the sinus floor after tooth loss.

Misch[179] postulated that septa might be bone reinforcement pillars from parafunction when the teeth were present. He noticed these structures occur more often in SA-3 sinuses and after a shorter history of tooth loss. Long-term edentulous sites and SA-4 sinuses have fewer septa. The prevalence of septa has been reported to be in the range of 33% of the maxillary sinuses in the dentate patient and as high as 22% in the edentulous patient.[187,188] The septa may be complete or incomplete on the floor, depending on whether they divide the bottom of the sinus into compartments. The septa may also be incomplete from the lateral wall or, the medial wall, or it should extend from the floor.

The shape of an incomplete maxillary sinus septum often resembles an inverted gothic arch that arises from the inferior or lateral walls of the sinus. In rare instances, they may divide the sinus into two compartments that radiate from the medial wall toward the lateral wall (Figure 38-35).

The most common location of septa in the maxillary sinus has been reported to be in the middle (second bicuspid–first molar) region of the sinus cavity. CT scan studies have shown that 41% of septa are seen in the middle region, followed by the posterior region (35%) and the anterior region (24%). For diagnosis and evaluation of septa, CT scans are the most accurate method of radiographic evaluation.[188,189] Panoramic radiography has been shown to be very inaccurate with a high incidence of faulty diagnoses.

Sinus septa may create added difficulty at the time of surgery. Maxillary septa can prevent adequate access and visualization to the sinus floor; therefore inadequate or incomplete sinus grafting is possible. These dense projections complicate the surgery in

WWW.HIGHDENT.IR

همیار دندانسازان

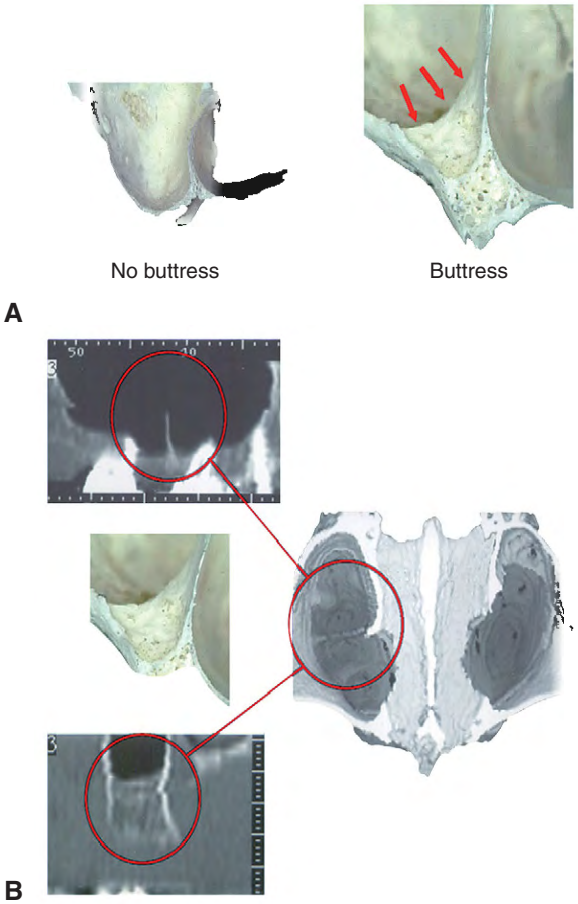


Figure 38-35 **A,** Septa or buttresses are frequently found on the floor and/or walls of the maxillary sinus. Looking down into the sinus on the left shows a smooth floor and walls. The maxillary sinus on the right has a septum. **B,** The patient's right maxillary sinus has a septum in the middle. The radiographic images are highlighted. This is the most common location for a septum.

several ways. After scoring the lateral-access window in the usual fashion, the lateral-access window may not greenstick fracture and rotate into its medial position. The strut reinforcement is also more likely to tear the membrane during the releasing of the access window. The sinus membrane is often torn at the apex of the buttress during sinus membrane manipulation, because difficulty exists in elevating the membrane over the sharp edge of the web, and the curette easily tears the membrane at this position. However, because septa are mainly composed of cortical bone, immediate implant placement may engage this dense bone, allowing for strong intermediate fixation. Moreover, septa allow for faster bone formation because they act as an additional wall of bone for blood vessels to grow into the graft.

Management of Septa Based on Location

The use of CT radiographs before sinus graft surgery permits the surgeon to observe and plan the necessary modifications to the sinus graft procedure as a result of the septa. The modification to the surgery is variable depending on its location. The septa may be in the anterior, middle, or distal compartment of the antrum. When the septum is found in the anterior section, the lateral-access window is divided into sections: one in front of the septa and another distal to the structure. This permits the release of each section of the lateral wall after tapping with a blunt instrument. The elevation of each released section permits investigation into the exact location of the septa and to continue the mucosal elevation.

The mucosal tissue may often be elevated from the lateral walls above the septa. The curette may then slide down the side walls and release the mucosa from the bottom half of the septum on each side. The sinus curette should then approach the crest of the buttress from both directions, up to its sharp apex. This permits elevation of the tissue over the web region without tearing the membrane (Figure 38-36).

When the strut is located in the middle region of the sinus, it is more difficult to make two separate access windows within the direct vision of the surgeon. As a result, one access window is made in front of the septa. The sinus curette then proceeds up the anterior aspect of the web, toward its apex. The curette then slides toward the lateral wall and above the septa apex. The curette may then slide over the crest of the septum approximately 1 to 2 mm. A firm, pulling action fractures the apex of the septum. Repeated similar curette actions can fracture the web off the floor. Once the septum is separated off the floor, the curette may proceed more distal along the floor and walls (Figure 38-37).

When the septum is in the posterior compartment of the sinus, it is often distal to the last implant site. When this occurs, the posterior septum is treated as the posterior wall of the sinus. The sinus membrane manipulation and sinus graft is placed up against and anterior to the posterior septum.

Bleeding

Bleeding from the lateral-approach sinus elevation surgery is rare; however, it has the potential to be troublesome. Three main arterial vessels should be of concern with the lateral-approach sinus augmentation. Because of the intra- and extraosseous anastomoses that are formed by the infraorbital and posterior superior alveolar arteries, intraoperative bleeding complications of the lateral wall may occur. The soft tissue vertical-release incisions of the facial flap in a resorbed maxilla may sever the extraosseous anastomoses. The extra-osseous anastomosis on average is located 23 mm from the crest of the dentate ridge; however, in the resorbed maxilla, it may be within 10 mm of the crest. When this artery is severed, significant bleeding has been observed. These vessels originate from the maxillary artery and have no bony landmark to compress the vessel. Therefore vertical release incisions in the soft tissue should be kept to a minimum height with delicate reflection

WWW.HIGHDENT.IR

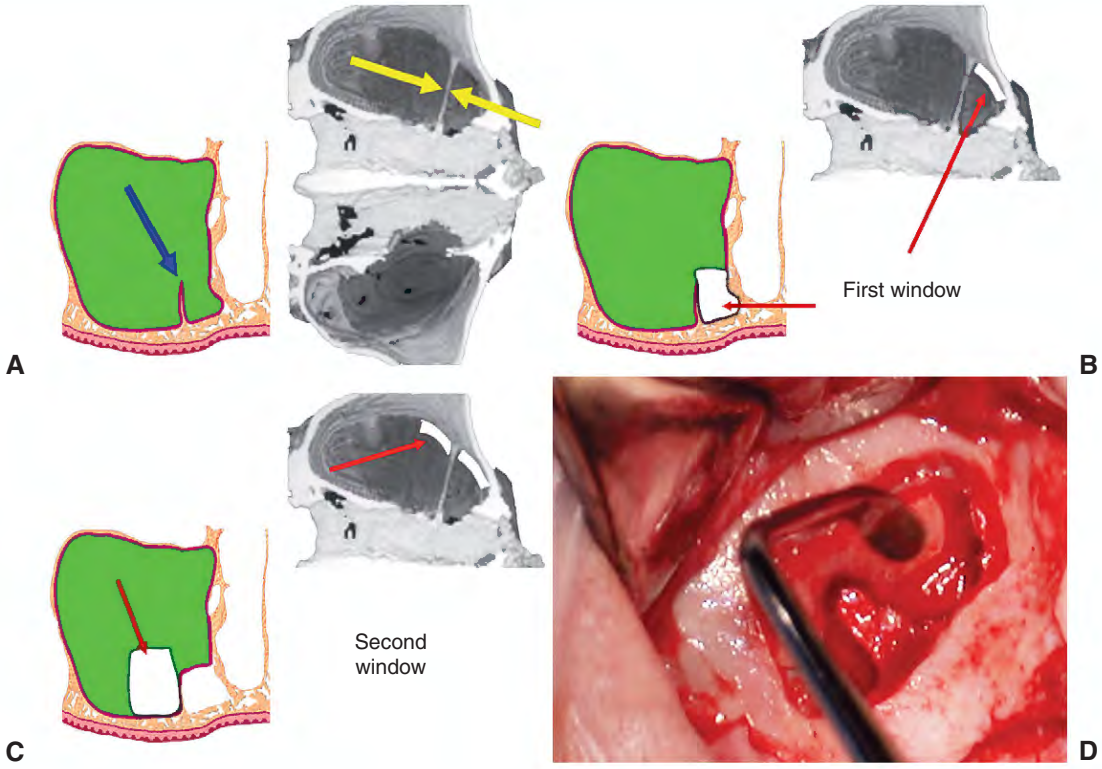


Figure 38-36 **A,** A maxillary septum in the anterior section of the maxillary sinus floor. **B,** An anterior lateral-access window is made, and the sinus membrane is elevated to the top of the septum. **C,** A second window is made distal to the anterior septum, and the sinus graft is placed in both anterior and posterior regions. **D,** An intraoral view of two access windows in front and behind a septum in the anterior section of the sinus.

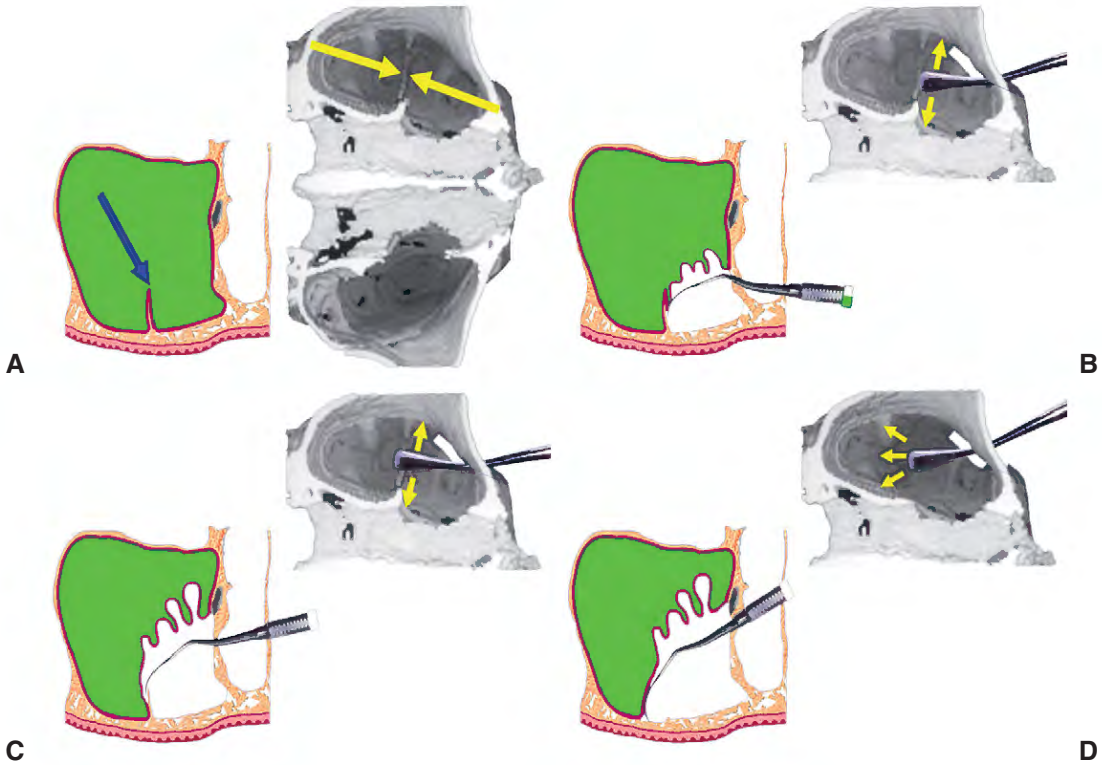


Figure 38-37 **A,** A maxillary septum found on the floor in the middle of the sinus. **B,** An access window and curette elevates the mucosa anterior to the septum. **C,** The curette slides over the top of the septum and pulls it off by greenstick fracture. **D,** Once the septum is removed, the rest of the sinus mucosa may be elevated.

WWW.HIGHDENT.IR

همیار دندانسازان

of the periosteum. Hemostats are usually difficult to place on the facial flap to arrest the bleeding. Significant pressure at the posterior border of the maxilla and elevation of the head to reduce the blood pressure to the vessels usually stops this bleeding. The elevation of the head may reduce nasal mucosal blood flow by 38%.[190,191] When this is not effective, pressure against the fourth cervical vertebra (C4) in the neck to reduce blood flow to the external carotid and collagen sponges in the posterior region can be used to reduce and arrest the hemorrhage. Microfibrillar purified sheep collagen (Avitene) may adhere to a wet surface and promote clot formation and stabilization.[192]

The vertical component of the lateral-access wall for the sinus graft often severs the intraosseous anastomoses of the posterior alveolar artery and infraorbital artery, which is on average approximately 15 to 20 mm from the crest of a dentate ridge. Methods to limit this bleeding, which is far less of a risk, have been addressed and include cauterization by the hand piece and diamond bur without water, electrocautery, or pressure on a surgical sponge while the head is elevated.

The third artery of which the implant surgeon should be cautious is the posterior lateral nasal artery. This artery is a branch of the sphenopalatine artery that is located within the medial wall of the antrum. As it courses anteriorly, it anastomoses with terminal branches of the facial artery and ethmoidal arteries. A significant bleeding complication may arise if this vessel is severed during elevation of the membrane off the thin medial wall.

If the excessive bleeding occurs while the medial wall is elevated, then the sinus may be packed with Avitene, followed by packing with large 4 × 4-inch surgical sponges, elevation of the head, and pressure on the C4 space on the same side as the sinus to stop the bleeding. Once the bleeding is arrested, the sponges are removed, the layered graft materials may be inserted, and the procedure is completed.

Epistaxis (active bleeding from the nose) is a common disorder; however, it has been reported that 6% of patients who experience this in the general population require medical treatment to control and stop the hemorrhage because it lasts longer than 1 hour. Of these patients, 15% are on anticoagulant therapy. Treatment options to treat epitasis include nasal packing, electrocautery, and the use of vasoconstrictive drugs. Vessel ligation and/or endoscopic surgery are necessary on rare occasion.

The most common site (90%) of nasal bleeding is from a plexus of vessels at the anteroinferior aspect of the nasal septum and the anterior nasal cavity (which is anterior to the sinus cavity and within the anterior projection of the nose). The posterior nasal cavity accounts for 5% to 10% of epitasis events and is in the region of the sinus graft. If the orbital wall of the sinus is perforated, or if an opening into the nares is already present from a previous event, then the sinus curette may enter the nares and cause bleeding. The arteries involved in this site are composed of branches of the sphenopalatine and descending palliative arteries, which are branches of the internal maxillary artery. The posterior half of the inferior turbinate has a venous network, the Woodruff plexus. Lavage of the nares with warm saline and oxymetazoline decongestant sprays provides excellent vasoconstritive activity to treat the condition. A cotton roll with silver nitrate or lidocaine with 1:50,000 epinephrine is also effective.

Bleeding from these arteries during the sinus graft procedure may also be controlled by nasal packing with bovine collagen, electrocautery, head elevation, or packing the sinus with surgical sponges (gauze). Pressure against the C4 (at the level of the thyroid cartilage) on the same side as the bleeding may also be of benefit.

Bleeding from the nose may also be observed after sinus graft surgery. Placing a cotton roll, coated with petroleum jelly with dental floss tied to one end, within the nares may obtund nose bleeding after the surgery. After 5 minutes the dental floss is gently pulled and removes the cotton roll. The head is also elevated, and ice is applied to the bridge of the nose. If bleeding cannot be controlled, then reentry into the graft site and endoscopic ligation by an ENT surgeon may be required.

SHORT-TERM POSTOPERATIVE COMPLICATIONS

Short-term complications are defined as those that occur within the first few months after surgery.

Incision Line Opening

Incision line opening is uncommon for this procedure because the crestal incision is in attached gingiva and at least 5 mm away from the lateral-access window. Routinely, the soft tissue requires release before primary approximation and suturing. Because a collagen membrane is placed over the window, the soft tissue will not approximate without tension unless the surgeon expands the facial flap by releasing the periosteum above the mucogingival junction (where the tissue becomes thicker). Incision line opening occurs more commonly when lateral-ridge augmentation is performed at the same time as sinus graft surgery, or when implants are placed above the residual crest and covered with the soft tissue. It may also occur when a soft tissue–supported prosthesis compresses the surgical area during function before suture removal.

The consequences of incision line opening are delayed healing, leaking of the graft into the oral cavity, and increased risk of infection. However, if the incision line failure is not related to a lateral onlay graft and is only on the crest of the ridge and away from the

WWW.HIGHDENT.IR

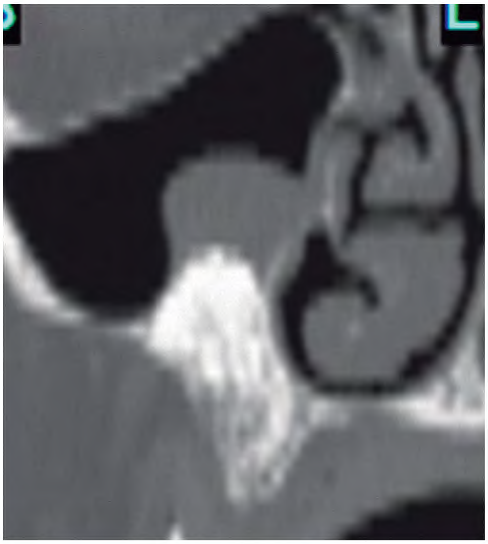


Figure 38-38 Ostium blockage caused by a sinus graft procedure resulting in the blockage of the maxillary osteomeatal complex.

sinus access window, then the posterior crestal area is allowed to heal by secondary intention. During this time, a soft tissue–borne restoration should be aggressively relieved, with no reline material in contact with the ridge. If incision line opening includes a portion of a nonresorbable membrane (i.e., for lateral-ridge augmentation), then the membrane should be cleaned at least twice daily with an abrasive devise and oral rinses of chlorhexidine. If the incision line is not closed after 2 months, then a surgical procedure should reenter the site, expand the tissues, remove the bone regeneration membrane, and reapproximate the tissue.

Nerve Impairment

Special attention must be given to the course of the infraorbital nerve. As described previously, the infraorbital nerve lies within the orbital floor and exits the foramen approximately 6.1 to 7.2 mm from the orbital rim. However, anatomical variants have been reported to be as far as 14 mm from the orbital rim in some individuals. In the severely atrophic maxilla, the infraorbital neurovascular structures exiting the foramen may be close to the intraoral residual ridge and should be avoided when performing sinus graft procedures to minimize possible nerve impairment. This is of particular concern on soft tissue reflection and the bone preparation of the superior aspect of the window. Because the infraorbital nerve is responsible for sensory innervations to the skin of the molar region between the inferior border of the orbit and the upper lip, iatrogenic injury to this vital structure can result in significant neurosensory deficits of this anatomical area. Most often, the nerve is not severed, and a neurotmesis is present. This paresthesia usually resolves within 1 month after the surgery.

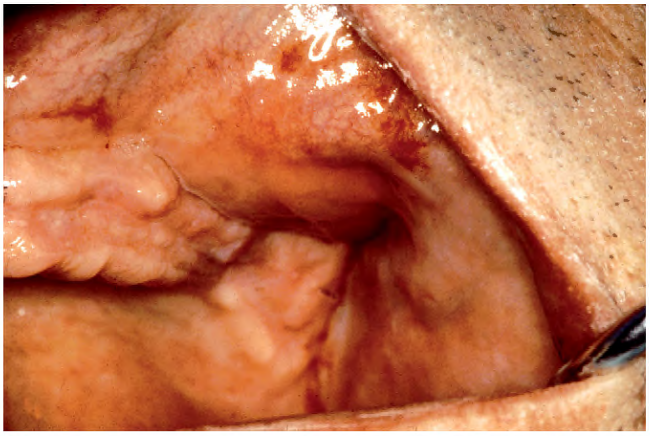


Figure 38-39 The most common clinical sign of a maxillary sinus infection after a sinus graft is a swelling from the lateral-access window.

Acute Maxillary Rhinosinusitis

The manipulation and altering of the sinus membrane shows no significant long-term changes. This has been seen clinically and reported through in-depth studies. However, mild inflammatory mucosal changes do occur immediately postoperatively. This is a temporary reaction to the normal physiologic activity of the mucosal airway defense mechanism. According to the literature, acute postoperative sinusitis occurs as a complication in approximately 3% to 20% of sinus graft procedures,* and it represents the most common short-term complication. Most often, the infection begins more than 1 week after surgery, although it may begin as soon as 3 days later. Because the surgical field is close to several vital structures, postoperative infections may arise and spread very rapidly with possible life-threatening complications.

Maxillary sinusitis is the most widely reported postoperative complication after sinus augmentation procedures. This disorder should be considered if the patient postoperatively complains of any of the following symptoms: headache, pain or tenderness in the area of the maxillary sinus, and rhinorrhea. Studies have supported the fact that patients who had predisposing factors for sinusitis were more at risk of developing postoperative transient sinusitis. The wide range of reported percentages (3% to 20%) may be the result of different methods used for diagnosis (i.e., clinical, radiographic, endoscopic) (Figures 38-38 and 38-39).

Sinus Physiology

Elevation of the sinus mucosa and bone grafting does alter the overall maxillary sinus environment by reducing the size of the sinus and repositioning the mucociliary transport system. In spite of this, only short-term clearance impairment exists, resulting in only subclinical effects on the sinus physiology. However, in cases of preoperative sinusitis histories, sinus

*References 8, 13, 69, 76, 105, 106, 127.

WWW.HIGHDENT.IR

همیار دندانسازان

elevation surgery may predispose a patient to sinus-related complications. It has been shown that these procedures do alter the microbial environment. Studies reveal that at 3 months after surgery, positive sinus cultures were present compared with cultures taken for the same patients preoperatively. However, after 9 months the cultures were similar to the preelevation results. The key is maintenance of the osteomeatal opening between the maxillary sinus and the nasal cavity.

Very few cases of long-term pathologic symptoms have been reported in the literature after sinus graft surgery. However, numerous case studies in the medical literature have reported on postoperative sinusitis after sinus surgery. Resolution of these conditions has been accomplished with the use of antibiotic drugs and/or Caldwell-Luc procedures. No long-term chronic sinusitis cases have been reported or documented.

Prophylactic antibiotic medications and sound surgical principles minimize postoperative infections and complications.[48] However, autogenous bone, allografts, and alloplasts in the sinus graft may be subject to infection. The resistance to contamination is low and may be increased if contaminated by intraoral or sinus pathogens.

Cases of maxillary sinusitis after dental implant surgery have rarely been reported in the dental literature. However, recently in the medical literature, numerous cases of minor to severe complications after sinus surgery have been documented. Although very infrequent, severe infections may lead to more severe complications, such as orbital cellulitis, optic neuritis, cavernous sinus thrombosis, epidural and subdural infection, meningitis, cerebritis, blindness, osteomyelitis, and, although rare, brain abscess and death.[79] Therefore it is recommended to develop a professional relationship with an otolaryngologist before the dental surgeon's first sinus graft is performed. After infections are eliminated, oroantral fistulas are possible that may be difficult to correct, possibly requiring numerous surgical procedures that include soft tissue and bone grafts. In addition, prosthetic obturators may be required until the oral antral fistula is corrected.

Although the incidence of infection after the procedure is usually low, the damaging consequences on osteogenesis and the possibility of serious complications require that any infection be aggressively treated. In case of postoperative infection, it is recommended that the clinician perform a thorough examination of the area by palpation, percussion, and visual inspection to identify the area primarily affected. Infection follows the path of least resistance and is observed by changes in specific anatomical sites to which it spreads[66] (see Table 38-1).

Clinical Evaluation

Radiographic evaluation of acute rhinosinusitis is both expensive and often inaccurate (Figure 38-40). As such, a patient history for acute sinusitis is a benefit and is diagnostic when two or more of the following factors are present: (1) facial congestion or fullness, (2) nasal obstruction or blockage, (3) nasal discharge, (4) purulence or discolored postnasal discharge, (5) facial pain or pressure, (6) hyposomia or anosmia (decrease or no ability to detect an odor), (7) purulence in the nares on physical examination, (8) fever, (9) headache, (10) halitosis, (11) dental pain, (12) cough, and (13) ear pain. When multiple symptoms are evident, antibiotic drugs for acute rhinosinusitis are in order. In addition, when associated with unresolved symptoms after a sinus graft, a CT scan may be necessary.

Therapeutic Treatment of Postoperative Infections

If infection occurs postoperatively, then treatment must be aggressive because of the possible complications and morbidity of the graft material. Antibiotic therapy is the first line of treatment for these related symptoms. Diagnosis is often presumptive without microbiological confirmation and usually based on empirical evidence. Identification of the causative pathogen is usually difficult, requiring either an antral puncture or endoscopy to obtain an uncontaminated specimen for culture.

When selecting antibiotic medications for sinus infections, a variety of factors must be evaluated. These include the most common type of pathogens involved, antimicrobial resistance, pharmacokinetic and pharmacodynamic properties, and the sinus tissue penetration of the various antibiotic drugs. The antibiotic medication of choice should be effective against respiratory pathogens and should have known activity against resistant strains of the common pathogens. Two such factors are used when evaluating sinus antibiotic medications: (1) the minimum inhibitory concentration (MIC) and (2) the concentration of antibiotic drugs penetrating inflamed diseased sinus tissue. The MIC is the lowest concentration of the antimicrobial agent that results in the inhibition of growth of a microorganism. The MIC is usually expressed by MIC 50 or MIC 90,

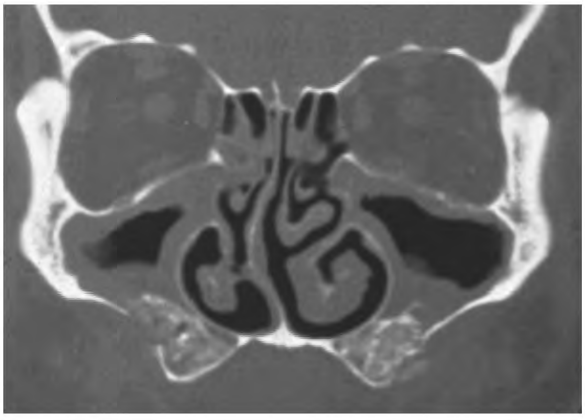


Figure 38-40 A computed tomography (CT) scan taken after sinus graft of a patient with no history of infection or complications. The maxillary sinus mucosa appears thickened, yet no infection was observed.

WWW.HIGHDENT.IR

meaning that 50% or 90% of the microbial isolates are inhibited, respectively.

Previous studies and treatment modalities used amoxicillin as the first drug of choice. However, with the increasing prevalence of penicillinase- and β-lactamase–producing strains of *Haemophilus influenzae* and *Moraxella catarrhalis*, along with penicillin-resistant strains of *Streptococcus pneumoniae*, other alternative antibiotic drugs should be selected.

Antimicrobial Classes

β-Lactam Medications. The most common β-lactam antibiotic drugs used in the treatment of sinusitis are penicillin (amoxicillin, Augmentin) and cephalosporin (Ceftin, Vantin). Amoxicillin has been the drug of choice for years to combat the bacterial strains associated with sinusitis. However, its effectiveness has been questioned recently because of the high percentage of β-lactamase–producing bacteria and penicillin-resistant *S. pneumoniae*. Augmentin (amoxicillin-clavulanate) has the added advantage of activity against β-lactamase bacteria. It has been associated with a high incidence of gastrointestinal side effects; however, with a new dosing regimen (twice a day [bid]), these complications have been significantly decreased.

Two recommended cephalosporin medications have also been suggested to treat sinusitis and include cefuroxime axetil (Ceftin) and cefprodoxime proxetil (Vantin). Other cephalosporin drugs fail to achieve adequate sinus fluid levels against the causative pathogens. Ceftin and Vantin have good potency and efficacy and exhibit strong activity against resistant *S. pneumoniae* and *H. influenzae*.

Fluoroquinolone Medications. Fluoroquinolone drugs are bactericidal antibiotic medications that are classified into four different generations. The third-generation quinolone drugs are well-suited, broad-spectrum antibiotic medications and have been labeled by the U.S. Food and Drug Administration (FDA) for use against sinus pathogens. They exhibit excellent absorption and achieve very significant sinus blood levels, even in pathologic conditions. Quinolone drugs are distributed extensively throughout the sinus, with high levels being found in inflamed tissue and maxillary sinus cysts. The tissue/blood ratio is approximately 4:1, making it extremely potent within the diseased sinus. The three most common quinolone medications used for sinus treatment include levofloxacin (Levaquin), gatifloxacin (Tequin), and moxifloxacin (Avelox).

Macrolide Medications. Macrolide drugs are bacteriostatic agents that include erythromycin, clarithromycin (Biaxin), and azithromycin (Zithromax). Macrolide medications have good activity against susceptible pneumococci; however, with the increasing rate of macrolide resistance, their use in combating sinus pathogens is becoming associated with a high likelihood of clinical failure. These antibiotic drugs are very active against *M. catarrhalis*, although their activity on *H. influenzae* is questionable. Therefore these antibiotic medications are not suggested to treat postoperative sinus infections.

Lincosamide Medications. Clindamycin (Cleocin) is the primary lincosamide drug used in clinical practice today that is considered to be bacteriostatic. However, in high concentrations, bactericidal activity may be present. Clindamycin is mainly used for the treatment of gram-positive aerobes and anaerobes. With acute sinusitis disease, clindamycin is usually not indicated because it exhibits no activity against *H. influenzae* and *M. catarrhalis*. This drug may be used in chronic sinus conditions because anaerobic organisms play a much larger role in the disease process.

Tetracycline Medications. Doxycycline (Vibramycin) is a bacteriostatic agent with adequate activity against penicillin-susceptible pneumococci and *M. catarrhalis*. This drug does not exhibit any activity against penicillin-resistant bacteria and is not effective against *H. influenzae*. Severe side effects of this medication are photosensitivity and esophageal caustic burns. As a consequence, tetracycline drugs are not used to treat postoperative sinus infections.

Sulfonamide Medications. The most common sulfonamide drug, trimethoprim-sulfamethoxazole (Bactrim), is bacteriostatic. Recently, a high rate of resistance to these drugs has been seen with *S. pneumoniae*, *H. influenzae*, *M. catarrhalis,* and other sinus pathogens. Therefore this drug is not considered to treat postoperative infections, unless a culture and sensitivity test has been performed.

Nitroimidazole Medications. Metronidazole is the most important member of the nitroimidazole group. It is bactericidal and is effective against gram-positive and gram-negative anaerobic bacteria. Its main use would be in the treatment of chronic sinus conditions; however, it should be used with another antibiotic drug to be effective against aerobic bacteria.

In the evaluation of different antibiotic drugs used for the treatment of pathologic conditions of the sinus, meticulous analysis of the activity against the most common pathogens must be evaluated. With all the antibiotic medications evaluated, amoxicillin-clavulanate, cefuroxime axetil, levofloxacin, and moxifloxacin showed significant sinonasal and MIC 90 blood levels against the most common pathogens associated with sinus infections. Moxifloxacin, a third-generation fluoroquinolone drug, has been shown to have superior qualities compared with many other antibiotic medications. It shows extensive distribution throughout the sinuses in both inflamed and noninflamed sinus tissue, with significantly high concentration within maxillary sinus cysts. The tissue/blood ratio is 4:1, with blood levels occurring 3 to 4 hours after administration. Because of the potency and expense of this medication, it is used only in the treatment for severe infections.

Decongestant Medications. If acute sinusitis symptoms are present, then a nasal decongestant may be used to maintain the patency of the ostium. These

WWW.HIGHDENT.IR

همیار دندانسازان

sympathomimetic drugs influence α-adrenergic receptors to reopen a blocked ostium and facilitate drainage. Oxymetazoline 0.05% (Afrin or Vicks Nasal Spray) is the most effective over-the-counter topical decongestant. The vasoconstrictor action of this medication will last for approximately 5 to 8 hours. However, proper administration positioning must be adhered to, which includes the patient lying down with the head down. The antimicrobial solution is then directly applied to drain into the sinus ostium area. The use of this medication is limited to 3 to 4 days to prevent a rebound phenomenon and the development of rhinitis medicamentosa.

No Response to Antibiotic and Decongestant Medications

If symptoms are not alleviated with antibiotic and decongestant medications, then possible referral to the patient's physician or otolaryngologist is warranted. Emergency consultation should be considered if the patient complains of severe headache that is not relieved by mild analgesics, as well as persistent or high fever, lethargy, visual impairment, or orbital swelling.

Saline Rinses

An important procedure for the patient with the presence of a sinus graft infection is saline rinses with a bulb syringe or a squeeze bottle in the nostril used to lavage the sinus through the ostium. The nasal saline rinse has a long history for treatment of sinonasal disease. Hypertonic and isotonic saline rinses have proven efficiency against chronic sinusitis. These techniques of nasal irrigation have been evaluated, with the best option of a positive-pressure irrigation using a squeeze bottle that delivers a gentle stream of saline to the nasal cavity (NeilMed, Santa Rosa, Calif.). The syringe or squeeze bottle should not seal the nasal opening, because this may force bacteria up toward the ethmoidal sinus. Instead, a gentle lavage with sterile saline rinses the sinus and flushes out the mucus and exudate. Ideally the head is placed down and forward so that the saline can reach the ostium in the superior and anterior portion of the sinus. The course of therapy should continue for at least 7 days.[193]

The most current, comprehensive study on the treatment of sinus disease involves guidelines established by the Sinus and Allergy Health Partnership, Centers for Disease Control and Prevention, and the FDA in 2000. With this information as a guide, the following recommendations for antibiotic use in the treatment of infections after sinus graft are suggested.

Mild Infection

Symptoms

- Purulent and nonpurulent nasal drip
- Nasal blockage
- Facial pain and pressure
- Intraoral and extraoral swelling
- Cough

Treatment

1. Amoxicillin-clavulanate (Augmentin) 825 mg/125 mg (1 tablet bid for 10 days)

   a. If nonanaphylactic allergy to amoxicillin, cefuroxime axetil (500 mg) 1 tablet bid for 10 days
   b. If anaphylactic allergy to amoxicillin, levofloxacin (500 mg) 1 tablet bid for 7 days

2. Over-the-counter decongestant (oxymetazoline 0.05% for 3 days)
3. Nasal saline rinses

NOTE: If the patient does not respond to the initial treatment for mild infections within 5 days, then the patient should immediately be placed on moxifloxacin and a Medrol dosepack. A patient still symptomatic after 3 days of moxifloxacin treatment should have a medical referral for evaluation and treatment. Removal of the graft is often indicated in those cases.

Moderate to Severe Infection

Symptoms from mild infection

- Severe headache
- High persistent fever (>102.5° F)
- Periorbital swelling
- Ocular symptoms (diplopia, proptosis)
- Altered mental status
- Infraorbital hyperesthesia

Treatment

1. Moxifloxacin (400 mg) 1 tablet bid for 10 days
2. Medrol dosepak (4 mg), as directed
3. Nasal saline rinses

NOTE: If reduction of symptoms for moderate and severe infections is not seen within 5 days after moxifloxacin treatment, then the patient should be referred for medical consultation and treatment.

Spread of Infection

Because of the anatomical and topographic location of the maxillary sinus, infections from oral or sinus pathogens may spread quickly to adjacent sites.

Sinus-related pathologic conditions are the most common cause of orbital infection, accounting for 60% to 84% of cases. Because of the seriousness of ocular infections, early diagnosis and aggressive treatment is paramount.

Various routes may predispose this area to infection from the maxillary sinus and include the following:

1. The venous plexus of the maxillary sinus drains through the posterior wall into the deep facial vein, through the pterygoid plexus, and finally into the cavernous sinus.
2. Veins also perforate the osseous roof of the maxillary sinus, entering the orbit through the superior and

WWW.HIGHDENT.IR

inferior ophthalmic vein. These veins also are connected to the pterygoid plexus and cavernous sinus.
3. Additionally, numerous veins perforate the anterior wall that drain into the superior ophthalmic vein and into the cavernous sinus. From the cavernous sinus, drainage through the deep middle cerebral vein communicates with the white substance of the brain's superficial venous system.

Because of the elaborate maxillocerebral venous anastomoses, spread of infection from the maxillary sinus may result in possible sequelae such as brain abscesses, intraorbital abscesses, orbital cellulitis, cavernous sinus thrombosis, and osteomyelitis.

Implant Penetration into the Sinus

Brånemark et al.[194] reported on animal histologic studies and 44 clinical cases of implants penetrating the maxillary sinus. They reported success rates comparable to other maxillary implants, and no postoperative signs or symptoms were found with these implants. An animal study by Boyne[22] led to the same conclusion. The assumption was that direct connection between hard and soft tissues to the integrated implant created a barrier to the migration of microorganisms. However, it should be noted these animals do not have the same incidence of maxillary sinusitis comparable to humans.

It is possible that an implant that penetrates the sinus floor may contribute to a source of periodic sinusitis, because a bacterial smear layer would be difficult to remove through regular phagocytic activity. When this is suspected, an apicoectomy of the implant apex, from a lateral-access window, may be of benefit.

Oroantral Fistula

Oroantral fistulae may develop postoperatively, especially if the patient has a history of infection. Small oroantral fistulae (<5 mm) usually will close spontaneously after treatment with systemic antibiotic drugs and daily rinses with chlorhexidine. However, larger fistulae (>5 mm) will normally require additional surgical intervention. Larger fistulae are associated with an epithelialized tract, which is the result of the fusion of the sinus membrane mucosa to the oral epithelium. When this occurs, patients will usually complain of fluids entering the nasal cavity upon eating or drinking.

Closure of oroantral fistulae can be achieved by using broad-based lingual or facial rotated flaps. Buccal flaps to close the fistula may be more difficult after sinus graft because of the location of the graft site. In addition, the buccal tissue is very thin, and rotated or expanded buccal flaps usually result in loss of vestibular depth.

Before the initiation of the flap design, the soft tissue around the fistula is excised and the sinus floor curetted to ensure direct bone contact. A tension-free rotated flap is then made for complete covering of the communication. For oroartral closure after sinus graft procedures, a lingual flap is recommended because of the abundance of keratinized mucosa with an adequate blood supply. Flap designs include island flaps, "tongue-shaped" flaps, or rotational and advanced flaps, depending on the size of the exposure.

A key to close the oroartral opening is the dissection of the buccal flap lateral to the fistula. An incision that extends 15 mm anterior and posterior to the fistula is of benefit. The fistula then has an elliptical incision on each side of the opening. The core of tissue and the fistulous tract is excised. The facial flap is underminded and expanded well into the tissues of the cheek. The palatal aspect of the incision is adjacent to the tongue-shaped flap. Placement of the incision for the pedicle flap should be split thickness and take into account the location and depth of the greater palatine artery. Once the attached palatal pedicle graft is rotated to the lateral and attached to the facial flap, horizontal mattress sutures are placed to invert the flap to achieve a watertight seal. Sutures with high tensile strength should be used and allowed to remain in place for at least 2 weeks (Figure 38-41).

Overfilling of the Sinus

The goal of the sinus graft is to obtain enough vertical height of bone to place endosteal implants with long-term success. The maximum length requirement of an implant with adequate surface of design is rarely more than 15 mm, and as a result, the goal of the initial sinus graft is to obtain at least 16 mm of vertical bone from the crest of the ridge. This usually means the bottom one half of the sinus is filled with graft material, because most sinuses approximate 35 mm in height. A CT scan of the sinus before surgery may be used to estimate the amount of graft material required for the ideal volume of sinus graft material. Care should be given to the amount of graft material placed into the sinus. Overfilling the sinus can result in blockage of the ostium, especially if membrane inflammation or the presence of a thickened sinus mucosa exists.

The majority of sinus graft overfills do not have postoperative complications. If, however, a postoperative sinus infection occurs without initial resolution, reentry and removal of a portion of the graft and changing the antibiotic protocol may be appropriate.

Postoperative Maxillary Surgical Cysts

Postoperative maxillary surgical cysts have been reported in the literature after sinus graft surgery; however, they are extremely rare. In 1992, Misch et al.[92] reported one incidence of a maxillary surgical cyst associated with a past sinus graft and blade implant. Complete enucleation was accomplished, and healing was uneventful.

In 1927, Kubo reported a postoperative maxillary cyst arising in the maxilla as a delayed complication

WWW.HIGHDENT.IR

همیار دندانسازان

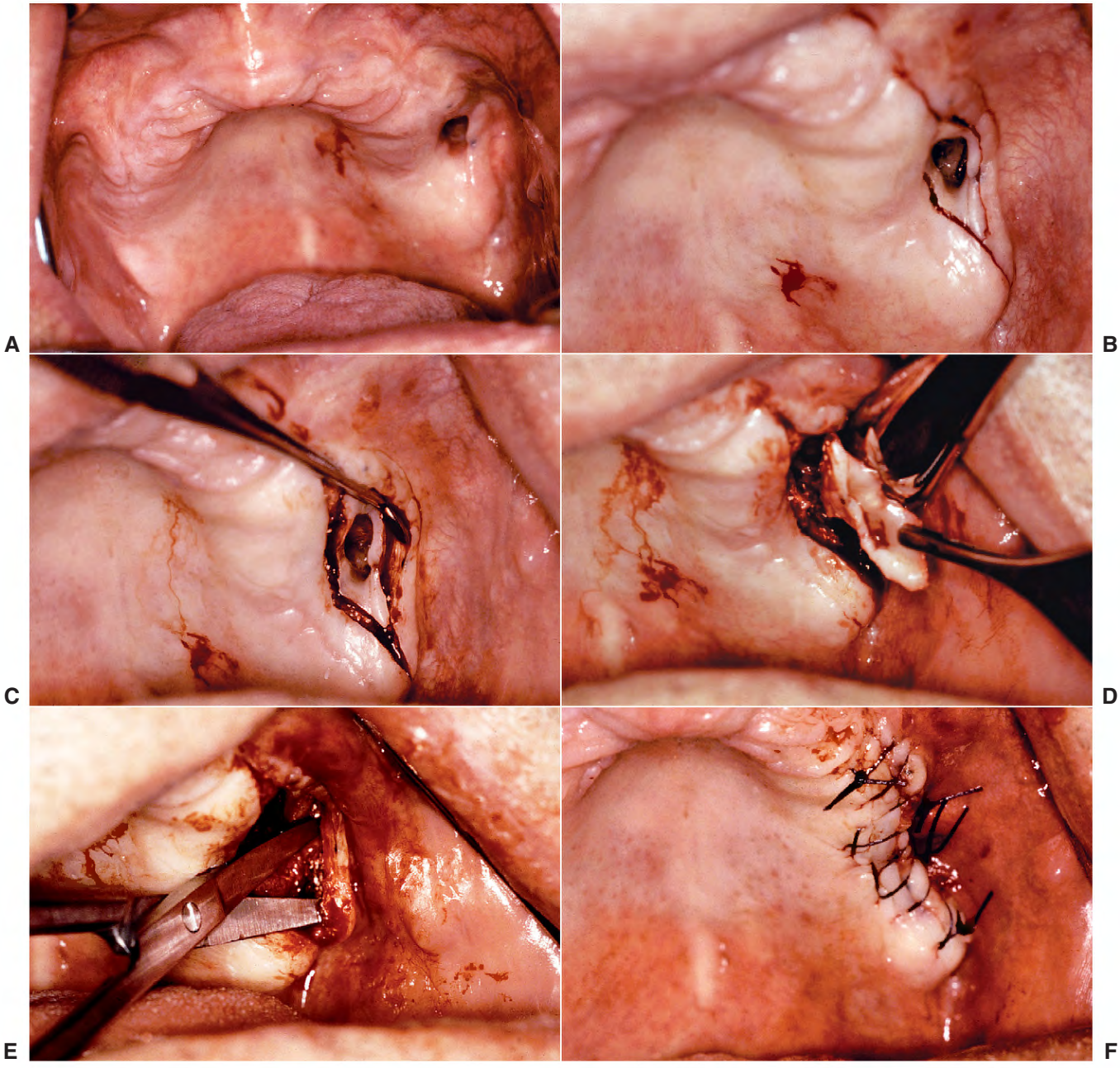


Figure 38-41 **A,** An oroantral fistula after sinus graft infection is evident on the patient's left side. **B,** An elliptical incision is made around the fistula and extends both anterior and posterior to the site. **C,** The tissues are reflected. **D,** The fistulous tract is removed, complete with the epithelial lining. **E,** The facial flap is undermined, and a submucosal space is created. **F,** Horizontal mattress sutures are used for the watertight closure.

of radical surgical intervention in the maxillary sinus.[91] Incidence reports are more common in Japan, where sinus disease is often treated aggressively with surgery,[93] but are rare in other parts of the world.

LONG-TERM RESULTS

The primary method of long-term evaluation of sinus grafts has been implant survival in these regions. Hypothetically, if the graft is composed of good-quality bone, then the endosteal implant should be maintained in health. Of course, proper implant and prosthetic procedures are required for these implants. In general, most reports in the literature contain high survival rates for all sinus graft materials and implant designs studied. The data presented at the Sinus Graft Consensus Conference reflected the high predictability of the procedure.[92] Thirty-eight clinicians provided data on 1007 sinus grafts that had received 3354 implants in function for at least 3 years. Long-term survival rates were in the 90% to 97% range. Material combinations consisted of only autogenous bone or combinations of alloplasts, allografts, and autogenous bone, yet all

WWW.HIGHDENT.IR

seemed to yield similar results. The most important factor for implant survival was the implant design, with HA-coated implants having a higher 3-year survival rate than the machined, noncoated screw design implants.

The treatment of the posterior maxillary can now be considered to be one of the most predictable regions in the mouth. Sinus grafting has been a major reason for this accomplishment. Del Fabbro et al.[29] performed a systematic review of the literature for sinus grafts and associated implants in 2004. A total of 252 articles related to this subject were reviewed, and 39 of these met inclusion criteria for a qualitative data analysis. There were 2046 patients and 6913 implants in this report, ranging from 1 to 6 years.

Implant survival was 87.7% for 100% autologous bone, 94.88% when autograft was combined with a variety of bone substitutes, and 95.98% when the bone graft was only a bone substitute mixture. The majority of articles that used autograft alone used a machined or smooth-surface implant. When surface condition was considered rather than graft materials, the smooth-surface implants had an 85.64% survival rate versus 95.98% for a rough-surface condition.

Between 1980 and 1990, the author reported on 385 human SA augmentations performed using the Tatum lateral-wall approach (SA-3 or SA-4). Of the 385 SA augmentations, only two maxillary antrum regions did not grow sufficient bone for implant placement, and the implant procedure was aborted after the 8-month healing interval. Additional data to 2005 brings these numbers to almost 2000 grafts, for which only nine antrum grew insufficient bone to place endosteal implants of adequate size. Indeed, the sinus graft surgery is the most predictable bone graft procedure available to grow adequate bone height in the mouth.

SUMMARY

In the past, implant treatment in the posterior maxilla was reported as the least predictable region for implant survival. Causes cited include inadequate bone height, poor bone density, and high occlusal forces. Past implant modalities attempted to avoid this region, with procedures such as excessive cantilevers from anterior implants or excess numbers of pontics when implants are placed anterior and posterior to the antrum.

The maxillary sinus may be elevated and SA bone regenerated to improve available bone height. Tatum began to develop these techniques as early as the mid-1970s.[3] Misch developed four options for treatment of the posterior maxilla in 1984, based on the height of bone between the floor of the antrum and the crest of the residual bone. These options were further modified to reflect the width of available bone, once adequate height was obtained. Root form implants of adequate size are indicated in the posterior maxilla. When the ridge anatomy is too narrow for root form implants, these ridges may be treated by bone spreading or with autogenous grafts. The higher forces and less dense bone often require larger-diameter implants.

It is the observation of the authors, using the sinus graft procedures described in this chapter for more than 20 years, in clinical practice, universities, and private implant institutes, that the sinus graft procedure is more than 97% effective. This region of the mouth predictably grows more bone in height than any other intraoral region.

References

1. Marcus SE, Drury JF, Brown LS et al: Tooth retention and tooth loss in the permanent dentition of adults: United States, 1988-1991, *J Dent Res* 75(special issue):684-695, 1996.
2. Misch CE: Treatment planning for edentulous maxillary posterior region. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.
3. Tatum OH: Lecture presented at Alabama Implant Study Group, Birmingham, Ala, 1977.
4. Tatum OH: Maxillary and sinus implant reconstruction, *Dent Clin North Am* 30:107-119, 1986.
5. Boyne PJ, James RA: Grafting of the maxillary sinus floor with autogenous marrow and bone, *J Oral Surg* 38:613-616, 1980.
6. Smiler DG, Holmes RE: Sinus lift procedure using porous hydroxylapatite: a preliminary clinical report, *J Oral Implantol* 13:2-14, 1987.
7. Chanavaz M: Sinus grafting related to implantology statistical analysis of 15 years of surgical experience, 1979-1994, *J Oral Implantol* 22:119-130, 1996.
8. Tidwell JK, Blijdorp PA, Stoelinga PJW et al: Composite grafting of the maxillary sinus for placement of endosteal implants, *Int J Oral Maxillofac Surg* 21:204-209, 1992.
9. Smiler DG, Johnson PW, Lozada JL et al: Sinus lift grafts and endosseous implants: treatment of the atrophic posterior maxilla, *Dent Clin North Am* 36:151-186, 1992.
10. Jensen J, Sindet-Petersen S, Oliver AJ: Varying treatment strategies for reconstruction of maxillary atrophy with implants: results in 98 patients, *J Oral Maxillofac Surg* 52:210-216, 1994.
11. Chiapasco M, Ronchi P: Sinus lift and endosseous implants: preliminary surgical and prosthetic results, *Eur J Prosthodont Rest Dent* 3:15-21, 1994.
12. Blomqvist JE, Alberius P, Isaksson S: Retrospective analysis of one-stage maxillary sinus augmentation with endosseous implants, *Int J Oral Maxillofac Implants* 11:512-521, 1996.
13. Jensen OT, Leonard BS, Block MS et al: Report of the Sinus Consensus Conference of 1996, *Int J Oral Maxillofac Implants* 13(suppl):11-30, 1998.
14. Valentini P, Abensur DJ: Maxillary sinus grafting with anorganic bovine bone: a clinical report of long-term results, *Int J Oral Maxillofac Implants* 18:556-560, 2003.
15. Lozada JL, Emanuelli S, James RA et al: Root form implants in subantral grafted sites, *J Calif Dent Assoc* 21:31-35, 1993.
16. Albrektsson T, Zarb G, Worthington P et al: The long-term efficacy of currently used dental implants: a review and proposed criteria of success, *Int J Oral Maxillofac Implants* 1:11-25, 1986.

WWW.HIGHDENT.IR

همیار دندانسازان

17. Adell R, Eriksson B, Lekholm U et al: A long-term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws, *Int J Oral Maxillofac Implants* 5: 347-359, 1990.
18. Lekholm U, van Steenberghe D, Herrmann I et al: Osseointegrated implants in the treatment of partially edentulous jaws: a prospective 5-year multicenter study, *Int J Oral Maxillofac Implants* 9:627-635, 1994.
19. Wood RM, Moore DL: Grafting of the maxillary sinus with intraorally harvested autogenous bone prior to implant placement, *Int J Oral Maxillofac Implants* 3:209-214, 1988.
20. Tawil G, Mawla M: Sinus floor elevation using a bovine bone material (Bio-Oss) with or without the concomitant use of a bilayered collagen barrier (Bio-Gide): a clinical report of immediate and delayed implant placement, *Int J Oral Maxillofac Implants* 16:713-721, 2001.
21. Buser D, Mericske-Stern R, Bernard JP et al: Long-term evaluation of non-submerged ITI implants. I: 8-year life table analysis of a prospective multicenter study with 2359 implants, *Clin Oral Implants Res* 8:161-172, 1997.
22. Boyne PT: Analysis of performance of root form endosseous implants placed in the maxillary sinus, *J Long Term Eff Med Implants* 3:143-159, 1993.
23. Weber HP, Crohin CC, Fiorellini JP: A 5-year prospective clinical and radiographic study of non-submerged dental implants, *Clin Oral Implants Res* 11:144-153, 2000.
24. Leonhardt A, Grondahl K, Bergstrom C et al: Long-term follow-up of osseointegrated titanium implants using clinical, radiographic and microbiological parameters, *Clin Oral Implants Res* 13:127-132, 2000.
25. Raghoebar FM, Timmenga NM, Reitsema H et al: Maxillary bone grafting for insertion of endosseous implants: results after 12-124 months, *Clin Oral Implants Res* 12:279-286, 2001.
26. Olson JW, Dent CD, Morris HF et al: Long-term assessment (5 to 71 months) of endosseous dental implants placed in the augmented maxillary sinus, *Ann Periodontol* 5:152-156, 2000.
27. Wallace SS, Froum SJ: Effect of maxillary sinus augmentation on the survival of endosseous dental implants: a systematic review, *Ann Periodontol* 8:328-343, 2003.
28. Degidi M, Piattelli M, Scarano A et al: Maxillary sinus augmentation with a synthetic cell-binding peptide: histological and histomorphometrical results in humans, *J Oral Implantol* 30:376-383, 2004.
29. Del Fabbro M, Testori T, Francetti L et al: Systematic review of survival rates for implants placed in the grafted maxillary sinus, *Int J Periodontics Restorative Dent* 24:565-577, 2004.
30. Butz SJ, Huys LW: Long-term success of sinus augmentation using a synthetic alloplast: a 20 patients, 7 years clinical report, *Implant Dent* 4:36-42, 2005.
31. Zijderveld SA, Zerbo IR, van den Bergh JP et al: Maxillary sinus floor augmentation using a beta-tricalcium phosphate (Cerasorb) alone compared to autogenous bone graft, *Int J Oral Maxillofac Implants* 20:432-440, 2005.
32. Maiorana C, Sigurta D, Mirandola A et al: Bone resorption around dental implants placed in grafted sinuses: clinical and radiologic follow-up after up to 4 years, *Int J Oral Maxillofac Implants* 20:261-266, 2005.
33. Cammack GV 2nd, Nevins M, Clem DS 3rd et al: Histologic evaluation of mineralized and demineralized freeze-dried bone allograft for ridge and sinus augmentations, *Int J Periodontics Restorative Dent* 25:231-237, 2005.
34. Gapski R, Neiva R, Oh TJ et al: Histologic analyses of human mineralized bone grafting material in sinus elevation procedures: a case series, *Int J Periodontics Restorative Dent* 26:59-69, 2006.
35. Herzberg R, Dolev E, Schwartz-Arad D: Implant marginal bone loss in maxillary sinus grafts, *Int J Oral Maxillofac Implants* 21:103-110, 2006.
36. Peleg M, Garg AK, Mazor Z: Healing in smokers versus nonsmokers: survival rates for sinus floor augmentation with simultaneous implant placement, *Int J Oral Maxillofac Implants* 21:551-559, 2006.
37. Aghaloo TL, Moy PK: Which hard tissue augmentation techniques are most successful in furnishing bony support implant placement? *Int J Oral Maxillofac Implants* 22:49-70, 2007.
38. Wheeler SL: Sinus augmentation for dental implants: the use of alloplastic materials, *J Oral Maxillofac Surg* 55:1287-1293, 1997.
39. Pietrokovski J: The bony residual ridge in man, *J Prosthet Dent* 34:456-462, 1975.
40. Misch CE: Divisions of available bone in implant dentistry, *Int J Oral Implantol* 7:9-17, 1990.
41. Misch CE: Treatment planning for the edentulous posterior maxilla. In Misch CE, editor: *Contemporary implant dentistry,* ed 2, St Louis, 1999, Mosby.
42. Misch CE: Bone character: second vital implant criterion, *Dent Today* 7:39-40, 1988.
43. Goodacre JC, Bernal G, Rungcharassaeng K et al: Clinical complications with implants and implant prostheses, *J Prosthet Dent 2*:121-132, 2003.
44. Misch CE, Qu Z, Bidez MW: Mechanical properties of trabecular bone in the human mandible: implications for dental implants treatment planning and surgical placement, *J Oral Maxillofac Surg* 57:700-706, 1999.
45. Leonardo de Vinci Codex Medicus Collection, Queen Elizabeth II, Windsor, England, ca l488.
45. Williams PI, Warwick R, editors: *Gray's anatomy*, ed 36, Philadelphia, 1980, WB Saunders.
46. Takahashi R: The formation of the human paranasal sinuses, *Acta Otolaryngol* 408:1-28, 1984.
47. Lang J, editor: *Clinical anatomy of the nose, nasal cavity and paranasal sinuses,* New York, 1989, Thieme.
48. Blitzer A, Lawson W, Friedman WH, editors: *Surgery of the paranasal sinuses,* Philadelphia, 1985, WB Saunders.
49. Anon JB, Rontal M, Zinreich SJ: *Anatomy of the paranasal sinuses,* New York, 1996, Thieme.
50. Stammberger H: History of rhinology: anatomy of the paranasal sinuses, *Rhinology* 27:197-210, 1989.
51. Kaufman E: Maxillary sinus elevation surgery: an overview, *J Esthet Restor Dent* 15:272-282, 2003.
52. Karmody CS, Carter B, Vincent ME: Developmental anomalies of the maxillary sinus, *Trans Am Acad Ophthalmol Otolaryngol* 84:723-728, 1977.
53. Moss-Salentija L: Anatomy and embryology. In Blitzer A, Lawson W, Friedman W, editors: *Surgery of the paranasal sinuses,* Philadelphia, 1985, WB Saunders.
54. Rice DH, Schaefer SD: *Endoscopic paranasal sinus surgery,* ed 3, Philadelphia, 2003, Lippincott Williams & Wilkins.
55. Ulm CW, Solar PG, Sellmann B et al: The edentulous maxillary alveolar process in the region of the maxillary sinus: a study of physical dimension, *Int J Oral Maxillofac Surg* 24:279-282, 1995.
56. Ulm CW, Solar P, Krennmar G et al: Incidence and suggested surgical management of septa in sinus lift procedures, *Int J Oral Maxillofac Implants* 10:462-465, 1995.

WWW.HIGHDENT.IR

57. Solar P, Geyerhofer U, Traxler H: Blood supply to the maxillary sinus relevant to sinus floor elevation procedures, *Clin Oral Implants Res* 10:34-44, 1999.
58. Elian N, Wallace S, Cho SC et al: Distribution of the maxillary artery as it relates to sinus floor augmentation, *Int J Oral Maxillofac Implants* 20:784-787, 2005.
59. Sharawy M, Misch CE: The maxillary sinus membrane—a histologic review (unpublished data), 2007.
60. Morgensen C, Tos M: Quantitative histology of the maxillary sinus, *Rhinology* 15:129, 1977.
61. Scadding GK, Lund VJ, Darby YC: The effect of long-term antibiotic therapy upon ciliary beat frequence in chronic rhinosinusitis, *J Laryngol Otol* 109:24-26, 1995.
62. Wilson R, Alton E: Upper respiratory viral infection and muccoiliary clearance, *Eur J Respir Dis* 70:272-279, 1987.
63. Holstrom M, Lund VJ, Scadding GK: Nasal ciliary beat frequency after nasal allergen challenge, *Am J Rhinology* 6:101-105, 1992.
64. Bell RD, Stone HE: Conservative surgical procedures in inflammatory disease of the maxillary sinus, *Otolaryngol Clin North Am* 9:175, 1976.
65. Zinreich SJ, Kennedy DW, Rosenbaum AE et al: Paranasal sinuses: CT imaging requirements for endoscopic surgery, *Radiology* 163:769-775, 1987.
66. Sandler NA, Johns FR, Braun TW: Advances in the management of acute and chronic sinusitis, *J Oral Maxillofac Surg* 54:1005-1013, 1996.
67. Cook HE, Haber J: Bacteriology of the maxillary sinus, *J Oral Maxillofac Surg* 45:1011-1014, 1987.
68. Jiang RS, Liang KL, Jang JW: Bacteriology of endoscopically normal maxillary sinuses, *J Larynogol Otol* 113:825-828, 1999.
69. Misch CE: Maxillary sinus augmentation for endosteal implants: organized alternative treatment plans, *Int J Oral Implantol* 4:49-58, 1987.
70. American Academy of Otolaryngology—Head and Neck Surgery: *Fact sheet: 20 questions about your sinuses.* Available at http://www.entnet.org/healthinfo/sinus/sinus_questions.cfm. Accessed October 7, 2007.
71. McAlister WH, Lusk R, Muntz HR: Comparison of plain radiographs and coronal CT scans in infants and children with recurrent sinusitis, *Am J Roentgenol* 153:1259-1264, 1989.
72. Tonje G, Bolin A, Eliasson I: Panoramic radiography of edentulous jaws: I. Frequency and distribution of pathological findings, *Dentomaxillofac Radiol* 9:21-25, 1980.
73. McGowan DA, Baxter PW, James J: *The maxillary sinus and its dental implications,* Oxford, 1993, Butterworth-Heinemann.
74. Worth HM: *Principles and practice of oral radiologic interpretation,* Chicago, 1963, Year Book.
75. Bolger WE, Butzin CA, Parsons DS: Paranasal sinus bony anatomic variations and mucosal abnormalities: CT analysis for endoscopic surgery, *Laryngoscope* 101:56-64, 1991.
76. Timmenga MN, Marius N: *Maxillary sinus floor elevation surgery: effects on maxillary sinus performance* [doctoral dissertation], Groningen, The Netherlands, 2003, University of Groningen.
77. McKenzie M: *Manual of diseases of the nose and throat,* London, 1980, Churchill Livingstone.
78. Maloney PL, Doku HC: Maxillary sinusitis of odontogenic origin, *J Can Dent Assoc* 34:591, 1968.
79. Smith D, Goycollea M, Meyerhoff WL: Fulminant odontogenic sinusitis, *Ear Nose Throat J* 58:411, 1979.
80. Daley DL, Sande M: The runny nose infection of the paranasal sinuses, *Infect Dis Clin North Am* 2:131, 1988.
81. Messerklinger W: *Endoscopy of the nose,* Baltimore, 1978, Urban & Schwartzenberg.
82. Zinreich SJ, Messerklinger W, Drettner B: The obstruction of the maxillary ostium, *Rhinology* 5:100-104, 1967.
83. Aust R, Drettner B: Oxygen tension in the human maxillary sinus under normal and pathological conditions, *Acta Otolaryngol* 78:264, 1974.
84. Bolzer WE, Kennedy DW: Changing concepts in chronic sinusitis, *Hosp Pract* 27:20, 1992.
85. Ponikau JU, Sherris DA, Kern EB: The diagnosis and incidence of allergic fungal sinusitis, *Mayo Clin Proc* 74:877-884, 1999.
86. Beninger MS, Mickleson SA: Functional endoscopic sinus surgery, morbidity and early results, *Henry Ford Hosp Med J* 38:5, 1990.
87. Dufour X, Kauffmann-Lacroix C, Ferrie JC et al: Paranasal sinus fungus ball: epidemiology, clinical features and diagnosis. A retrospective analysis of 173 cases from a single medical center in France, 1989-2002, *Med Mycol* 44:61-67, 2006.
88. Yoshiura K, Ban S, Hijiya K et al: Analysis of maxillary sinusitis using computed tomography, *Dentomaxillofac Radiol* 22:86, 1993.
89. Gardner DG: Pseudocysts and retention cysts of the maxillary sinus, *Oral Surg Oral Med Oral Pathol* 58:561-567, 1984.
90. Kudo K et al: Clinicopathological study of postoperative maxillary cysts, *J Jpn Stomatol Soc* 21:250-257, 1972.
91. Kubo I: A buccal cyst occurring after a radical operation of the maxillary sinus, *Z Otol (Tokyo)* 33:896, 1927.
92. Misch CM, Misch CE, Resnik RR et al: Postoperative maxillary cyst associated with sinus elevation procedure: a case report, *J Oral Implantol* 18:432-437, 1991.
93. Lockhart R, Ceccaldi J, Bertrand JC: Postoperative maxillary cyst following sinus bone graft: report of a case, *Int J Oral Maxillofac Implants* 15:583-586, 2000.
94. Blitzer A, Lawson W, Friedman W, editors: *Surgery of the paranasal sinuses*, Philadelphia, 1985, WB Saunders.
95. Blaschke FF, Brady FA: The maxillary antrolith, *Oral Surg Oral Med Oral Pathol* 48:187-191, 1979.
96. Evans J: Maxillary antrolith: a case report, *Br J Oral Surg* 13:73-77, 1975.
97. Crist RF, Johnson RI: Antrolith: report of case, *J Oral Surg* 30:694-695, 1972.
98. Karges MA, Eversol LR, Poindexter BJ: Report of case and review of literature, *J Oral Surg* 29:812-814, 1971.
99. Klokkevold PR, Ham TJ: How do smoking, diabetes and periodontitis affect outcomes of implant treatment? *Int J Oral Maxillofac Implants* 22:173-202, 2007.
100. Levin L, Schwartz-Arad D: The effect of cigarette smoking on dental implants and related surgery, *Implant Dent* 4:357-361, 2005.
101. Levin L, Herzberg R, Dolev E et al: Smoking and complications of onlay bone grafts and sinus lift operations, *Int J Oral Maxillofac Implants* 19:369-373, 2004.
102. Dietsh-Misch F, Cianciola L, Misch CE: Sinus grafting in a smoker population—a retrospective case series study, *Int J Oral Maxillofac Implants* (in press).
103. Barone A, Santini S, Sbordone L et al: A clinical study of the outcomes and complications associated with maxillary sinus augmentation, *Int J Oral Maxillofac Implants* 21:81-85, 2006.
104. Schwartz-Arad D, Herzberg R, Dolev E: The prevalence of surgical complications of the sinus graft procedure and their impact on implant survival, *J Periodontol* 75: 551-556, 2004.

WWW.HIGHDENT.IR

همیار دندانسازان

105. Ueda M, Kaneda T: Maxillary sinusitis caused by dental implants, report of 2 cases, *J Oral Maxillofac Surg* 50:285, 1992.
106. Quiney R, Brimble E, Hodge M: Maxillary sinusitis from dental osseointegrated implants, *J Laryngol Otol* 104:333, 1970.
107. Block MS, Kent JN: Sinus augmentation for dental implants, the use of autogenous bone, *J Oral Maxillofac Surg* 55:1281-1286, 1997.
108. Timmenga NM, Raghoebar GM, Boering G et al: Maxillary sinus function after sinus lifts for the insertion of dental implants, *J Oral Maxillofac Surg* 55:936-939, 1997.
109. Peterson LJ: Antibiotic prophylaxis against wound infections in oral and maxillofacial surgery, *J Oral Maxillofac Surg* 48:617-620, 1990.
110. Olson M, O'Connor M, Schwartz ML: Surgical wounds infection: a 5 year prospective study of 10,193 wounds at the Minneapolis VA Medical Center, *Ann Surg* 199:253, 1984.
111. Dent CD, Olson JW, Farish SE et al: The influence of preoperative antibiotics on success of endosseous implants up to and including stage II surgery: a study of 2641 implants, *J Oral Maxillofac Surg* 55(suppl 115):19-24, 1997.
112. Lebowitz AS: Antimicrobic therapy in rhinologic infection. In Goldsmith J, editor: *The principles and practice of rhinology,* New York, 1987, John Wiley & Sons.
113. Mulliken JB, Glowacki J, Kaban LB et al: Use of demineralized allogenic bone I implants for the correction of maxillocraniofacial deformities, *Ann Surg* 194:366-372, 1981.
114. Marx RE, Kline SN, Johnson RD et al: The use of freeze-dried allogenic bone in oral and maxillofacial surgery, *J Oral Surg* 39:264-274, 1981.
115. Jacobs MR, Felmingham D, Appelbaum PC et al: The Alexander Project 1998-2000: susceptibility of pathogens isolated from community-acquired respiratory tract infection to commonly used antimicrobial agents, *J Antimicrob Chemother* 52:229-246, 2003.
116. Snydor A, Gwaltney J, Cachetto DM et al: Comparative evaluation of cefuroxime axetil and cefaclor for treatment of acute bacterial maxillary sinusitis, *Arch Otolaryngol Head Neck Surg* 115:1430, 1989.
117. Gallagher DM, Epker BN: Infection following intraoral surgical correction of dentofacial deformities: a review of 140 consecutive cases, *J Oral Surg* 38:117-120, 1980.
118. Urist MR, Silverman BF, Buring K et al: The bone induction principle, *Clin Orthop Relat Res* 53:243-283, 1967.
119. Mabry TW, Yukna RA, Sepe WW: Freeze-dried bone allografts combined with tetracycline in the treatment of juvenile periodontitis, *J Periodontol* 56:74-81, 1985.
120. Beardmore AA, Brooks DE, Wenke JC et al: Effectiveness of local antibiotic delivery with an osteoinductive and osteoconductive bone-graft substitute, *J Bone Joint Surg Am* 87:107-112, 2005.
121. Petri WH: Osteogenic activity of antibiotic-supplemented bone allografts in the guinea pig, *J Oral Maxillofac Surg* 42:631-636, 1984.
122. Mabry RC: Corticosteroids in rhinology. In Goldsmith J, editor: *The principles and practice of rhinology,* New York, 1987, John Wiley & Sons.
123. Falck B, Svanholm H, Aust R: The effect of xylometazoline on the mucosa of human maxillary sinus, *Rhinology* 28:239-477, 1990.
124. Ragno JR, Szkutnik AJ: Evaluation of 0.12% chlorhexidine rinse on the prevention of alveolar osteitis, *Oral Surg Oral Med Oral Pathol* 72:524-526, 1991.
125. Lambert PM, Morris H, Ochi S: The influence of 0.12% chlorhexidine digluconate rinses on the incidence of infectious complications and implant success, *J Oral Maxillofac Surg* 55(suppl 5):25-30, 1997.
126. Tatum OH: The Omnii implant system. In Hardin JF, editor: *Clarke's clinical dentistry,* Philadelphia, 1984, Lippincott Williams & Wilkins.
127. Tatum OH Jr, Lebowitz MS, Tatum CA et al: Sinus augmentation: rationale, development, long term results, *N Y State Dent J* 59:43-48, 1993.
128. Jensen O: *The sinus graft,* Chicago, 1991, Quintessence.
129. Chiapasco M: Tecniche ricostruttive con innesti e/o osteotomie. In Chiapasco M, Romeo E: *Riabilitazione implanto-protesica dei casi complessi,* Torino, Italy, 2003, UTET ed.
130. Misch CE, Chiapasco, Jensen OT: Indications for and classifications of sinus bone grafts. In Jensen OT, editor: *The sinus bone graft,* ed 2, Chicago, 2006, Quintessence.
131. Summers RB: Maxillary implant surgery: the osteotome technique, *Compend Cont Educ Dent* 15:152-162, 1994.
132. Rosen PS, Summers R, Mellado JR et al: The bone-added osteotome sinus floor elevation technique: multicenter retrospective report of consecutively treated patients, *Int J Oral Maxillofac Implants* 14:853-858, 1999.
133. Worth HM, Stoneman DQ: Radiographic interpretation of antral mucosal changes due to localized dental infection, *J Can Dent Assoc* 38:111, 1972.
134. Palma VC, Magro-Filho O, de Oliveria JA et al: Bone reformation and implant integration following maxillary sinus membrane elevation: an experimental study in primates, *Clin Implant Dent Relat Res* 8:11-24, 2006.
135. Reiser GM, Rabinovitz Z, Bruno J et al: Evaluation of maxillary sinus membrane response following elevation with the crestal osteotome technique in human cadavers, *Int J Oral Maxillofac Implants* 16:833-840, 2001.
136. Malamed SF: Techniques of maxillary anesthesia. In Malamed S, editor: *Handbook of local anesthesia,* ed 3, St Louis, 1990, Mosby.
137. Reinert S, Lonig S, Bremerich A et al: Stability of bone grafting and placement of implants in the severely atrophic maxilla, *Br J Oral Maxillofac Surg* 41:249-255, 2003.
138. Wannfors K, Johnansson B, Hallman M et al: A prospective randomized study of 1- and 1-stage sinus inlay bone grafts: 1-year follow-up, *Int J Oral Maxillofac Implants* 15:625-632, 2000.
139. Wiltfang J, Schultze-Mosgau S, Nkenke E et al: Onlay augmentation versus sinus lift procedure in the treatment of the severely resorbed maxilla: a 5-year comparative longitudinal study, *Int J Oral Maxillofac Surg* 34:885-889, 2005.
140. Daelemans P, Hermans M, Godet F et al: Autologous bone graft to augment the maxillary sinus in conjunction with immediate endosseous implants: a retrospective study up to 5 years, *Int J Periodontics Restorative Dent* 17:27-39, 1997.
141. Keller EE, Tolman DE, Zuck SL et al: Mandibular endosseous implants and autogenous bone grafting in irradiated tissue: a 10-year retrospective study, *Int J Oral Maxollofac Implants* 12:800-813, 1997.
142. Hurzeler MB, Kirsch A, Ackermann KL et al: Reconstruction of the severely resorbed maxilla with dental implants in the augmented maxillary sinus—a 5

WWW.HIGHDENT.IR

year clinical investigation, *Int J Oral Maxillofac Implants* 11:466-475, 1996.
143. Raghoebar GM, Brouwer TJ, Reintsema H et al: Augmentation of the maxillary sinus floor with autogenous bone for the placement of endosseous implants: a preliminary report, *J Oral Maxillofac Surg* 51:1198-1203, 1993.
144. Momtaheni DM, Schweitzer K, Muenchinger F: Technique for stabilization of autogenous cancellous bone grafts in sinus lift procedures, *Oral Surg Oral Med Oral Pathol* 78:14-16, 1994.
145. Hirsch JM, Ericsson I: Maxillary sinus augmentation using mandibular bone grafts and simultaneous installation of implants—a surgical technique, *Clin Oral*
146. Chiapasco M, Romeo E, Vogel G: Three-dimensional reconstruction of a knife-edge edentulous maxilla by sinus elevation, onlay grafts, and sagittal osteotomy of the anterior maxilla: preliminary surgical and prosthetic results, *J Oral Maxillofac Implants* 13:394-399, 1998.
147. Jensen J, Simonsen EK, Sindet-Pedersen S: Reconstruction of the severely resorbed maxilla with bone grafting and osseointegrated implants. A preliminary report, *J Oral Maxillofac Surg* 48:27-32, 1990.
148. Misch CE, Dietsh F: Endosteal implants and iliac crest grafts to restore severely resorbed totally edentulous maxillae, a retrospective study, *J Oral Implantol* 20:100-110, 1994.
149. Whittaker JM, James RA, Lozada J et al: Histological response and clinical evaluation of heterograft and allograft materials in the elevation of the maxillary sinus of the preparation of endosteal implant sites. Simultaneous sinus elevation and root form implantation, an eight month autopsy report, *J Oral Implantol* 15:141-144, 1989.
150. Hurzeler MB, Quinones CR, Kirsch A et al: Maxillary sinus augmentation using different grafting materials and dental implants in monkeys, part III, *Clin Oral Implants Res* 8:401-411, 1997.
151. Jensen OT, Greer R: Immediate placement of osseointegrated implants into the maxillary sinus augmented with mineralized cancellous allograft and Gore-Tex, 2nd stage surgical and histological findings in time integration. In Tolman DE, editor: *Tissue integration in oral orthopedic and maxillofacial reconstruction changes*, Chicago, 1992, Quintessence.
152. Nishibori M, Betts NJ, Salama H et al: Short term healing of autogenous and allergenic bone grafts after sinus augmentation—a report of 2 cases, *J Periodontol* 65:958-966, 1994.
153. Valentini P, Abensur D, Wenz B et al: Sinus grafting with porous bone mineral (Bio-Oss) for implant placement: a 5-year study on 15 patients, *Int J Periodontics Restorative Dent* 20:245-253, 2000.
154. Noumbissi SS, Lozada JL, Boyne PJ et al: Clinical, histologic, and histomorphometric evaluation of mineralized solvent-dehydrated bone allograft (Puros) in human maxillary sinus grafts, *J Oral Implantol* 31:171-179, 2005.
155. Kassolis JD, Rosen PS, Reynolds MA: Alveolar ridge and sinus augmentation utilizing platelet-rich plasma in combination with freeze-dried bone allograft: case series, *J Periodontol* 71:1654-1661, 2000.
156. Mangano C, Bartolucci EG, Mazzocco C: A new porous hydroxyapatite for promotion of bone regeneration in maxillary sinus augmentation: clinical and histologic study in humans, *Int J Oral Maxillofac Implants* 18:23-30, 2003.
157. Mazor Z, Peleg M, Gross M: Sinus augmentation for single tooth replacement in the posterior maxilla: a 3-year follow-up clinical report, *Int J Oral Maxillofac Implants* 14:55-60, 1999.
158. Mazor Z, Peleg M, Garg AK et al: The use of hydroxyapatite bone cement for sinus floor augmentation with simultaneous implant placement in the atrophic maxilla: a report of 10 cases, *J Periodontol* 71:1187-1194, 2007.
159. Tadjoedin ES, de Lange GL, Bronckers AL et al: Deproteinized cancellous bovine bone (Bio-Oss) as bone substitute for sinus floor elevation: a retrospective, histomorphometrical study of five cases, *J Clin Periodontol* 30:261-270, 2003.
160. Sartori S, Silvestri M, Forni F et al: Ten-year follow up in a maxillary sinus augmentation using anorganic bovine bone (Bio-Oss): a case report with histomorphometric evaluation, *Clin Oral Implants Res* 14:369-372, 2003.
161. Furusawa T, Mizunuma K: Osteoconductive properties and efficacy of resorbable bioactive glass as a bone grafting material, *Implant Dent* 6:93-101, 1997.
162. Tadjoedin ES, de Lange GL, Holzmann PJ et al: Histological observations on biopsies harvested following sinus floor elevation using a bioactive glass material of narrow size range, *Clin Oral Implants Res* 11:334-344, 2000.
163. Peleg M, Mazor Z, Garg AK: Augmentation grafting of the maxillary sinus and simultaneous implant placement in patients with 3 to 5 mm of residual alveolar bone height, *Int J Oral Maxillofac Implants* 14:549-556, 1999.
164. Fugazzotto PA, Vlassis J: Long-term success of sinus augmentation using various surgical approaches and grafting materials, *Int J Oral Maxillofac Implants* 13:52-58, 1998.
165. Moy PK, Lundgren S, Holmes RE: Maxillary sinus augmentation: histomorphometric analysis of graft materials for maxillary sinus floor augmentation, *J Oral Maxillofac Surg* 51:857-862, 1993.
166. Wetzel AC, Stich H, Caffesse RG: Bone apposition onto oral implants in the sinus area filled with different grafting materials—a histological study in beagle dogs, *Clin Oral Implants Res* 6:155-163, 1995.
167. Wallace SS, Froum SJ, Tarnow DP: Histologic evaluation of sinus elevation procedure a clinical report, *Int J Periodontics Restorative Dent* 16:47-51, 1996.
168. Schwartz Z, Goldstein M, Raviv E et al: Clinical evaluation of demineralized bone allograft in a hyaluronic acid carrier for sinus lift augmentation in humans: a computed tomography and histomorphometric study, *Clin Oral Implants Res* 18:204-211, 2007.
169. Landi L, Pretel RW, Hakimi NM et al: Maxillary sinus floor elevation using a combination of DFDBA and bovine-derived porous hydroxyapatite: a preliminary histologic and histomorphometric report, *Int J Periodontics Restorative Dent* 20:574-583, 2000.
170. Boeck-Neto RJ, Gabrielli M, Lia R et al: Histomorphometrical analysis of bone formed after maxillary sinus floor augmentation by grafting with a combination of autogenous bone and demineralized freeze-dried bone allograft or hydroxyapatite, *J Periodontol* 73:266-270, 2002.
171. Nevins M, Kirker Head C et al: Bone formation in the goat maxillary sinus induced by absorbable collagen sponge implants impregnated with recombinant human bone morphogenetic protein-2, *Int J Periodontics Restorative Dent* 16:9-19, 1996.

WWW.HIGHDENT.IR

همیار دندانسازان

172. Danesh-Meyer MJ, Filstein MR, Shanaman R: Histological evaluation of sinus augmentation using platelet rich plasma (PRP): a case series, *J Int Acad Periodontol* 3:48-56, 2001.
173. Wiltfang J, Schiegel KA, Schultze-Mosgau S et al: Sinus floor augmentation with beta-tricalciumphosphate (beta-TCP): does platelet-rich plasma promote its osseous integration and degradation, *Clin Oral Implants Res* 12:213-218, 2003.
174. Tolman DE: Advanced residual ridge resorption, surgical management, *Int J Prostodont* 6:118, 1993.
175. Loukota RA, Isaksson SG, Linner EL et al: A technique for inserting endosseous implants in the atrophic maxilla in a single stage procedure, *Br J Oral Maxillofac Surg* 30: 46-49, 1992.
176. Block MS, Kent JN: Maxillary sinus grafting for totally and partially edentulous patients, *J Am Dent Assoc* 124:139, 1993.
177. Rabie ABM, Lie Ken Jie KP: Integration of endochondral bone grafts in the presence of demineralized bone matrix, *Int J Oral Maxillofac Surg* 25:311-318, 1996.
178. Misch CE, Dietsh F: Subantral augmentation in *Macaca fascicularis*—pilot study, Am Acad Implant Dent 40th Mtg Abs, *J Oral Implantol* 17:340, 1991.
179. Misch CE: Maxillary sinus left with subantral augmentation. In Misch CE, editor: *Contemporary implant dentistry,* St Louis, 1993, Mosby.
180. Wittbjer J, Palmer B, Robbin M et al: Osteogenetic activity in composite grafts of demineralized compact bone and marrow, *Clin Orthop* 173:229, 1993.
181. Marx RE, Saunders TR: Reconstruction and rehabilitation of cancer patients. In Fonseca RJ, Davis WH, editors: *Reconstructive preprosthetic oral and maxillofacial surgery,* Philadelphia, 1986, WB Saunders.
182. Froum SJ, Wallace SS, Elian N et al: Comparison of mineralized cancellous bone allograft (Puros) and anorganic bovine bone matrix (Bio-Oss) for sinus augmentation: histomorphometry at 26 to 32 weeks after grafting, *Int J Periodontics Restorative Dent* 26:543-551, 2006.
183. Vlassis JM, Hurzeler MB, Quinones CR: Sinus lift augmentation to facilitate placement of non submerged implants—a clinical and histological report, *Pract Periodontics Aesthet Dent* 5:15-23, 1993.
184. Marx RE, Snyder RM, Kline SN: Cellular survival of human marrow during placement of marrow cancellous bone grafts, *J Oral Surg* 37:712-718, 1979.
185. Merkx MA, Maitha JC, Stoelinga PJ: Assessment of the value of anorganic bone additives in sinus floor augmentation: a review of clinical reports, *Int J Oral Maxillofac Surg* 32:1-6, 2003.
186. Aimetti M, Romagnoli R, Ricci G et al: The effect of macrolacerations and microlacerations of the sinus membrane as determined by endoscopy, *Int J Periodontics Restorative Dent* 21:581-589, 2001.
187. Krennmair G, Ulm CW, Lugmayr H et al: The incidence, location and height of maxillary sinus septa in the edentulous and dentate maxilla, *J Oral Maxillofac Surg* 57:667-671, 1999.
188. Kim MJ, Jung UW, Kim CS et al: Maxillary sinus septa: prevalence, height, location and morphology: a reformatted computed tomography scan analysis, *J Periodontol* 77:903-908, 2006.
189. Betts NJ, Miloro M: Modification of the sinus lift procedure for septa in the maxillary antrum, *J Oral Maxillofac Surg* 52:332-333, 1994.
190. Gurr P, Callahan V, Baldwin D: Laser-Doppler blood flowmetry measurement of nasal mucosa blood flow after injection of the greater palatine canal, *J Laryngol Otol* 110:124-128, 1996.
191. Flanagan D: Arterial supply of maxillary sinus and potential for bleeding complication during lateral approach sinus elevation, *Implant Dent* 14:336-339, 2005.
192. Walike JW, Chinn J: Evaluation and treatment of acute bleeding from the head and neck, *Otolaryngol Clin North Am* 12:455, 1979.
193. Olson DEL, Rosgon BM, Hilsinger RL: Radiographic comparison of three nasal saline irrigation, *Laryngoscope* 112:1394-1398, 2002.
194. Brånemark P-I, Adell R, Albrektsson T et al: An experimental and clinical study of osseointegrated implants penetrating the nasal cavity and maxillary sinus, *J Oral Maxillofac Surg* 42:497-505, 1984.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 39

Mandibular Donor Block Bone Grafts: Symphysis and Ramus

Carl E. Misch

INTRODUCTION

Treatment plans in implant dentistry in the 1980s often used existing bone volume to determine the location and type of implant abutment. In abutment bone (Division A), root forms were inserted; in bone of moderate width (Division B), blade implants were placed; and in inadequate height of bone (Division C–h), subperiosteal implants were the treatment options (Figure 39-1). Today, the final prosthesis type and design is first determined, followed by the ideal implant position, number, and size. Often the bone available is inadequate to perform the ideal, predictable treatment and build a proper foundation. As a consequence, bone grafting has become a more frequent modality to achieve long-term success.

In addition to the biomechanical and functional needs of a prosthesis, there are often esthetic considerations. Bone grafting is often necessary to place the implant in the proper location for an ideal esthetic result. The soft tissue drape often needs enhancement in the esthetic zone. The bone foundation sets the tone for the soft tissue drape (Figure 39-2). Therefore when ideal crown contours (FP-1) and soft tissue are desired, bone augmentation is often an important aspect of the treatment.

As a result of biomechanical-based foundations and esthetic desires, a primary diagnostic consideration for implant prostheses is the available bone in the edentulous span. The placement of endosteal dental implants requires adequate bone volume at the desired locations for ideal prosthetic support. If inadequate bone exists, several surgical techniques may be used to reconstruct the deficient ridge for implant placement. The number of key factors present and the geometry of a bony defect are important considerations in the selection of a modality for ridge augmentation.[1] The fewer the number of remaining bony walls, the greater the need for osteopromotive techniques. Although allografts and guided bone regeneration techniques have been used

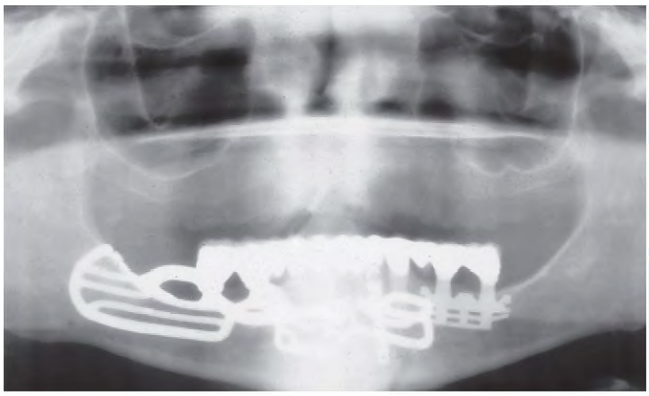


Figure 39-1 In the 1970s and 1980s, implants were often designed to adapt to the existing bone volume. Root forms were used in abundant bone (as in the anterior mandible), blade implants were inserted in narrow bone volume *(right side of radiograph)*, and subperiosteal implants were placed in inadequate bone volumes *(left side of radiograph)*.

Figure 39-2 Root form implants were placed in existing bone volumes in the 1980s. This implant prosthesis has functioned for 20 years but is an esthetic failure.

WWW.HIGHDENT.IR

همیار دندانسازان

predictably in slight-to-moderate bone regeneration (primarily for inadequate width), these methods have limitations and have been found to produce less favorable results in the treatment of larger bone deficiencies.[2-13] Autologous cortical/trabecular bone grafts may be considered the gold standard in the repair of moderate to severe alveolar atrophy and bone defects.[14-28]

The use of iliac crest autologous bone blocks with osteointegrated implants was originally presented in the literature by Brånemark et al.[29] and is now a well-accepted procedure in oral and maxillofacial rehabilitation. Although the iliac crest is often used in major jaw reconstruction for implants,[30-34] it has the disadvantages of higher costs, alteration of ambulation, and the need for hospitalization and general anesthesia.[35]

Mandibular symphysis donor site grafts were originally published to correct intraoral birth defects, such as cleft palates.[36,37] Misch et al. extended the indications with mandibular symphysis and ramus block bone grafts for use of endosteal dental implants in 1992 (Figure 39-3).[38] In the repair of more localized alveolar defects, cortical bone grafts from the mandible offer several benefits.[20,38-41] The obvious advantage of intraoral versus extraoral donor grafts is their convenient surgical access. The proximity of donor and recipient sites can reduce operative and anesthesia time, making them ideal for outpatient implant surgery. There is no cutaneous scar associated with extraoral donor sites. In addition, patients report minimal donor site discomfort, and these areas may offer a decreased morbidity from graft harvest compared with extraoral locations.[19,20,38-45]

Bone harvested from the maxillofacial region appears to have inherent biological benefits for augmentation. Research suggests that this may be attributed to the embryologic origin of the donor bone.[43,46-51] The majority of bones in the skeleton are of endochondral origin (from a cartilaginous precursor). With the exception of alveolar bone, the maxilla and body of the mandible develop intramembranously, whereas the condyles develop by endochondral bone formation.[52] Experimental evidence has shown that grafts from membranous bone show less resorption than endochondral bone.[46,47,49,53-56] Although cancellous grafts revascularize more rapidly than cortical grafts,[14] cortical membranous grafts revascularize more rapidly than endochondral bone grafts with a thicker cancellous component.[48,57]

Early revascularization of membranous bone grafts has been suggested as an explanation for the improved maintenance of graft volume.[48,56-58] Another hypothesis is that bone of ectomesenchymal origin, such as the mandible, has a better potential for incorporation in the maxillofacial region because of a biochemical similarity in the protocollagen of the donor and recipient bone.[60] However, more recent research suggests that grafted bone, independent from its embryogenic origin, will mimic the properties of the recipient bone.[60] The inductive capacity of cortical grafts is explained by

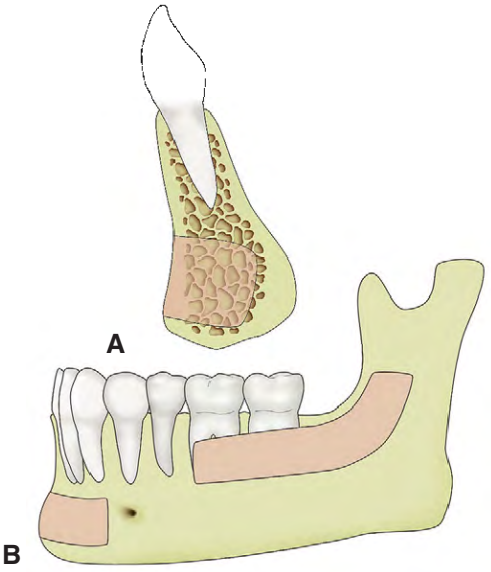


Figure 39-3 **A,** The symphysis block graft is usually 5 mm below the roots of the anterior teeth and extends to the lingual plate. **B,** The symphysis and ramus region may be harvested for intraoral defects.

their higher concentration of bone morphogenetic proteins,[61-63] and bone from the maxillofacial skeleton contains increased concentrations of growth factors, which may lead to a greater capacity for bone repair and graft retention.[64] Another hypothesis is that the improved survival of craniofacial bone grafts is simply caused by their three-dimensional structure.[65,66] Because these grafts have a thicker cortical layer, they resorb more slowly.[53,54,57,67]

An emphasis has been placed on the transplantation of viable osteoprogenitor cells from trabecular marrow grafts.[18] The concept has been that the majority of osteoblasts are present in trabecular bone. Because these cells are responsible for the formation of new bone, it is logical that trabecular grafts are advantageous. However, because of the significant resorption associated with corticocancellous block grafts from endochondral donor sites, these have lost favor in the treatment of mandibular continuity defects and ridge augmentation for soft tissue–borne prostheses.[16,18,55-57,68,69] It has been observed by Misch and others that the cortical/trabecular blocks harvested from the ilium have greater bone volume after initial healing compared with inlay trabecular grafts.[57] In addition, when endosteal implants are inserted within the first year, the bone is maintained long term. It appears the microarchitecture of the bone graft (cortical versus trabecular) and implant insertion to stimulate the bone is more important than whether the bone graft is from endochondial or mesenchymal origin.

Mandibular donor bone grafts, which are primarily cortical bone, exhibit little volume loss and show good incorporation at short healing times.[57] There are more mature bone cells (osteocytes) in the cortical bone,

WWW.HIGHDENT.IR

more growth factors, including insulin-like growth factors 1 and 2, and more bone morphogenetic protein. As important, the cortical bone acts like a barrier membrane and prevents resorption of the graft site. This permits the blood vessels from the host bone to invade the graft and bring osteoblasts into the graft along the sides of the invading blood vessels.

The cortical bone has more growth factors to encourage blood vessel growth from the host bone, including transforming growth factors. There are at least 50 known growth factors in this category (which includes bone morphogenetic proteins). Vascular endothelial growth factor is also present, which contributes to new blood vessel growth. Implant placement shortly after graft incorporation has a stimulating effect on the bone, maintaining the augmented bone volume and preventing further loss.[70-73] In addition, the dense structure of the cortical portion of the graft offers the benefit of improved implant stability during placement and healing and may even improve interfacial stress transmission upon implant loading.[74-76]

Over the years, intraoral cortical/trabecular block bone grafts have been used for residual bone augmentation prior to implant placement with extremely favorable results.[19-23,25,26,38-45,77-92] Block-type grafts may be harvested from the residual ridge, mandibular symphysis, body, or ramus area (Figure 39-4). Donor grafts may be harvested from the symphysis region of the edentulous mandible while placing implants in the region, provided there has been minimal atrophy. Implant placement in the lower jaw is then limited to the available bone superior to the graft harvest or postponed until healing of the donor site has occurred. The amount of bone available in many cases is limited only by the imagination of the surgeon. However, a common mistake in comprehensive treatment planning is to overestimate the volume of attainable bone graft from intraoral donor sites. It is prudent to consider extraoral donor sites for grafting alveolar defects, when more than a four-tooth span must be augmented (especially when height augmentation is required).

PREOPERATIVE EVALUATION OF HOST SITE

A comprehensive evaluation of the host graft site is necessary for planning of the surgery. This includes the graft size, esthetic concerns, soft and hard tissue topography, and the periodontal and endodontic health of the adjacent teeth.[38,93,94] The host site may be evaluated in width, height, and length. The most predictable bone augmentation sites require only the width dimension and extend for only one tooth. This provides mesiodistal lateral walls of bone, an apical wall, and a medial wall of host bone. A one-tooth span provides ease of soft tissue manipulation and minimal risk of incision line opening. Tooth-borne transitional restorations are easily fabricated. The donor bone harvest permits a complete fill of the region with an autograft. The least predictable bone graft sites are more than four teeth in length and require more than 5 mm of height and width of bone because the host site has primarily one wall of bone at the base and incision line complications, transitional prosthesis, inadequate autograft from the donor site combine to increase the overall risks.

The mesiodistal size of the host graft site with an intraoral harvest is most often four adjacent teeth or less in length and only deficient in width. However, in some larger mandibles with multiple harvest sites, a full-arch maxilla may be augmented in width (Figure 39-5). The mesiodistal size for a host site that requires height and width with an intraoral harvest is most often two adjacent teeth or less in length, although a span of four adjacent teeth may be restored with multiple harvest sites.

When an FP-1 prosthesis is the treatment of choice, the adjacent teeth next to the host graft site should have bone on the roots to a level within 2 mm of the cement-enamel junction. When a bone graft is placed adjacent to a tooth root (rather than bone), the graft most often resorbs to the level of the existing bone on the adjacent tooth root. Therefore, in a one-intratooth defect, a line drawn from each bone level on the adjacent roots is the most bone height that can be predictably expected.

When an ideal bone graft volume next to a tooth with vertical bone loss on the root is required, there are several options to accomplish this goal more predictably. Orthodontic extrusion of the natural tooth with bone loss can elevate the height of bone to the desired level. After orthodontic extrusion, a crown and often endodontic therapy is required. On occasion, extraction of the tooth and a bone graft extending to the next tooth (when the bone is in the correct position on that tooth) is another option.

In esthetic areas, an emphasis must be placed on not only providing adequate osseous volume, but also developing a soft tissue profile for the final implant restoration.[93] When the soft tissue region is in the esthetic zone, an increased height and thickness of keratinized mucosa before the bone graft is of benefit. A connective tissue graft is a common method to increase the desired tissue over the graft site before bone grafting. The soft tissue of the recipient site must then be completely healed for several months before bone graft surgery. Removal of foreign bodies, soft tissue surgery, or tooth extractions should be completed at least 12 weeks before bone grafting. This period allows vascularization and mature elastic fibers to be present in the soft tissue before the bone augmentation process and reduces the risk of incision line opening. Soft tissue surgical procedures may be planned in conjunction with implant placement or implant stage II uncovery when the soft tissue drape is out of the esthetic zone.

WWW.HIGHDENT.IR

همیار دندانسازان

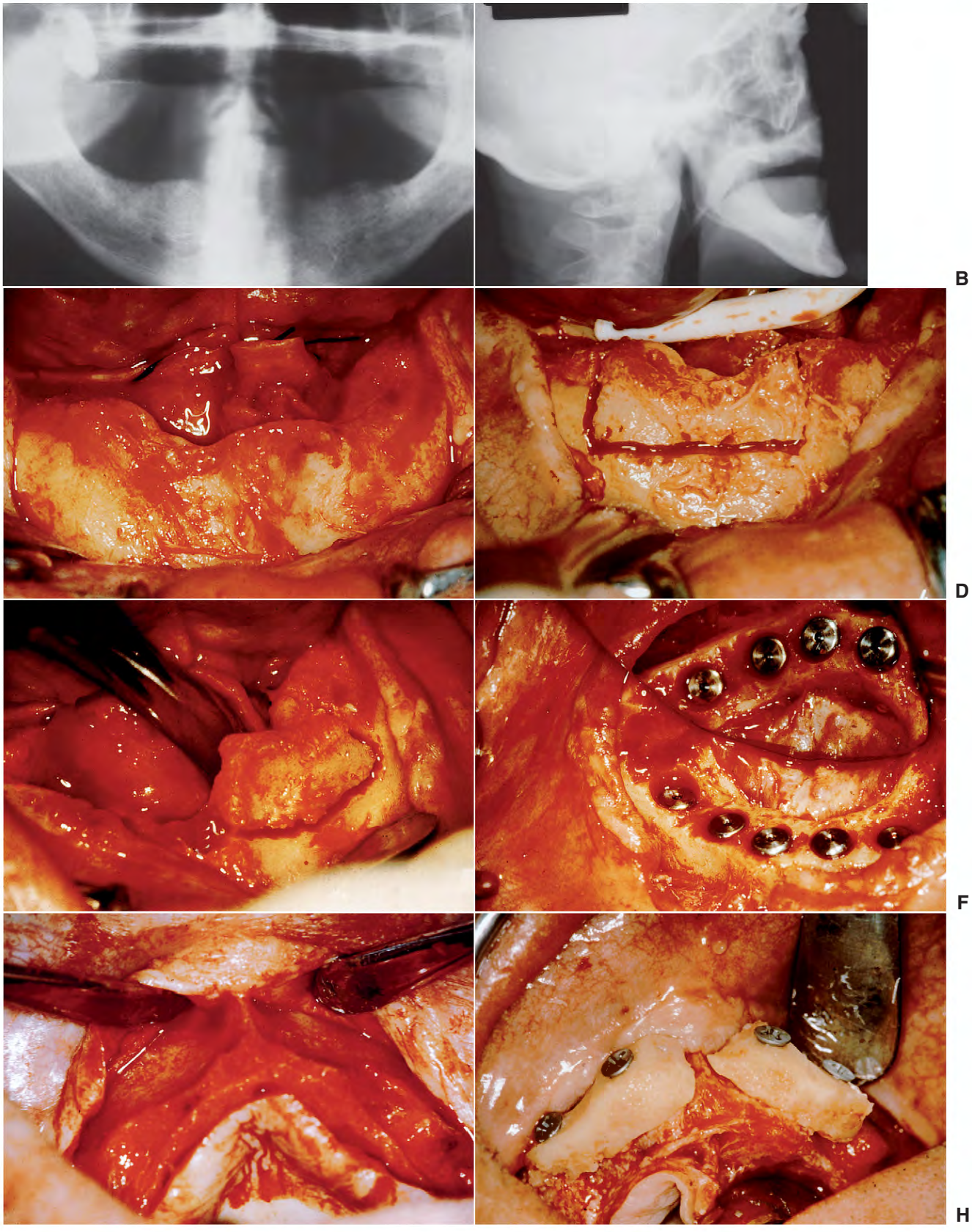
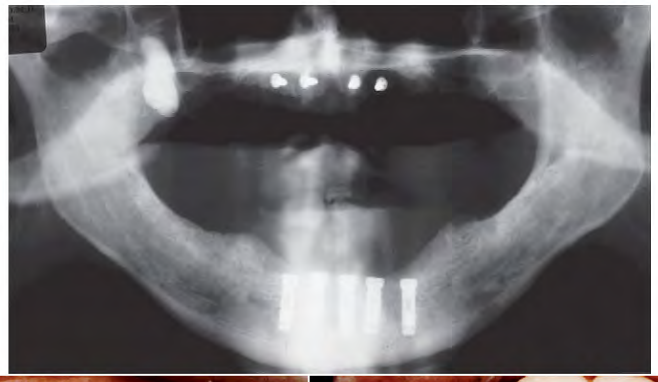


Figure 39-4 **A,** A panoramic radiograph of an edentulous maxilla and mandible (except for an impacted molar). **B,** A lateral cephalogram of the patient in Figure 39-3, *A*. The anterior maxilla and mandible is Division B bone volume. **C,** The soft tissue in the mandibular symphysis is reflected. **D,** A block of bone is outlined as a donor graft from the crest of the ridge. **E,** A second bone block is harvested from the crest. **F,** After osteoplasty, anterior implants are placed in the mandible. **G,** The maxillary anterior recipient ridge is exposed and decorticated. **H,** The two blocks are fixated to the anterior maxilla.

Continued

WWW.HIGHDENT.IR

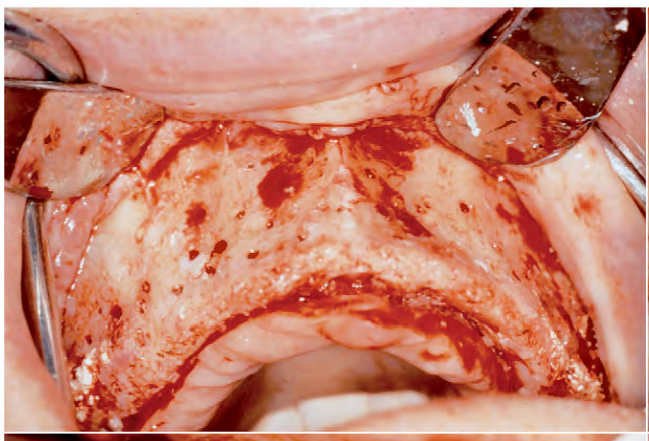


I

J

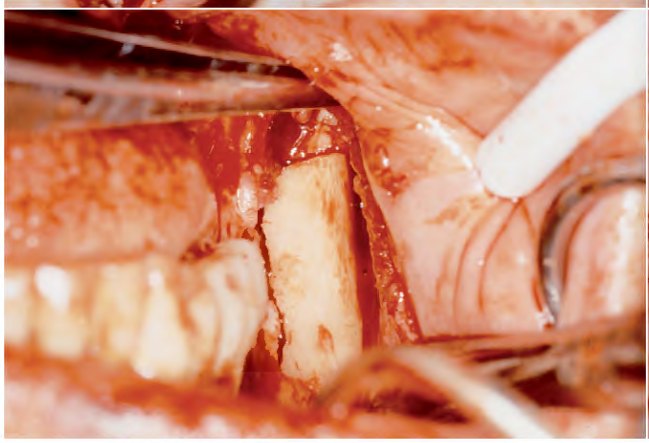
K

Figure 39-4, cont'd **I,** An immediate postoperative radiograph of the mandibular implants and maxillary block grafts. **J,** After sinus grafts and impacted molar removal, 10 implants were inserted into the grafted maxilla. **K,** A fixed prosthesis was delivered to the maxillary arch.

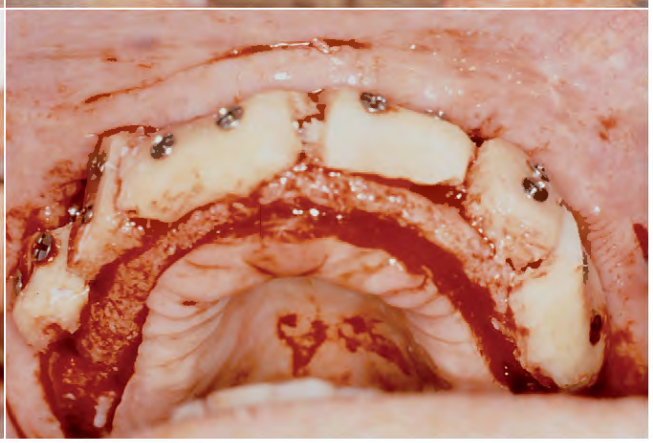


A

B



C



D

Figure 39-5 **A,** An edentulous maxilla inadequate in width may be augmented with block grafts harvested from multiple donor sites. **B,** A block bone graft is first harvested from the symphysis. **C,** A block bone graft from the ramus is then harvested. **D,** The multiple block grafts are fixated to the maxilla.

WWW.HIGHDENT.IR

همیار دندانسازان

The width and height requirements for augmentation will influence the donor site selected from the mandible. As a general rule, when more than 4 mm of width is desired (C–w bone volume), the mandibular symphysis is the most common donor site. A mandibular ramus is selected as a donor site when the bone graft width is less than 4 mm (Division B to B–w bone volume). When an augmentation for height is required, the most common site is the mandibular symphysis and includes its cortical inferior border (Figure 39-6).

Another influencing factor for the donor site is the location of the host/recipient site. When an anterior mandible is the recipient site, the symphysis is the donor site. A posterior mandible uses the ramus site (whenever possible). A maxillary host site uses the mandibular ramus, when possible, because a ramus graft has fewer postoperative complications of the donor site.

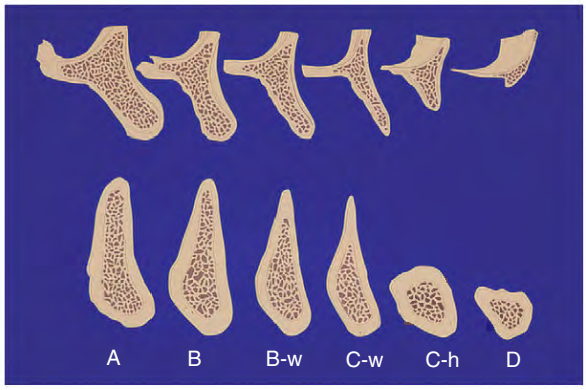


Figure 39-6 Division B and B–w bone volumes are deficient in width and are often augmented with ramus block bone grafts or barrier membranes and particulate grafts. Divisions C–w and C–h bone volumes are often grafted with symphysis block bone grafts.

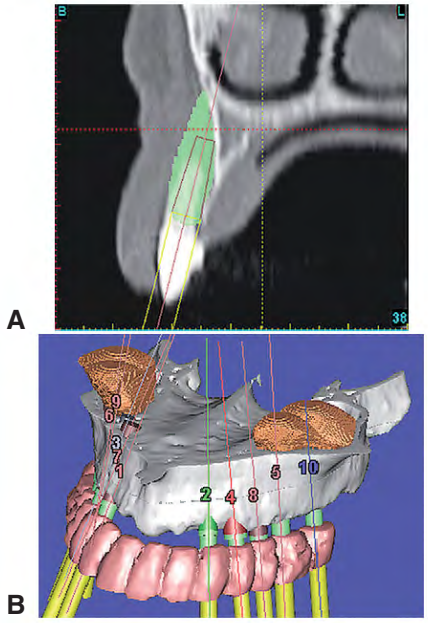


Figure 39-7 A computed tomography scan may be used to evaluate the host site volume required for proper rehabilitation.

The initial radiographic examination of the host site may include periapical, panoramic, or lateral cephalometric views and computed tomography (CT) scans. A lateral cephalometric view is useful in determining the anteroposterior (A-P) (width) dimension of the anterior mandible at the midline when an augmentation for width is required from this donor site. CT with reformatted images are most useful in preoperative planning and ridge mapping for both the host site and the donor site (Figure 39-7).[95-97] Mounted casts are also a benefit to allow the clinician to evaluate the ridge morphology in relationship to the adjacent teeth and opposing dentition. A diagnostic wax-up of the reconstructed ridge and restored dentition is advantageous in determining the graft width and position requirements and analyzing the occlusion. It is also used for fabrication of templates for CT, graft positioning, and implant surgery.[38,41,95,96] A CT model may also be generated to study the three-dimensional (3D) aspect of the host site. A mock surgery may be performed of the donor site, and this CT model may also be used to help recontour the donor bone graft to fit the model prior to the intraoral fixation (Figure 39-8).

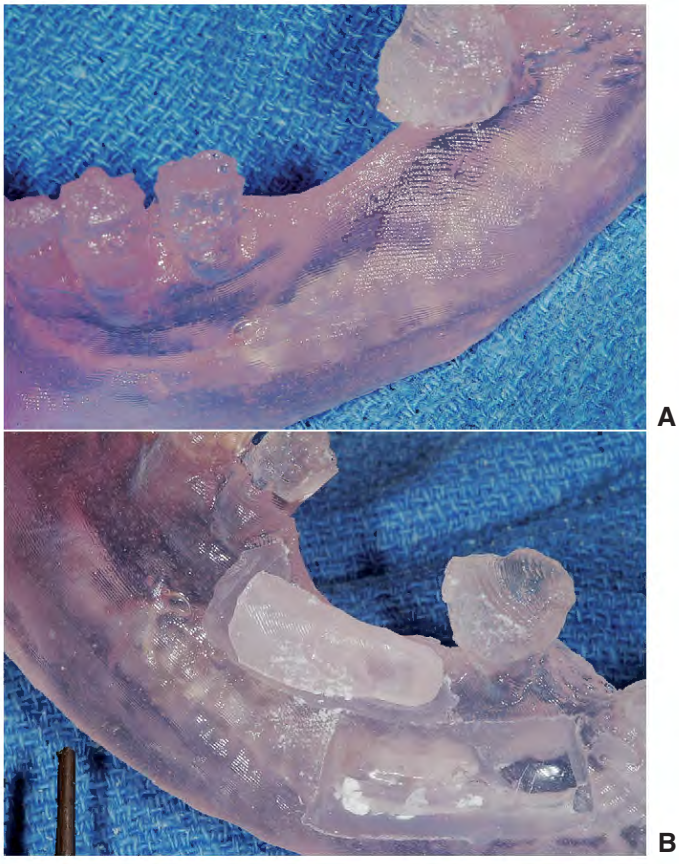


Figure 39-8 **A,** A computed tomography (CT) bone model may be used to evaluate the host donor sites before the surgery. **B,** The CT model may be used to demonstrate or practice ahead of the surgical procedure.

WWW.HIGHDENT.IR

Surgery

Preparation of Host Site

The recipient site should be completely exposed and prepared for grafting before harvesting the donor bone. In this manner, the dimensions of the graft to completely restore the bone defect may be evaluated, prepared, and measured. This approach also reduces the time period the donor graft is stored before fixation to the host site.

The concepts presented in the chapter on keys for bone grafting are incorporated into every step of the bone block grafting procedure. Incisions to surgically expose the recipient site are usually made within attached, keratinized tissue. The crestal incision in the edentulous host site is most often to the lingual aspect of the ridge, especially in the maxilla. This provides more keratinized tissue facially and reduces the risk of sutures tearing through the tissue if edema occurs after surgery, which results in incision line opening (Figure 39-9, *A*). When a block bone graft is anticipated, the vertical primary incisions usually are made one tooth distal to the tooth adjacent to the defect and include the distal interdental papilla. This provides a broader flap, which aids in primary closure and reduces incision line opening. The vertical release incision extends to the mucogingival junction (MGJ), not beyond. The soft tissue reflection of the flap distal to the graft site is a split-thickness reflection to aid the initial soft tissue healing and reduce incision line opening (see Figure 39-9, *B* to *D*).

Complete flap coverage of the block graft and tension-free wound closure are essential to achieve predictable results, and the soft tissue procedure to accomplish this goal begins before the donor graft is harvested. The traditional methods of flap release are to reflect the flap to a greater extent, exposing more host bone beyond the dimensions of the graft site, often beyond the opposing landmarks of the maxilla and including the inferior and lateral piriform rims of the nose and even the infraorbital foramen. A series of parallel incisions to the crestal incision would then be made through the periosteum of the facial flap. This procedure does allow the facial flap to advance several millimeters. However, larger grafts can not be covered without tension on the incision line. In addition, because the muscle layers are still in the facial flap, the tension and initial healing process often retract the flap and result in incision line opening, which is the most common complication of bone augmentation.

The submucosal space technique was first described by Misch to cover large block bone grafts from the iliac crest. This procedure reflects a full-thickness flap over the graft site and at least 5 mm above the height of the MGJ. The periosteum and tissues 5 mm above the MGJ remain on the bone and are not reflected. The facial flap is then lifted and 3 to 5 mm above the depth of the MGJ and a scalpel incises through the periosteum 1 to 2 mm deep, parallel to the crestal incision and extends over and beyond the vertical release incision. After the incision is made through the periosteum, pointed tissue scissors (Metzenbaum) may be introduced into the periosteal incision for 10 to 15 mm or more, parallel to the surface mucosa with the blades of the scissors closed, so the facial flap thickness is 3 to 5 mm. The tissue scissors are then opened, and this blunt dissection of the tissue allows the muscles to be separated from the flap and creates a submucosal space. With the periosteum, tissues and muscles attached to the bone on one side and a 3- to 5-mm-thick facial flap on the other side, the facial flap may now advance the depth of the submucosal space, 10 mm or more. This dramatically increases the ability to advance the soft tissue flap over a block graft (see Figure 39-9, *E* to *I*).

The advantage of the submucosal space technique is that a split-thickness flap is created and maintains the muscles on the periosteum, which is attached to the bone above the contours of the host site. Because the muscle attachments are the primary source of vascularization to the periosteum and the periosteum is the primary source of blood supply to the cortical bone, the blood supply of the cortical bone beyond the graft remains undisturbed. Muscle healing is a primary cause of flap retraction and incision line opening. With this technique, the muscles are no longer attached to the facial flap and the initial soft tissue is reduced. Because the flap may be advanced more than 10 mm, there is no tension on the incision line and sutures, which also reduces the risk of incision line opening.

Soft tissue procedures to enhance graft coverage will result in a loss of vestibular depth. The reduced vestibular depth is rarely an esthetic concern, and when the restoration is implant retained, the prosthesis does not rely on a valve seal for primary retention (as in a complete denture). Advancement of the facial flap for graft coverage will also often result in a reduction in keratinized mucosa over the facial of the ridge crest. In some cases, soft tissue grafts may be necessary, or more often, the attached mucosa may be repositioned facially at the stage II implant uncovery surgery.

The next step in the preparation of the host site is perforation of the lateral and crestal aspects of the host bone with a small-diameter drill, equal to or smaller than the drill size that corresponds to the bone screws used to fixate the donor bone (1.4 mm diameter). The holes are 3 to 5 mm apart in the entire area of the desired augmentation (Figure 39-9, *J*, *K*). The perforations in the bone are created under copious amounts of saline at 2500 rpm and penetrate both the facial and lingual plates of bone in the region of the graft, especially when augmentation is desired on both sides of the residual ridge. This procedure increases the availability of osteogenic cells, accelerates revascularization, increases the regional acceleratory phenomenon (RAP), and improves graft union.[97-99]

WWW.HIGHDENT.IR

همیار دندانسازان

The drill sites perforate the lingual plate. So that the fixation screw does not bottom out and strip the host bone and the dense, a cortical lingual plate ensures that the bone screws secure the bone block during the initial healing period. The host site is also recontoured with a large, pear-shaped carbide bur to improve the graft-to-recipient congruency. Ideally, the donor bone block should be slightly recessed in the host bone 1 to 2 mm, surrounded by a bony margin (Figure 39-9, *L*). After the block bone graft harvest and fixation, the tissues are approximated for primary closure (Figure 39-9, *M* to *S*). After a healing period of 4 to 6 months, the implants may be inserted (Figure 39-9, *T* to *V*).

MANDIBULAR SYMPHYSIS DONOR SITE

The ideal goal of a donor block harvest is to obtain sufficient bone, so the entire bone defect/augmentation dimensions are composed of the block autograft. The

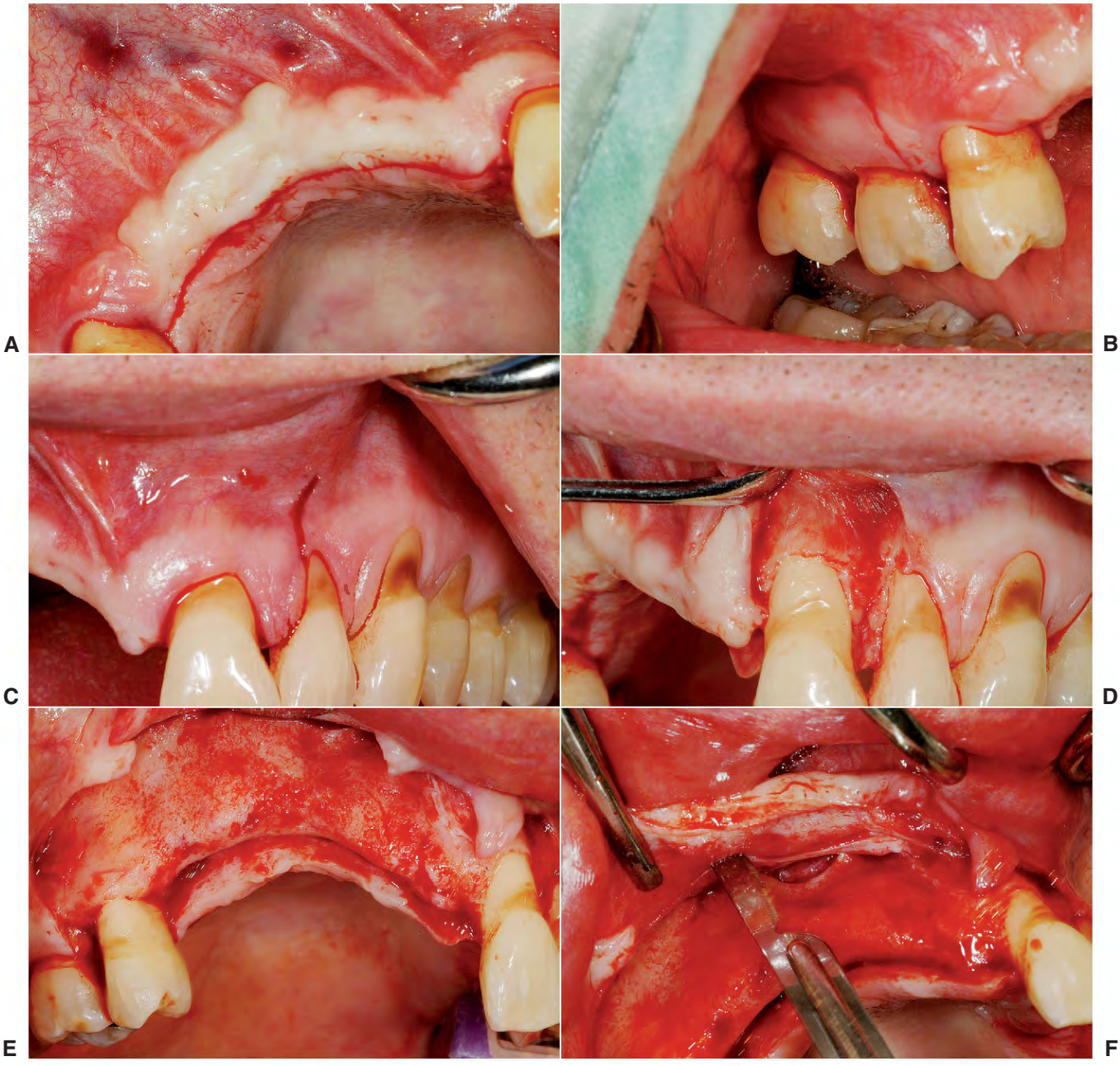


Figure 39-9 **A,** In the recipient site, incisions are designed primarily in attached, keratinized mucosa. The crestal incision is slightly toward the palate. **B,** The vertical release incision stops at the mucogingival junction and is one tooth distal to the edentulous site. **C,** The vertical release incision is distal to the interdental papilla. **D,** A split-thickness flap is reflected distal from the bone graft site. **E,** A full-thickness mucoperiosteal flap is reflected over the graft site and at least 5 mm above the level of the mucogingival junction. **F,** An incision 1 to 2 mm deep is made though the periosteum, 3 to 5 mm above the level of the mucogingival junction (which is on the other side of the flap), for the full length of the tissue flap. *Continued*

WWW.HIGHDENT.IR

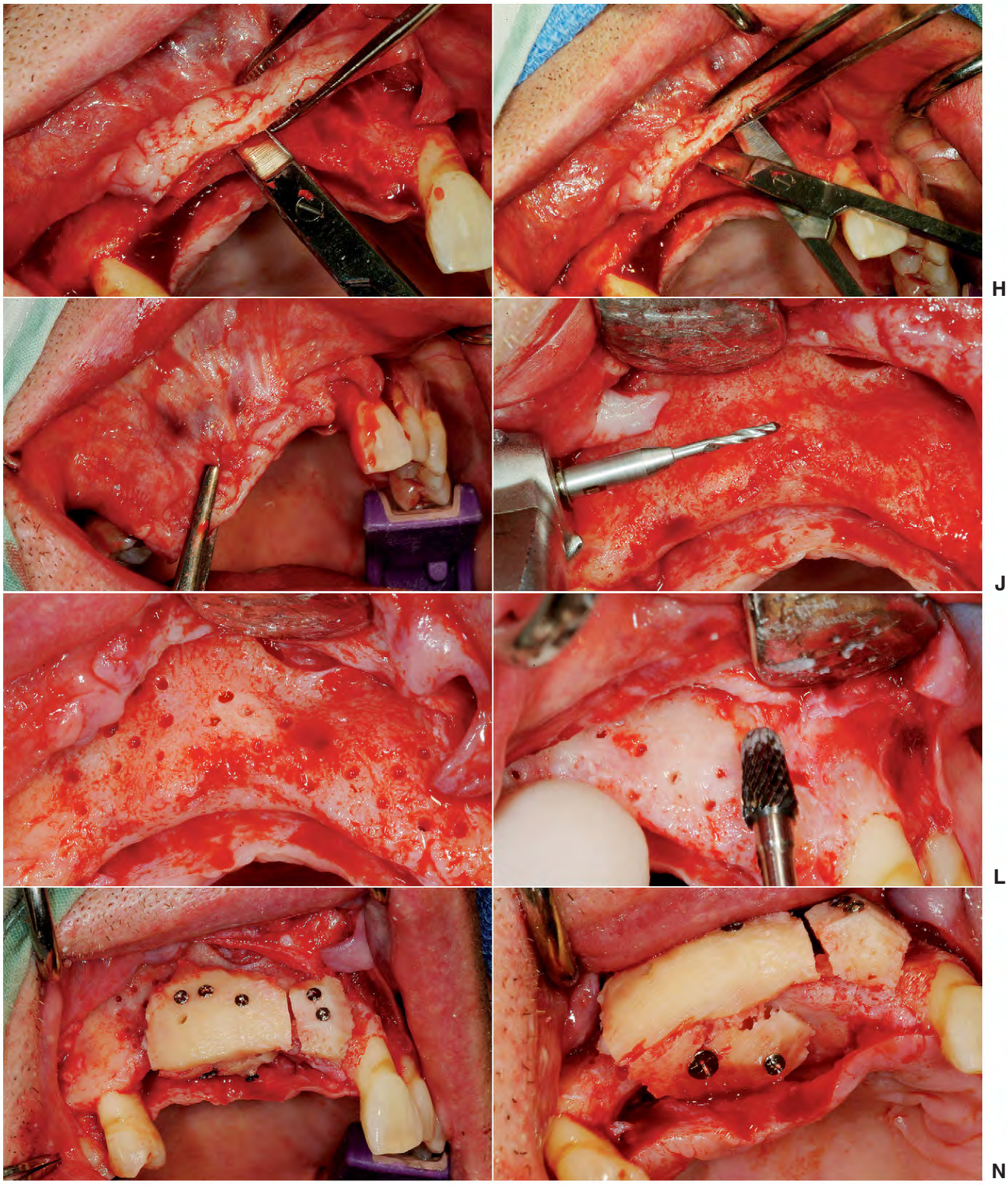


Figure 39-9, cont'd **G,** A tissue scissors is inserted through the incision, 10 to 15 mm deep and parallel to the surface mucosa. **H,** The tissue scissors blades are opened and create a submucosal space above the mucoperiosteum by blunt dissection; this is performed the full length of the flap. **I,** The submucosal space allows the flap to advance 10 to 15 mm for primary closure over the bone graft. **J,** A small-diameter bone screw drill is used to perforate the cortical plate over the bone graft site. **K,** The holes are 3 to 5 mm apart and, when a palatal graft is planned, also perforate the lingual plate. **L,** A large, pear-shaped carbide bur is used over the recipient site to eliminate any soft tissue and prepare the peripheral margins of the graft site. **M,** The block bone grafts are fixated to the graft site. At least two screws are placed in each block for proper immobilization and perforate the lingual cortical plate. **N,** A block graft is also fixated to the lingual aspect of the graft site. The graft is augmenting the site for width and height. *Continued*

WWW.HIGHDENT.IR

همیار دندانسازان

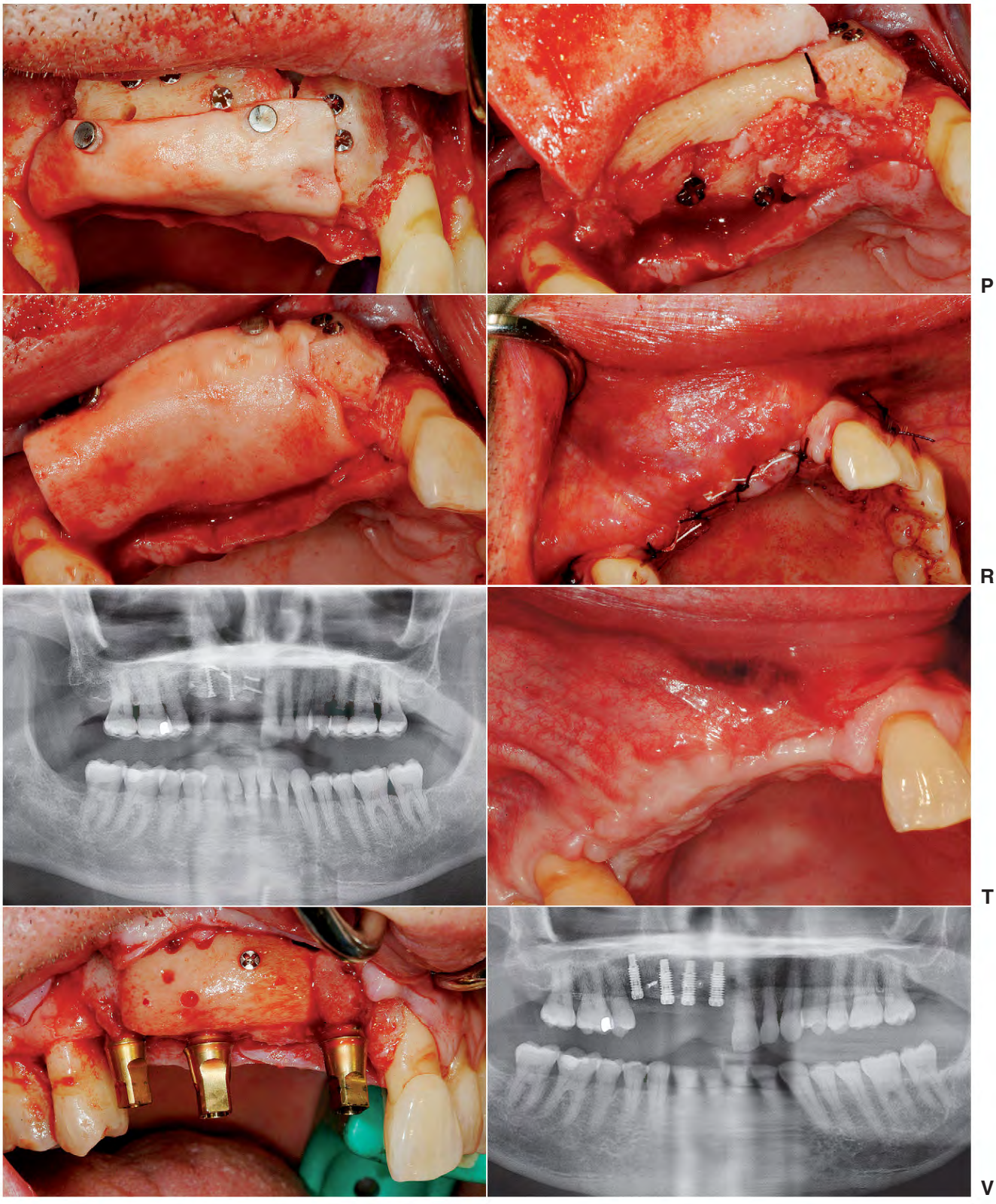


Figure 39-9, cont'd **O,** A barrier membrane (AlloDerm) is tacked to the bone block and draped over the crest. **P,** The barrier membrane is lifted to the facial and particulate bone mixed with platelet-rich plasma is placed around and between the bone blocks. **Q,** The barrier membrane is placed over the particulate graft and tucked inside the palatal flap. **R,** Primary closure, without tension covers the host site. **S,** An immediate postoperative radiograph demonstrates the augmented maxilla and harvested mandible. **T,** The hard and soft tissue are evaluated after a 6-month healing period. **U,** The augmented maxilla is reentered and endosteal implants inserted in key positions (the terminal abutments and the canine site). **V,** An immediate postoperative radiograph of four endosteal implants placed within the block grafts.

WWW.HIGHDENT.IR

mandibular ramus donor site has several advantages over a symphysis donor, including: easier graft harvest, less postoperative discomfort, less neurosensory complications, less incision line opening, less anesthesia, more profound local anesthesia with fewer drugs, and less concern of changes in facial morphology. As a consequence, it is the harvest site of choice, when possible.

Disadvantages of the ramus site include less width of bone in some patients and less length of bone on some patients. As a result, when inadequate bone is present in the ramus site, attention is brought to the mandibular symphysis region.

The mandibular symphysis exhibits a slight curved triangular shape in the midline, and this morphology is often well suited for reestablishing the arch form in maxillary anterior ridges.[100-104] The average interforaminal distance is greater than 4 cm; therefore bone deficiencies requiring larger intraoral grafts may be managed with the symphysis as a donor site. In most cases, sufficient block grafts may be harvested from the symphysis for deficiencies in width involving a span up to four teeth or sites involving up to three teeth that require gains in both vertical height and width.*

A panoramic radiograph is used initially to evaluate the available bone in this donor site. A panoramic radiograph may provide an approximate length of the mandibular teeth, the distance between the foramina, and the vertical height of bone between the root apices and the inferior border of the mandible. Periapical radiographs of the lower anterior teeth may be required for more exact measurements of their length to prevent damage to the roots. CT imagery is used to measure the bone width below the apices of the lower anterior teeth, confirm adequate donor bone volume (especially width), and assess the geometry and angulation of the symphysis.

The basal bone thickness of an anterior mandible ranges from 5 to 15 mm below the roots of the anterior teeth. However, the lingual plate should usually not be violated, so the donor graft thickness is 2 to 3 mm less than the total mandibular width. The width of the mandible in the midline is the greatest dimension. Therefore a lateral cephalogram only demonstrates the greatest bone thickness, not the prospective dimension of the graft. Most often the mandibular symphysis width is several millimeters narrower in the region of the premolar and canines compared with the midline. As a result, when an aggressive or large bone block harvest is desired, CT images of the symphysis should confirm adequate width is present for the host site reconstruction. The CT images are also more accurate than the panoramic radiographs to determine the vertical and horizontal dimension of the graft site.

The average dimension of an anterior mandible between the foramina is 44 mm, with black males having the greatest distance, followed by white males, black females, and white females having the least available donor bone between the foramen.[108] A general rule is to stay at least 5 mm mesial to the mental foramen on each side for a donor bone harvest. Because the average mesiodistal dimension for an implant is 7 mm, a 35-mm bone length between a 45-mm mental foramen length would be necessary for five teeth. In a study by Montazem et al.,[101] dentate cadaver mandibles were harvested from the mandibular symphysis. When two symmetric blocks were measured from each site, the average was 21 × 10 × 7 mm, the largest was 25 × 13 × 9 mm, and the smallest measured 21 × 6.5 × 6 mm.[101]

The vertical height of bone in the symphysis for a donor harvest is variable and ranges from 5 to 20 mm from one person to another. A general rule is to remain 5 mm from the root apices and 5 mm from the inferior border of the mandible. The inferior landmark dimension may be violated and the inferior border of the mandible may even be incorporated into the block graft when necessary.

Surgical Harvest

Anesthesia

The anterior symphyseal region of the mandible is innervated by the mandibular branch of the fifth cranial nerve (V3) and cervical nerves from C-3 and C-4. As a result, several injections are required. Bilateral dental or Akinosi blocks with lidocaine 2% (1:100,000 epinephrine) and marcaine 0.5% (1:200,000 epinephrine) accomplish anesthesia for the V3 innervation. Infiltration anesthesia with lidocaine 2% (1:100,000 epinephrine) is then performed with one-third carpule in front and below each mental foramen and one-third carpules in the midline at the base of the mental protuberance. Another carpule of anesthetic is divided in half with an injection on each side of the superior general tubercle near the base of the mandible.

Surgical access to the symphysis is obtained via a crestal incision, vestibular incision, mid-keratinized tissue incision, or sulcular approach, depending on the anatomy. Most often mandibular anterior teeth are present above the donor site.[109] The vestibular approach is used when there is 4 to 8 mm of keratinized tissue height around the lower anterior teeth, when subgingival crown margins are present in the area, when dehiscent roots are suspected, when root coverage is part of the history, or when sulcus depths around the mandibular anterior teeth are greater than 4 mm deep. In these situations, reflection of the soft tissue around the anterior teeth may result in tissue recession and root exposure soft tissue healing.

The vestibular incision is made in the mucosa at least 1 cm beyond the mucogingival junction and extends to each distal region of the canines. An inferior vertical

*References 21, 23, 26, 38, 101, 103, 105-107.

WWW.HIGHDENT.IR

همیار دندانسازان

release is made for approximately 10 mm between the canine and first premolar. Therefore this incision is anterior and above the mental foramen. A full-thickness mucoperiosteal flap is reflected toward the base of the mandible to the level of the pogonion, leaving the most inferior aspect of the periosteal attachment of the mentalis muscle intact (Figure 39-10).

The vestibular incision has the most intraoperative bleeding and the highest risk of incision line opening, but the least risk of soft tissue changes around the teeth and root exposure after healing. It also creates easier access to the chin. It is the easiest incision line to suture.

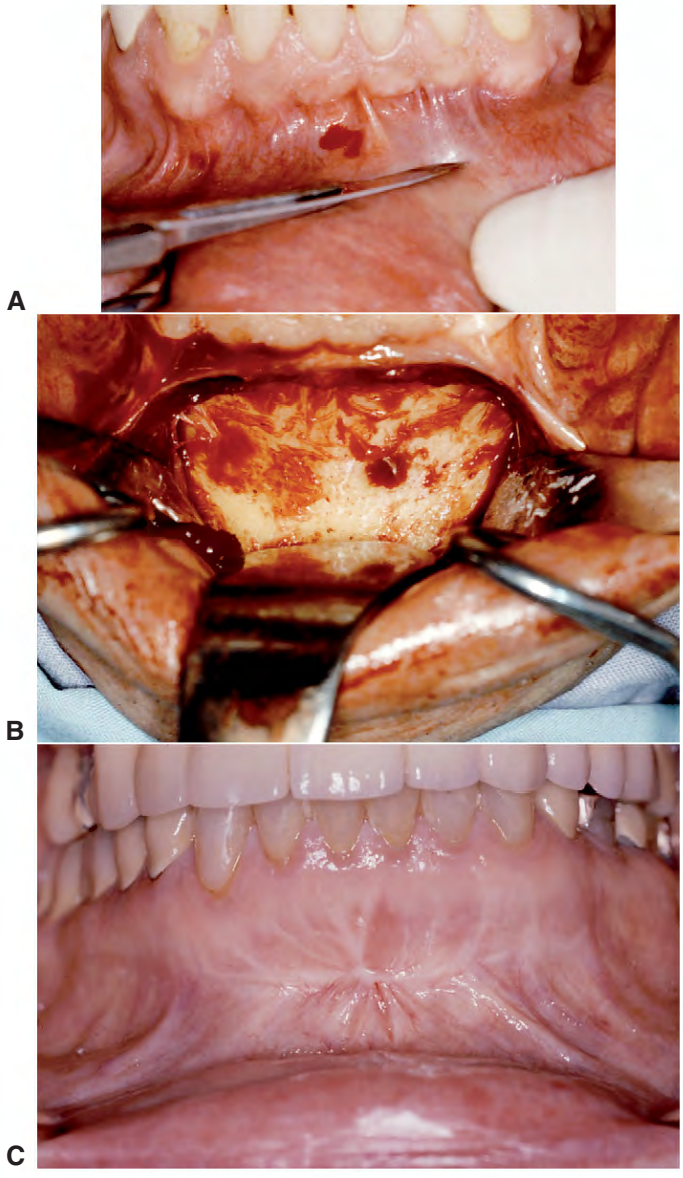


Figure 39-10 **A,** A vestibular incision is made 5 to 10 mm below the mucogingival junction when 4 to 9 mm of keratinized tissue height is observed on the facial of the mandibular anterior teeth. **B,** The incision extends to the distal of the canines and a vertical release incision is made for approximately 10 mm (above the height of the mental foramen). A full-thickness mucoperiosteal reflection exposes the mandibular symphysis. **C,** Vestibular incisions often heal with scar formation.

Limiting the distal extent of the vestibular incision to the canine tooth area will reduce the incidence of mental nerve paresthesia.[38,39,44]

An incision is made within the mandibular anterior keratinized tissue when it is 9 mm or greater in height. This condition is rarely observed. However, when present, this is the incision line of choice, because it reduces intraoperative bleeding, eliminates the risk of root exposure after healing, and soft tissue healing conditions are ideal. The incision is made from the distal of each canine and a vertical release is made 10 mm in front of and above the mental foramen (Figure 39-11).

A sulcular incision is made when there is less than 4 mm of keratinized mucosa in height around the lower anterior teeth. These conditions are associated with a high mucogingival junction and high muscle attachments. Under these conditions, incision line opening is a greater risk. This is especially observed when the mentalis muscle is large and parafunctional forces of this region exist. The sulcular incision carries the least risk of incision line opening after healing, but has an increased risk of root exposure after soft tissue healing. It is also the most time consuming for suturing (Figure 39-12).

Symphysis Donor Harvest (for Division B–w to C–w)

After the symphysis is exposed, the osteotomy for graft harvest is planned. The dimensions of the block are determined by the size of the bone defect. The osteotomies may be performed with a surgical fissure bur (557, 702) or oscillating saw. This is usually done under copious sterile irrigation with a 1:1 to 1:5 contraangle or straight hand piece. The most superior bone cut is made first, because the position of this border is less flexible. The superior osteotomy is ideally made

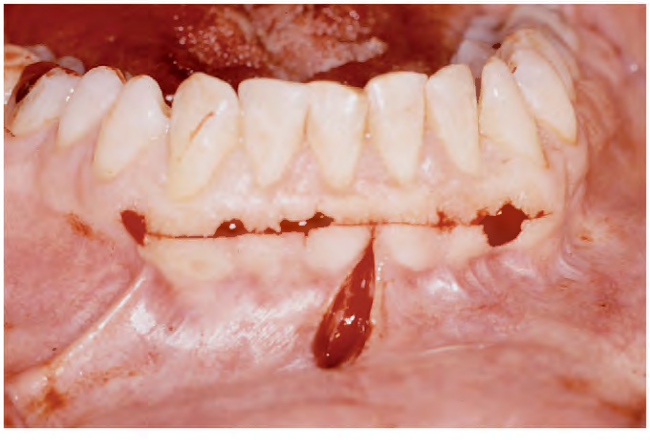


Figure 39-11 When the attached keratinized tissue on the facial aspect of the lower anterior teeth is greater than 9 mm, an incision in the keratinized tissue is of benefit. The incision is made to the distal of the canines. In this case, a midline vertical incision was made, because the symphysis harvest was limited in size.

WWW.HIGHDENT.IR

Figure 39-12 **A,** Sulcular incisions are usually made in the anterior mandible for a symphysis harvest when the keratinized tissue is less than 4 mm in height or when a heavier musculature is observed. **B,** The incision extends distal to the canines, and a vertical release incision is made anterior and above the height of the mental foramen. A full-thickness mucoperiosteal flap reflection exposes the symphysis for the harvest. **C,** A sulcular incision has less risk of postoperative incision line opening; however, there is a greater risk of root exposure after healing.

at least 5 mm below the root apices. The angulation of this cut is perpendicular to the cortex to prevent injury to the roots. The depth of the osteotomies should be at least through the outer cortex and to the opposite cortical plate. Perforation of the lingual cortex may inadvertently occur, but it is not encouraged. The osteotomy may slope below the longer canine roots when more mesiodistal distance is necessary for the host site (Figures 39-13 and 39-14).

In most cases, the inferior cortex of the mandible is maintained unless a block graft for vertical augmentation is harvested. When adequate size permits, the inferior cut is 5 mm or more above the inferior border. The inferior margin has a cut perpendicular or angled upward, so the block harvest may be lifted out of the jaw with an osteotome. This also may protect the inferior portion of the mandible from fracture during the harvest, because more cortical bone along the inferior border is maintained (Figure 39-15, *A*). The vertical cuts are made in relation to the topography needs of the host site and may extend 5 mm in front of the foramen, when necessary. A larger mesiodistal dimension harvest rarely fits the host site in one piece. Instead, two blocks of bone are usually easier to mortice into place. In addition, two blocks of bone are easier to harvest than is one long block. Therefore most often the blocks are divided into 15- to 17-mm-long pieces and three vertical cuts are often made to trim the block harvest (see Figure 39-15, *A* to *C*).

A straight bone chisel is tapped along the osteotomy to ensure the cuts have been made to the lingual cortical plate. The bone chisel is then introduced at an angle and attempts to slide along the lingual plate of bone as the mallet firmly taps the bone segment. The chisel is not positioned to leverage the bone block from the site along the inferior border, because the chin may fracture down rather than the bone block fracturing off. During this tapping and leverage process, the mandible should be closed and braced against the maxillary teeth, so the mallet force is not generated toward the condyles and temporomandibular joint (see Figure 39-15, *D*, *E*).

Often more than one piece of bone is harvested from the symphysis, which facilitates adaptation of the graft to the recipient site geometry. Two bone blocks are often easier to harvest, because the second larger segment may be removed with improved access to the lingual plate to harvest the second piece after the first segment has been removed. This also often allows the harvest of a thicker block of bone.

Additional cancellous bone may be procured from around the block of bone with a small gouge, rongeur, or chisel after the blocks are removed. This particulate bone can be used for augmentation or to fill discrepancies between the block graft and host bone. However, the volume of obtainable cancellous bone in the symphyseal area is usually meager.[105] Bone cores ranging from 4 to 10 mm in diameter may be harvested with a trephine bur for use in alveolar augmentation.[110] The trephine bur prepares the bone core to the inner aspect of the lingual cortical plate. A Molt elevator is placed into the circular osteotomy and is used to fracture the bone core off its lingual base and elevate it from the bone. The cores of bone may be transferred in toto to the recipient site or shaped to fit a defect. It is critical

WWW.HIGHDENT.IR

همیار دندانسازان

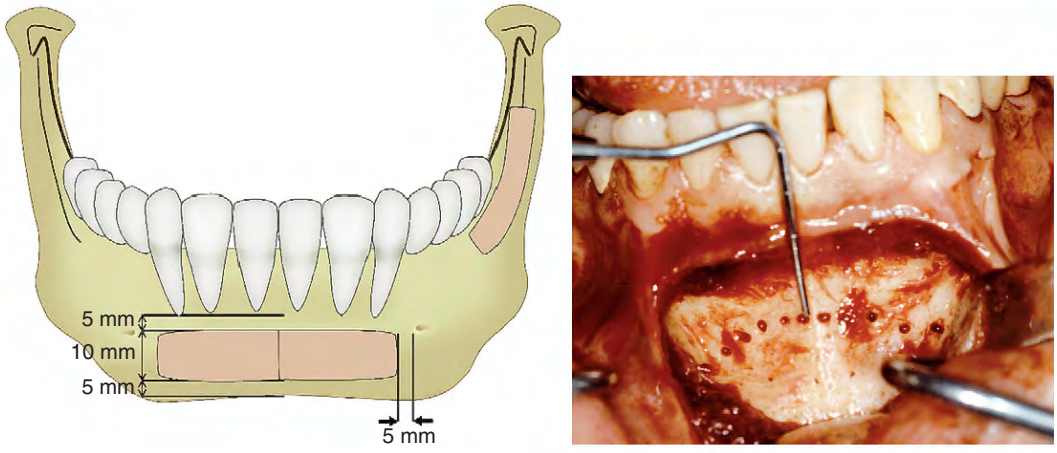


Figure 39-13 **A,** The guidelines for the symphysis block harvest is usually 5 mm from each mental foramen, 5 mm below the roots of the anterior teeth, and 5 mm from the inferior border of the mandible. **B,** The superior osteotomy for the bone block is made 5 mm from the apex of the mandibular anterior teeth. The block margin slopes down in the canine region because of the longer roots.

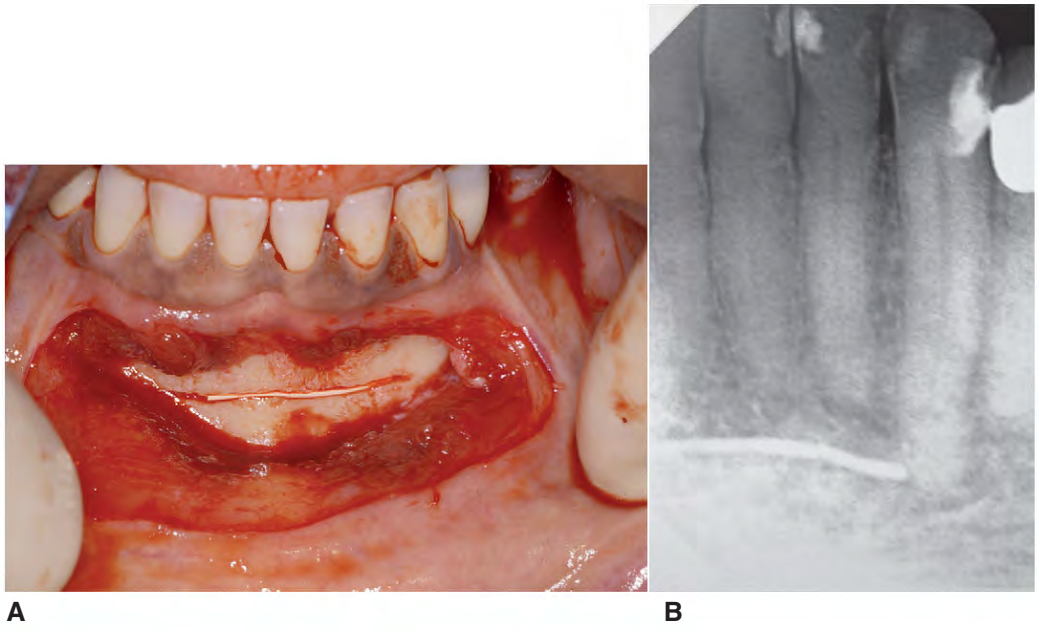


Figure 39-14 **A,** When in doubt, a piece of gutta percha may be placed into the initial osteotomy, and a radiograph of the site is made. **B,** The radiograph reveals the initial osteotomy is too close to the canine root and should be 6 mm more apical in this region.

that the bone cores or graft particles be fixated and immobilized during the healing phase when larger than 3×5 mm. This may be accomplished by mortising the bone into the defect, securing it with a membrane, or placing it into a protected space.

A branch of an artery from the submandibular complex enters the lingual cortex of this symphysis in the midline region, just above the superior genial tubercle. On occasion, this vessel bleeds with profusion. A blunt bone tap (or mirror handle with mirror removed) placed over the bleeder and a firm tap with a mallet crushes the bone over the foramen and arrests the bleeding. On occasion, the incisive branch of the mandibular nerve, artery, and vein are seen entering the bone block or is associated with slight hemorrhage. When possible, this nerve complex should stay intact. If bleeding is present, pressure with a hemostat or surgical sponge most often solves the problem.

WWW.HIGHDENT.IR

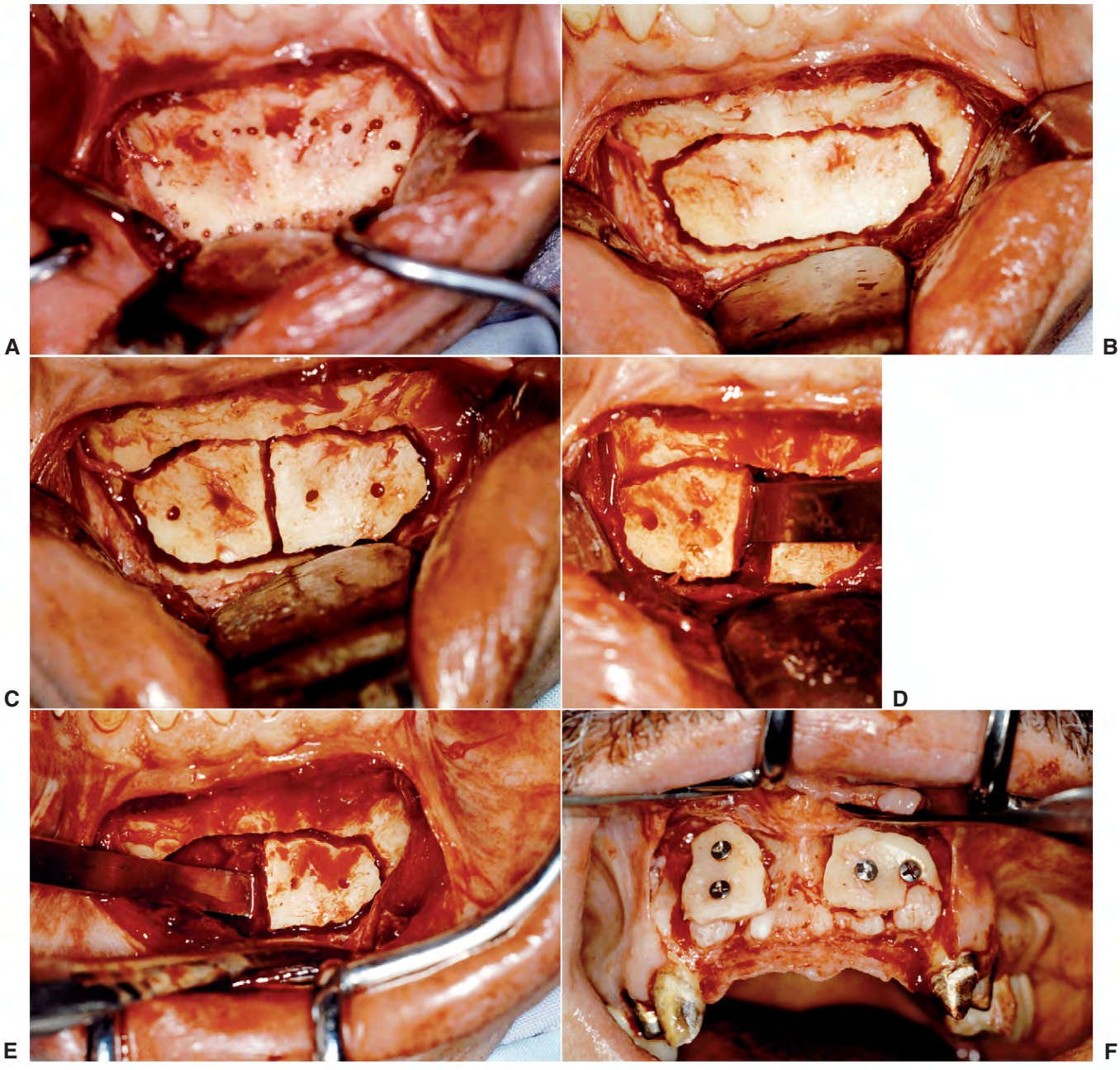


Figure 39-15 **A,** The inferior osteotomy is planned and is usually 5 mm above the inferior border of the mandible when a harvest is primarily for an increased width of bone. **B,** A fissure bur may be used to connect the dots of the planned bone block. **C,** The block is usually designed to be harvested in two pieces. **D,** After an osteotome is used to ensure the osteotomy is made up to the lingual plate, the chisel is angled to shear the one block from this landmark. **E,** The second bone block is easier to harvest, because the bone chisel can slide along the lingual plate with direct access. **F,** The bone blocks are positioned in key implant regions, with at least two fixation screws. *Continued*

The block bone grafts are then fixated to the host site in key implant positions (see Figure 39-15, *F* to *I*). The block graft may be reentered after 4 to 6 months for implant insertion (see Figure 39-15, *J* to *M*). After an additional 4-month period, the implants may be restored (see Figure 39-15, *N*).

Hemostatic materials (collagen, gelatin, sponge, oxidized regenerated cellulose) can be placed into the symphysis area after the bone harvest. Bone wax should only be used for heavier osseous bleeding because bone replacement may be inhibited.[111] Larger donor defects should be grafted with allogenic bone or resorbable hydroxyapatite (HA).

The donor site is closed after the block graft is fixated to the recipient site and the soft tissues are sutured. Before suturing the donor site, the soft tissue superior to the initial access incision (vestibular or within the keratinized tissue) is elevated a few millimeters to reduce

WWW.HIGHDENT.IR

همیار دندانسازان

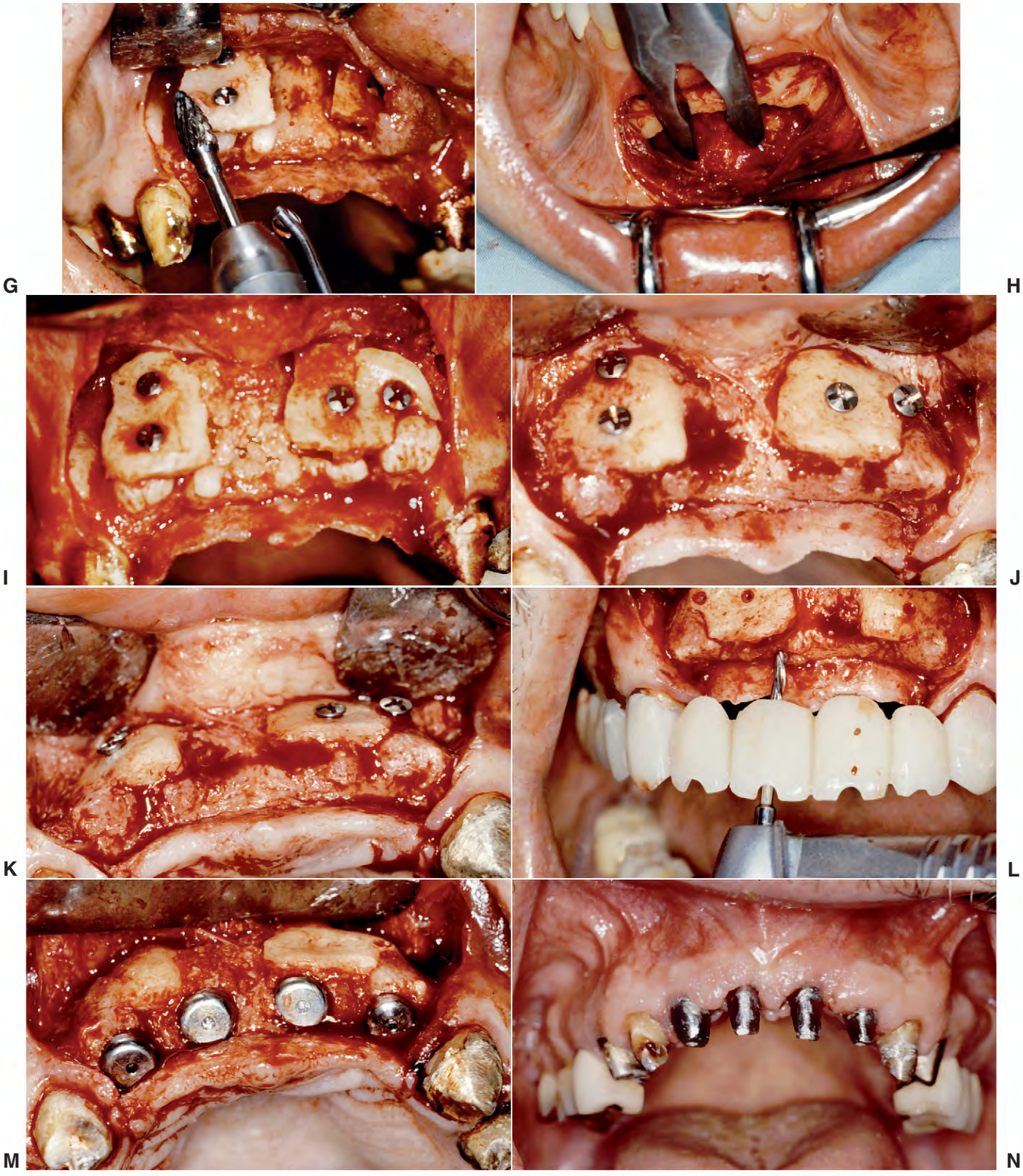


Figure 39-15, cont'd **G,** The bone blocks are recontoured in situ to smooth the edges, which might perforate the soft tissue flaps. **H,** Additional particulate bone may be harvested from the symphysis with a rongeur or trephine bur. **I,** The particulate bone is placed between the blocks and in any voids between the host bone and blocks. **J,** A reentry into the host site after 5 months. **K,** The block grafts usually exhibit less resorption compared with the particulate graft (in the center). **L,** A drill guide is used to position the implants into the graft site. **M,** Four implants are positioned in the grafted site. **N,** After 4 months of healing, the abutments are inserted and the prosthesis may be fabricated.

WWW.HIGHDENT.IR

tension on the flap from edema and lip movement. This also allows the suture needle to enter the superior flap without tearing the periosteum. A two-layered closure is recommended for suturing. The submucosal suture is a vertical or horizontal mattress with resorbable PMA or Vicryl suture on each side of the midline.

Postoperative pressure dressings can reduce the development of hematoma formation, incision line dehiscence, and infection. The patient wears a chin pressure bandage for at least 24 hours, and a large harvest should have 3 days of pressure. The patient is warned against pulling the lip to look at the site until the sutures are removed 2 weeks later. One of the most common complications is incision line opening of the donor site, which may become very painful and even infected. The patient should not suck on a straw or move the lower lip as part of any parafunctional activity.

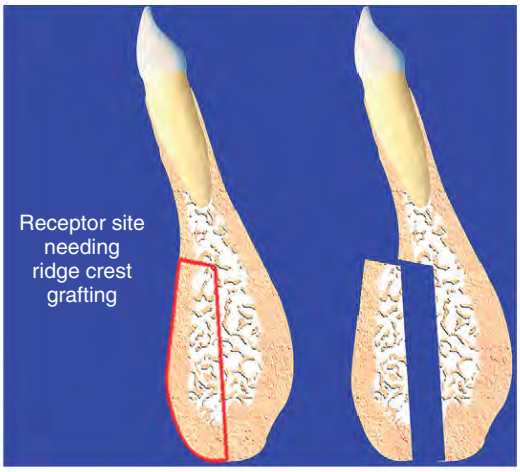


Figure 39-16 A mandibular symphysis block harvest to augment a C–h bone volume in height may include the inferior border of the anterior mandible.

C–h Symphysis Donor Harvest (for Division C–h Host Sites)

The anterior mandible may also be used as a donor site to an augment height of bone in an edentulous site, when the defect is three adjacent teeth or less. To obtain adequate donor bone volume, the inferior border of the mandible is included as part of the graft (Figures 39-16 and 39-17). A lateral cephalogram may be used to evaluate the bone height and width in the anterior mandible (Figure 39-18).

The initial incision and reflection of the anterior mandible is similar for the symphysis bone harvest for width. However, when the inferior border is included, the tissues must reflect beyond the facial line angle of the inferior border and include the inferior border and the lingual line angle of the mandible. When possible, 5 mm of the midline tissue may be maintained to help decrease the risk of ptosis after healing (Figure 39-19, *A* to *D*).

The most superior bone cut is made 5 mm from the apex of the teeth. The vertical bone cuts are made from this point through the inferior border of the mandible. The bone blocks are usually harvested in two pieces, with a midline section of 4 to 6 mm maintained. This segment decreases the risk of altering the postoperative appearance of the chin, especially when prominent. Therefore four vertical cuts are made through the inferior border of the mandible, two near the midline and two distal in relation to the recipient site requirement (see Figure 39-19, *E* to *I*).

The inferior bone cut is often the most difficult to perform. Rotary saws offer an advantage for access. However, most rotary bone saws cut less than 5 mm deep, because the blades are less than 10 mm in diameter. Because the cortex of the inferior border of the mandible is offer greater than 6 mm, attempts to fracture the bone block from the inferior border as the block is harvested are often challenging. The inferior bone cut should allow 3 mm or more of bone to remain on the lingual aspect so that the lingual plate does not fracture off during the harvest.

A small-width chisel is most often used in the inferior bone cut parallel to the lingual plate and tapped firmly with a mallet. The smaller size increases the stress (stress = force ÷ area) and more easily penetrates the cortical bone remaining on the inferior border. The bone chisel may become locked and difficult to dislodge after it is tapped into the site. Several chisels may be required, so if one is difficult to remove from the bone, another chisel adjacent is tapped and often frees the first one from the site. After the inferior border bone block is harvested, the defect is filled with microporous HA and a collagen membrane, to help restore the contour of the mandible (see Figure 39-19, *J* to *R*).

Pressure dressing should be worn for 3 days to reduce postoperative bleeding and ptosis of the chin. Sutures should remain in place for at least 2 weeks. Cortical steroid dosages may be increased (Decadron 12 mg/day 1, 6 mg/day 2, 3 mg/day 3). Antibiotics, pain medication, and ice protocol to the face for 3 days are also warranted.

Mandibular Ramus Donor Site

The limits of the ramus area are more often dictated by clinical access and variable anatomy. The overall anatomic limitations include the coronoid process, molar teeth, inferior alveolar canal, and width of the posterior mandible (Figure 39-20). There are three dominant landmarks that are variable, should be evaluated on a case-by-case basis, and require clinical radiographic and CT analysis (A-P width of the ramus, distance from external oblique of the ramus, and body to inferior alveolar nerve complex [IAN] and width of the posterior mandible and ramus). Harvesting bone

WWW.HIGHDENT.IR

همیار دندانسازان

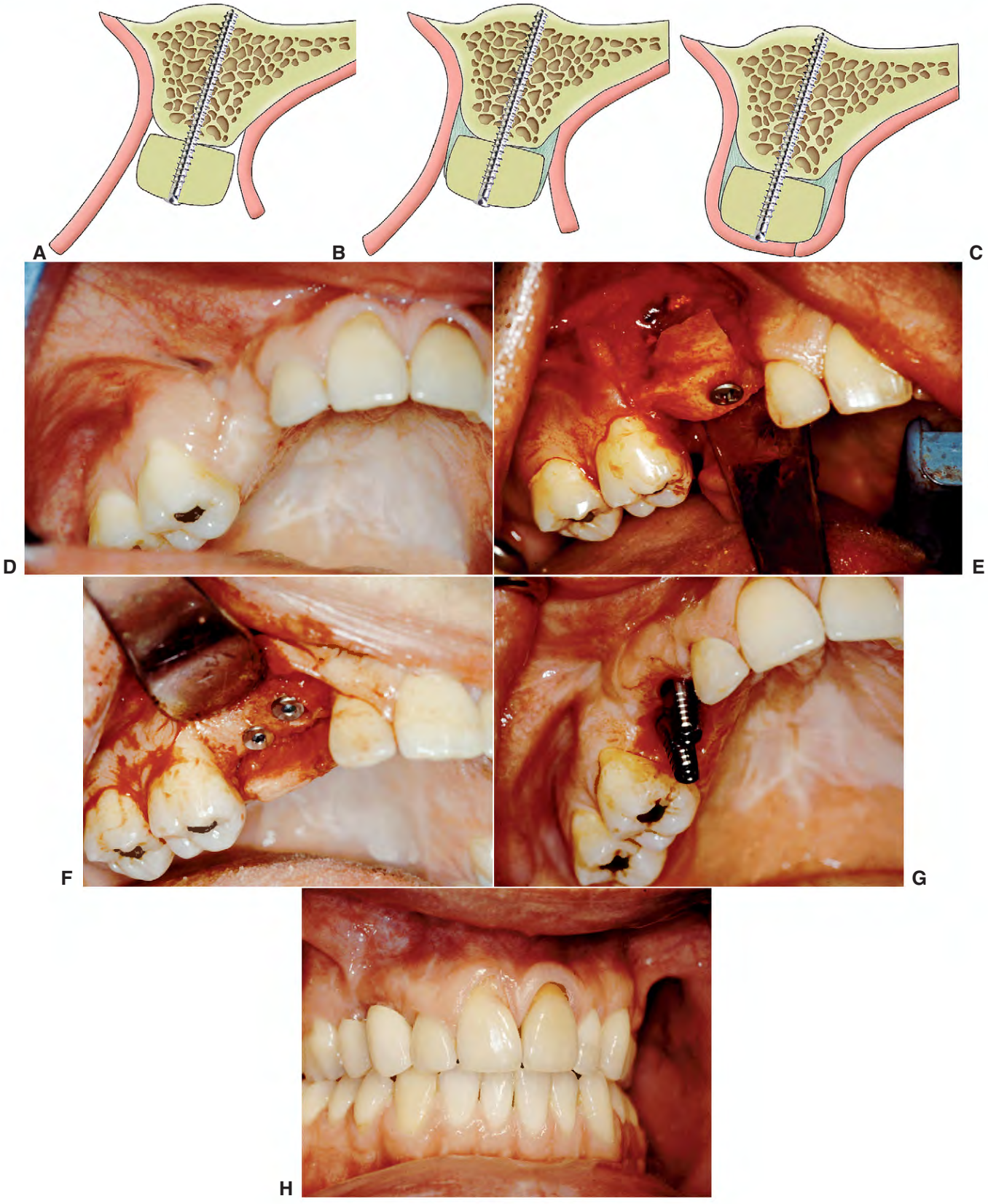


Figure 39-17 **A,** A C–h ridge may use a block graft on the crest, which is fixated by a screw that engages the opposing cortical plate. **B,** A particulate autograft or alloplast fills in any voids. **C,** The soft tissue must be approximated with primary closure. **D,** A vertical defect in the maxillary canine and premolar region. **E,** A block graft is placed to gain height in the defect. **F,** At reentry (6 months later), two endosteal implants are inserted. **G,** After 4 months, healing abutments are inserted into the implants. **H,** Two splinted crowns restore the two implants in the canine and first premolar sites.

WWW.HIGHDENT.IR

Figure 39-18 A lateral cephalogram may be used to evaluate the mandibular symphysis for adequate height and width.

from this region requires knowledge of the mandibular canal anatomy to prevent nerve injury (Figure 39-21).

The clinical evaluation deals with the presence of molars and the width and length of the external oblique in the body of the mandible. When all three molars are missing, the surgical access is easier and the width of the bone block harvest is often greater. The A-P length of the external oblique and prosthetic "buccal shelf" ranges from no presence from the third to first molar side, to a dominant projection lateral to the body of the mandible. An index finger may be placed on the external oblique of the ascending ramus and slid down the lateral aspect of the mandible. Often a ledge is felt lateral to the second molar region and begins to disappear at the medial of the first molar. The wider the "ledge" lateral to the molars or body of an edentulous mandible, the wider the ramus block bone harvest. Some mandibles have almost no "buccal shelf," whereas others have a 7-mm ledge. Most often the buccal shelf disappears at the mid-first molar region to the anterior and to the third molar region on the posterior aspect. The ramus length is variable, with the most common vertical limit below the coronoid process, as this structure is so thin that a block section would remove the entire segment.

Although the buccolingual position of the mandibular canal is variable, the distance from the canal to the medial aspect of the buccal cortical plate (medullary bone thickness) was found to be greatest at the distal half of the first molar.[112] Therefore when larger grafts are planned, the anterior vertical bone cut may be made in this area. The vertical bone cuts are progressively deepened until bleeding from the underlying cancellous bone is visible.[113,114]

The second variable is the distance from the external oblique and ramus to the IAN. The mean A-P width of the ramus is 30.5 mm, with the mandibular foramen located about two thirds of the distance from the anterior border.[113] A panoramic radiograph may be used to initially asses this dimension. The lingula on the medial of the ramus is the entry point of the IAN. It may be at the occlusal plane (most often), above the occlusal plane, or below the occlusal plane. The lingula may be in the anterior third of the ramus, the middle third, or the distal third of the width of the ramus. As a general rule, the higher and farther forward the lingula, the closer the IAN runs adjacent to the external oblique. As a result, the ramus block harvest must be lateral to the IAN and is less than 3 mm thick. The lower and more distal the lingual is in the ramus on the panoramic film, the lower the IAN is to the external oblique. As a result, the ramus block may be 6 mm in width.

The distance the IAN is from the external oblique in the body of the ramus (buccal shelf regions) is the anterior part of the second variable. When the dimension is greater than 10 mm in height, the surgical access is easier, there is less risk of paresthesia, and the block can usually be wider because the block harvest may include bone above the IAN. When the distance from the external oblique to the height of the IAN is less than 5 mm, the entire harvest site must be lateral to the IAN. Therefore the graft width is often limited to 3 mm.

The third variable of the ramus region is the width of the posterior ramus. In general, females have a thinner body and width of ramus compared with males. The best method to evaluate ramus width before surgery is a reformatted CT image.

As a result of these variables, a rectangular piece of cortical bone from 3 to 6 mm in thickness may be harvested from the ramus.[114] The length of the rectangular graft may range from 1 to 3.5 cm, and the height usually is not much greater than 1 cm.[20,44,115] These dimensions accommodate width deficiencies involving a span of three to four tooth sites. Although use of the coronoid process as an autologous graft has been reported,[116-118] the amount of bone for ridge augmentation is negligible considering the potential postoperative disability of a coronoidectomy.[119,120]

This morphology conforms especially well as a veneer graft to gain additional ridge width. The anatomic proximity makes the ramus well suited for augmentation to the posterior mandible inadequate in width.[42]

Surgery

The surgical procedure to harvest a block bone graft from the ramus presents many features similar to a sagittal split ramus osteotomy.[114,121-127] An incision begins on the midcrest of the ridge in the posterior

WWW.HIGHDENT.IR

همیار دندانسازان

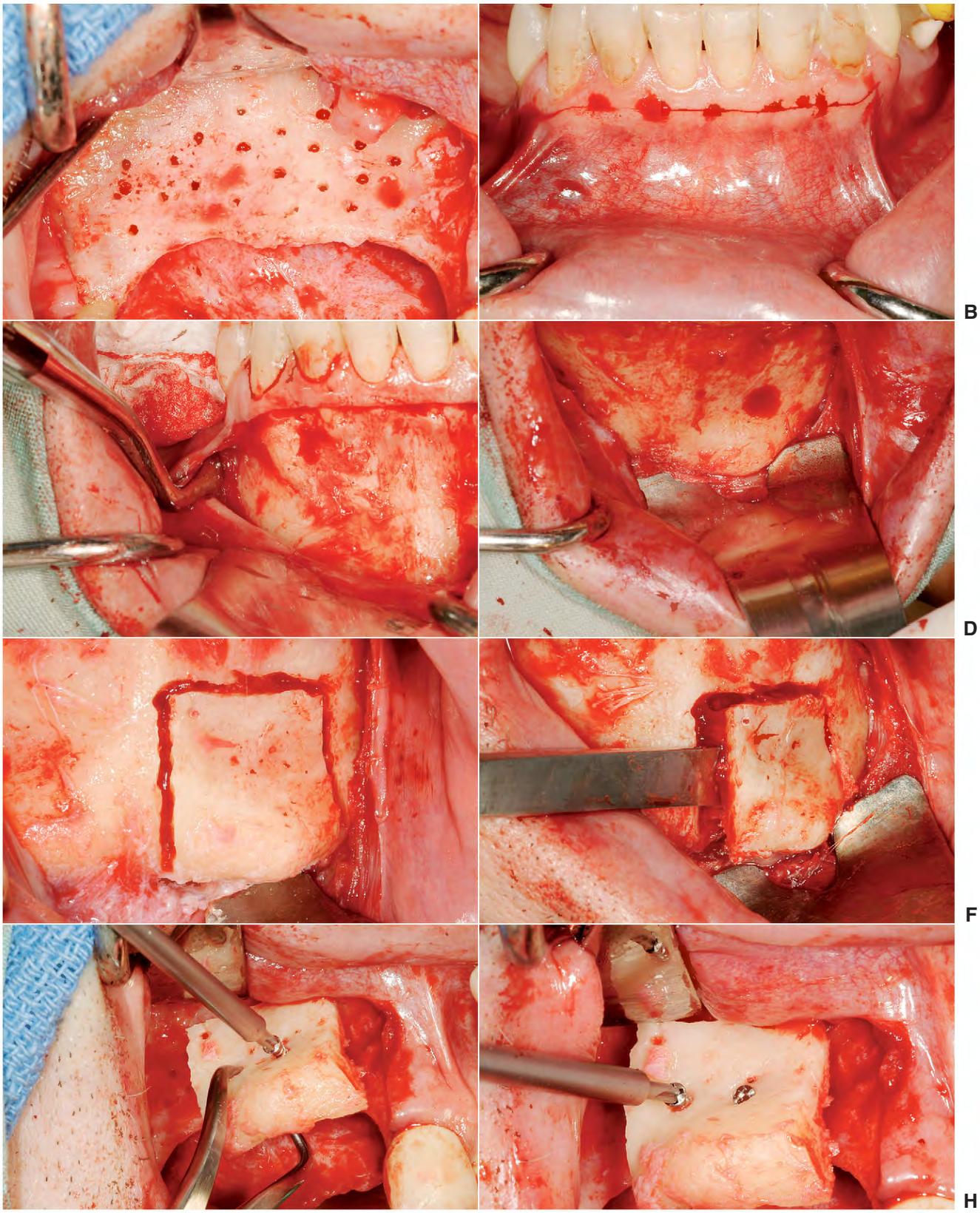


Figure 39-19 **A,** The host site is prepared for the block bone graft. **B,** The donor site incision is made from the distal of each canine and within the keratinized mucosa. **C,** A full-thickness mucoperiosteal flap reflects the anterior mandible, and the mental foramens are identified. **D,** The inferior border of the mandible is reflected. **E,** The first bone block is prepared. Some tissue is maintained on the inferior border at the midline. **F,** The first bone block is harvested with an osteotome. **G,** The first bone block is positioned at the host site to gain height and width, with the benefit of the inferior border of the mandible. **H,** Two fixation screws secure the block graft.

Continued

WWW.HIGHDENT.IR

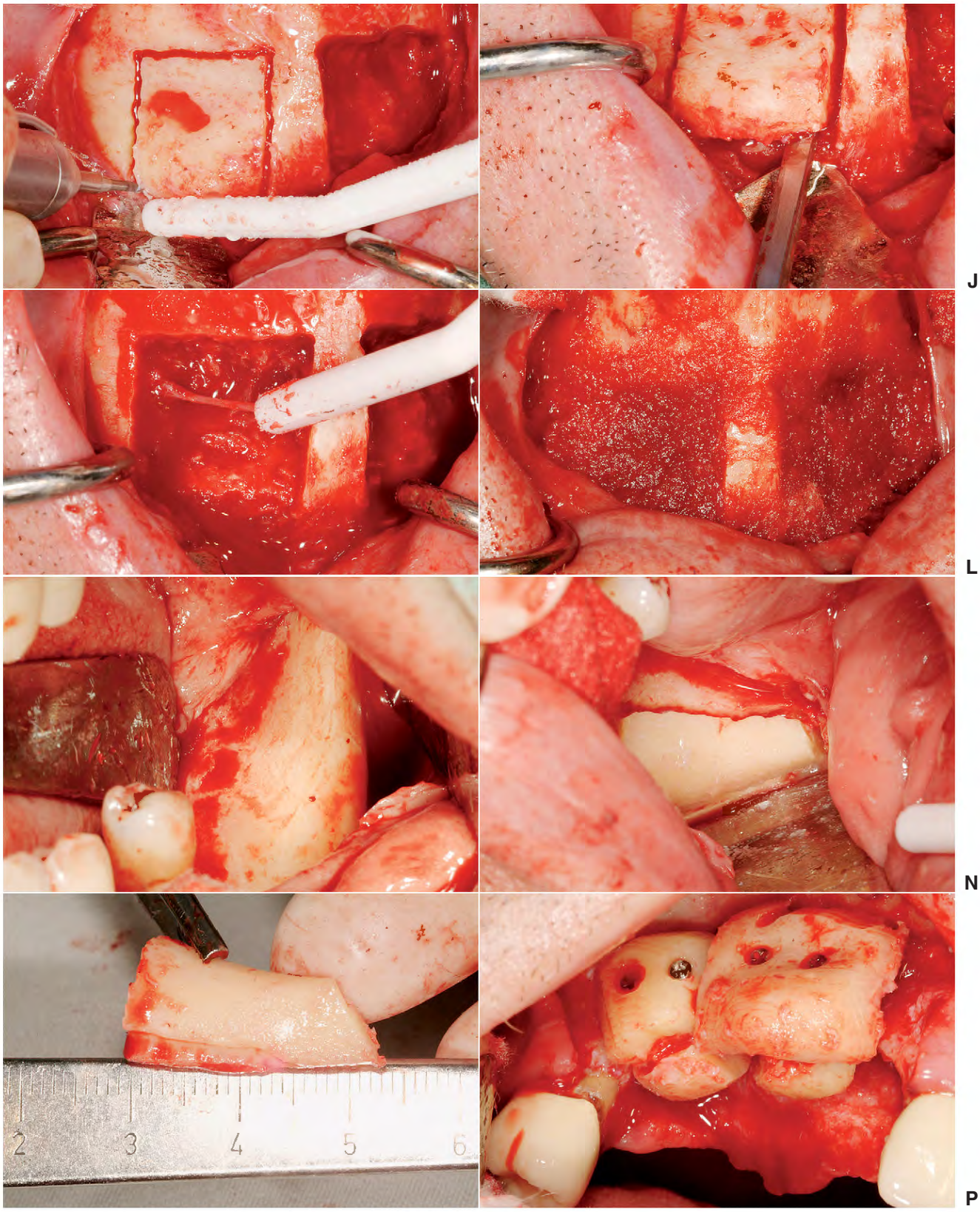


Figure 39-19, cont'd **I,** The second block is prepared. Bone is maintained in the midline to prevent fracture of the mandible or changes in chin morphology. **J,** A small chisel is used to separate the block from the cortical bone of the inferior border of the mandible. **K,** A branch of the mental nerve is identified and maintained when possible. **L,** The anterior mandibular donor sites are filled with demineralized freeze-dried bone (DFDB). **M,** The patient's left ramus is prepared for a block graft harvest. **N,** The block graft is prepared. **O,** The ramus block graft is evaluated. It measures 20 mm in length, 8 to 10 mm in height, and 4 to 6 mm in width on the crest. **P,** The ramus block graft is segmented and fixated at the host site.

Continued

WWW.HIGHDENT.IR

همیار دندانسازان

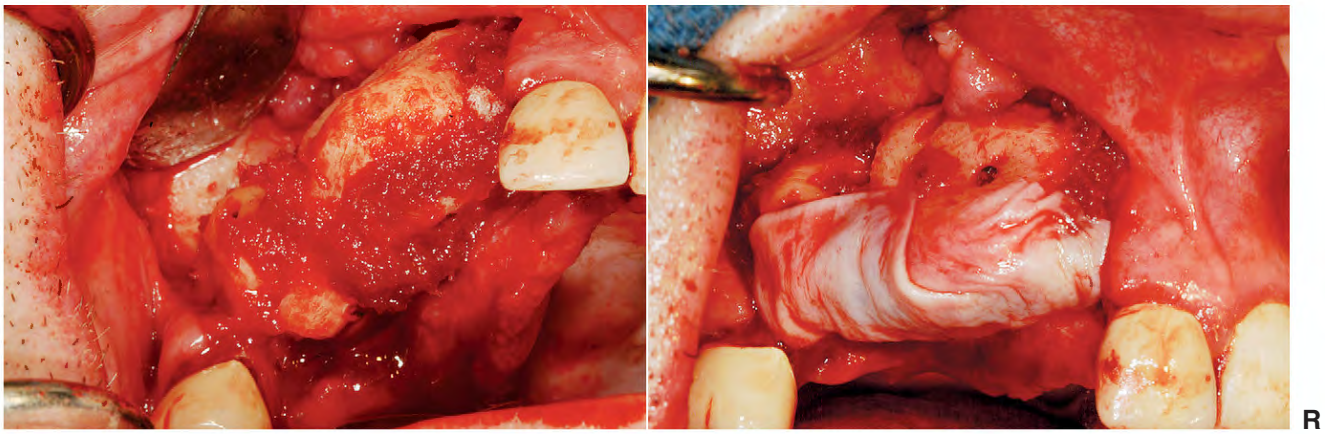


Figure 39-19, cont'd **Q,** Platelet-rich plasma and DFDB cortical fibers are placed between the blocks. **R,** A barrier membrane is placed over the graft site.

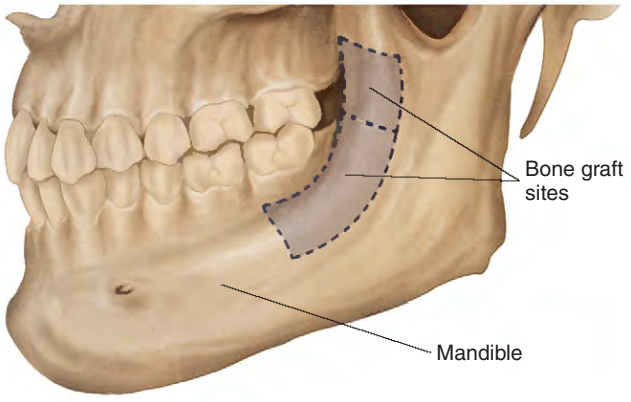


Figure 39-20 The ramus and posterior body may be used as a bone block harvest site.

Figure 39-21 A panoramic radiograph may be used to evaluate the anteroposterior dimensions of the ramus, the position of the inferior alveolar nerve complex, and the external oblique ridge.

edentulous patient, beginning at the retromolar papilla, or base of the retromolar pad. In the dentate patient, a buccal sulcular incision from the retromolar papilla to the second premolar is first made. The retromolar pad is then identified. An incision up the ramus is made, lateral to the retromolar pad to the height of the occlusal plane. The incision is on the middle of the bone of the ascending ramus lateral to the pad. The pad is often to the lingual of the ascending ramus and is not a bony landmark. On rare occasions (but possible), the lingual nerve is present in the retromolar pad, and incising in this region leads to paresthesia or dysphasia of the lingual nerve, the chordi tympani, and a sympathetic branch of the parasympathetic nerve to the submandibular gland (Figure 39-22).

At the height of the occlusal plane, the buccal artery crosses the ramus. The maxillary artery extends anteriorly and laterally to the retromolar pad. If severed, profuse bleeding is observed. Hemostats should be readily available to clamp this vessel on the lingual aspect of the incision. Starting the incision on the ascending ramus no higher than the level of the occlusal plane minimizes the possibility of severing the buccal artery or exposing the buccal fat pad.[114] The incision continues anteriorly into the buccal sulcus of the molar teeth or posterior ridge area.

A mucoperiosteal flap is reflected from the mandibular body, exposing the lateral aspect of the ramus. The attachment of the buccinator muscle is usually observed first. A periosteal elevator is placed medial to this structure and directly on the bone of the external oblique and slid along the ramus. The flap is elevated superiorly along the external oblique ridge. After the facial flap is reflected, the incision in the mid ramus may be extended to the attachment of the temporalis muscle, with one tendon on each side of the ramus. The periosteal elevator slides along the ramus 15 mm deep, down the ramus toward the first premolar region, and

WWW.HIGHDENT.IR

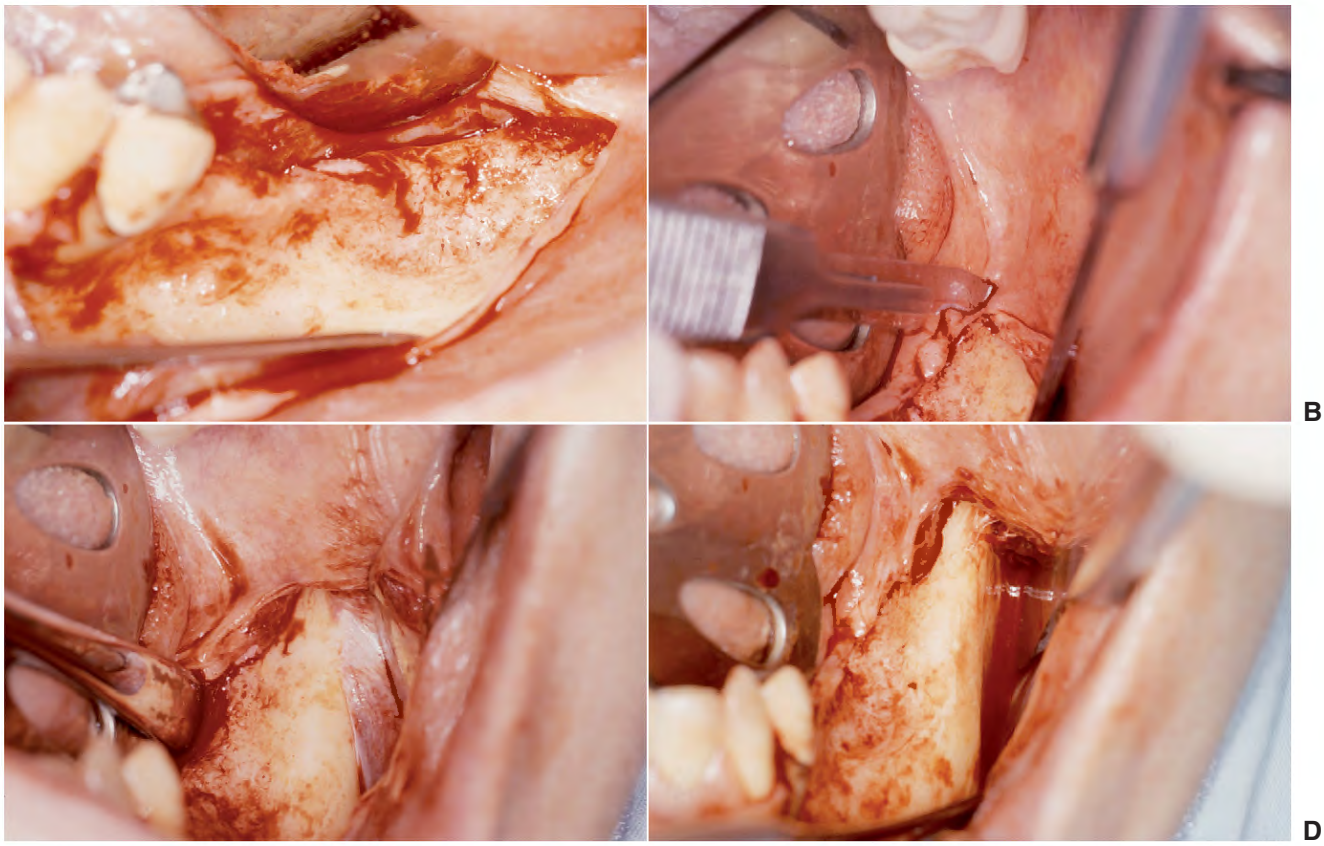


Figure 39-22 **A,** The ramus site is the first option for a block graft, especially when the posterior mandible requires augmentation. The incision in an edentulous posterior mandible starts at the retromolar pad and continues to the first premolar. **B,** After the full-thickness posterior mucoperiosteal flap is reflected, the incision is extended lateral to the retromolar pad and directly over the bone of the ascending ramus to the height of the occlusal plane. **C,** The facial flap is reflected, and the attachment of the buccinator muscle is identified. **D,** A periosteal elevator slides along the lateral aspect of the ramus, under the masseter muscle, for a depth of 15 mm. The incision is extended along the ascending ramus when the donor block requires additional length.

Continued

identifies the mental foramen (see Figure 39-22, *C* to *F*). The host site is reflected and prepared for the block graft (see Figure 39-22, *G* to *I*).

The donor site is evaluated and includes the ramus and body of the mandible (Figure 39-23). The anterior vertical cut may then be made and begins in relation to the existence and width of the buccal shelf (extend oblique ridge). Usually, the mid-first molar is in the position of the anterior vertical cut. A No. 557 surgical bur (or saw) may be used through the cortical bone and to the horizontal cut 2 mm above the IAN position. After the vertical cut approximates the position of the IAN, the cut is limited to the thickness of the cortical plate, usually 3 to 4 mm thick. Therefore the cut is progressively deepened until bleeding from the underlying trabecular is observed.

The anterior cut is usually 10 to 12 mm in length (Figure 39-24, *A*).

The posterior cut is then addressed and is usually above and lateral to the IAN (in front of the linguali on the lingual of the ramus). The posterior cut may be full thickness through the cortical plate to the horizontal cut. Because the nerve in this region is usually below or posterior, the cut is made through the entire depth of the cortex (see Figure 39-24, *B*).

The width of the ramus and the external oblique lateral of the mandibular body are identified. A straight hand piece and a small round drill (No. 2 to 4) punctures the bone 3 to 6 mm from the lateral aspect of the ramus and external oblique for the superior cut. The holes should allow at least 3 mm of bone on the lingual of the ramus and 2 mm of bone adjacent to the molar teeth (when present). The length of the graft is determined, as dictated by the host site (previously reflected and prepared). The penetrating holes, just through the cortical bone, are then connected with a fissure bur (No. 557 surgical length). The depth of the osteotomy may be 2 mm above the IAN (see Figure 39-24, *C, D*).

The inferior osteotomy connecting the posterior and anterior vertical cuts may be performed with an oscillating saw or a large round bur (No. 8) in a straight

WWW.HIGHDENT.IR

همیار دندانسازان

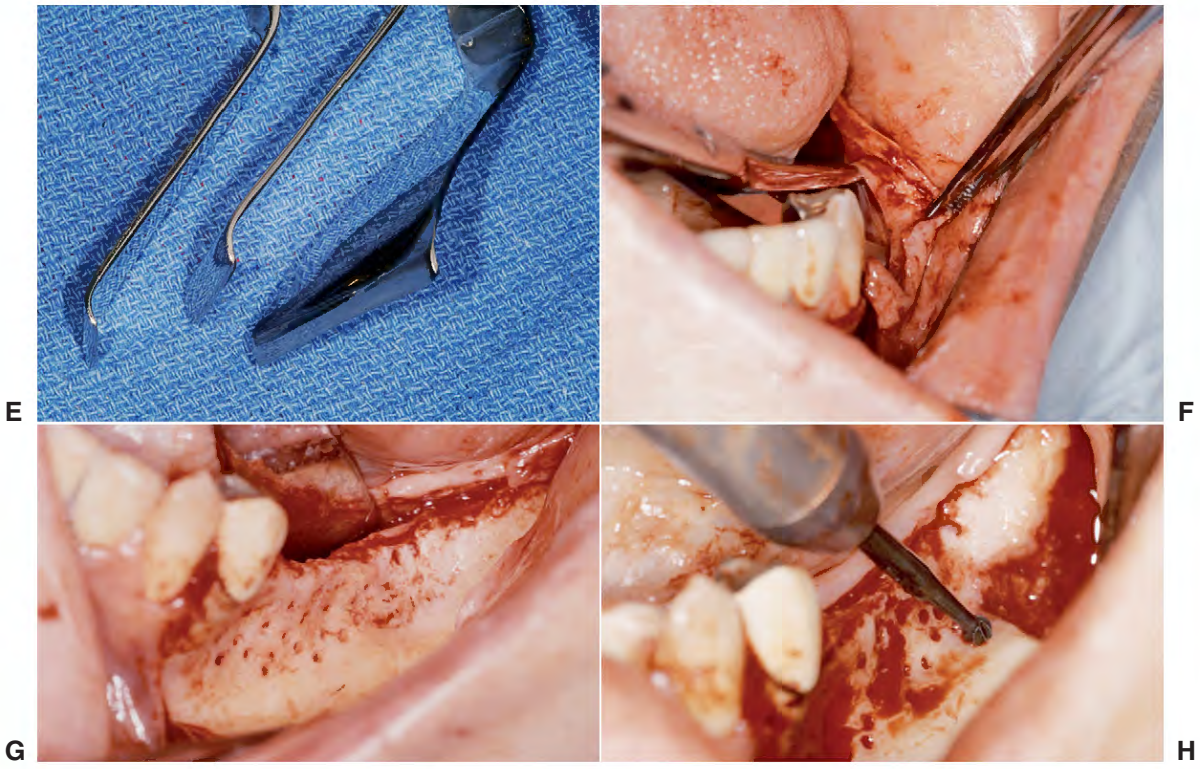


Figure 39-22, cont'd **E,** Ramus retractors are shaped to retract the masseter and curved to allow the preparation of the block at the inferior margin. **F,** The facial flap is advanced. Metzenbaum tissue scissors are used in a blunt dissection to create the submucosal space. **G,** The host site is prepared for the graft with small holes through the cortical plate, 3 to 5 mm apart. **H,** The host site is prepared with a round bur to create a wall of bone at the apical region of the graft.

hand piece. Because access and visibility are limited when making the inferior osteotomy, a more shallow cut may be extended into the cortex only, to create a line of fracture. This inferior cut should not be made completely through the cortex, as it may be located over the mandibular canal (see Figure 39-24, *E, F*).

A thin chisel is gently tapped along the entire length of the external oblique osteotomy, taking care to parallel the lateral surface of the ramus to avoid inadvertent injury to the inferior alveolar nerve. A wider wedge chisel or Potts elevator may then be inserted and levered to pry the bone block segment free and complete the greenstick fracture of the graft from the ramus. After removal of the block, any sharp edges around the ramus are smoothed with a bur or file. Bone wax is reserved for heavier osseous bleeding (see Figure 39-24, *H*). The block graft is then fixated to the host site (see Figure 39-24, *I* to *K*). The block graft may be reentered in 4 to 6 months, implants inserted, and restored 4 months later (see Figure 39-24, *L* to *S*; Figure 39-25). A hemostatic dressing (collagen, gelatin sponge, oxidized regenerated cellulose) may be placed into the donor area, and closure of the site may be completed after fixation of the graft to the receptor site.

Graft Fixation

After the donor bone harvest, the graft may be stored in sterile saline or immediately fixated to the recipient site. Minimal time should elapse before placement of the block on the host bone.[128,129] The trabecular surface of the graft (when present) should be in contact with the underlying cortex of the host bone, which has been decorticated.[82,130,131] The graft may be recontoured to better adapt to the recipient site; however, it should not be particulated or milled into finer pieces.[132,133] The host site should be contoured to fit the donor graft as often as the donor graft is contoured to fit the host site. After the donor graft adequately rests on the recipient site, without mobility or rocking under firm pressure, two or more fixation screw sites may be prepared for each bone block.

The holes in the donor block graft should be slightly larger than the outer diameter of the fixation screws,

WWW.HIGHDENT.IR

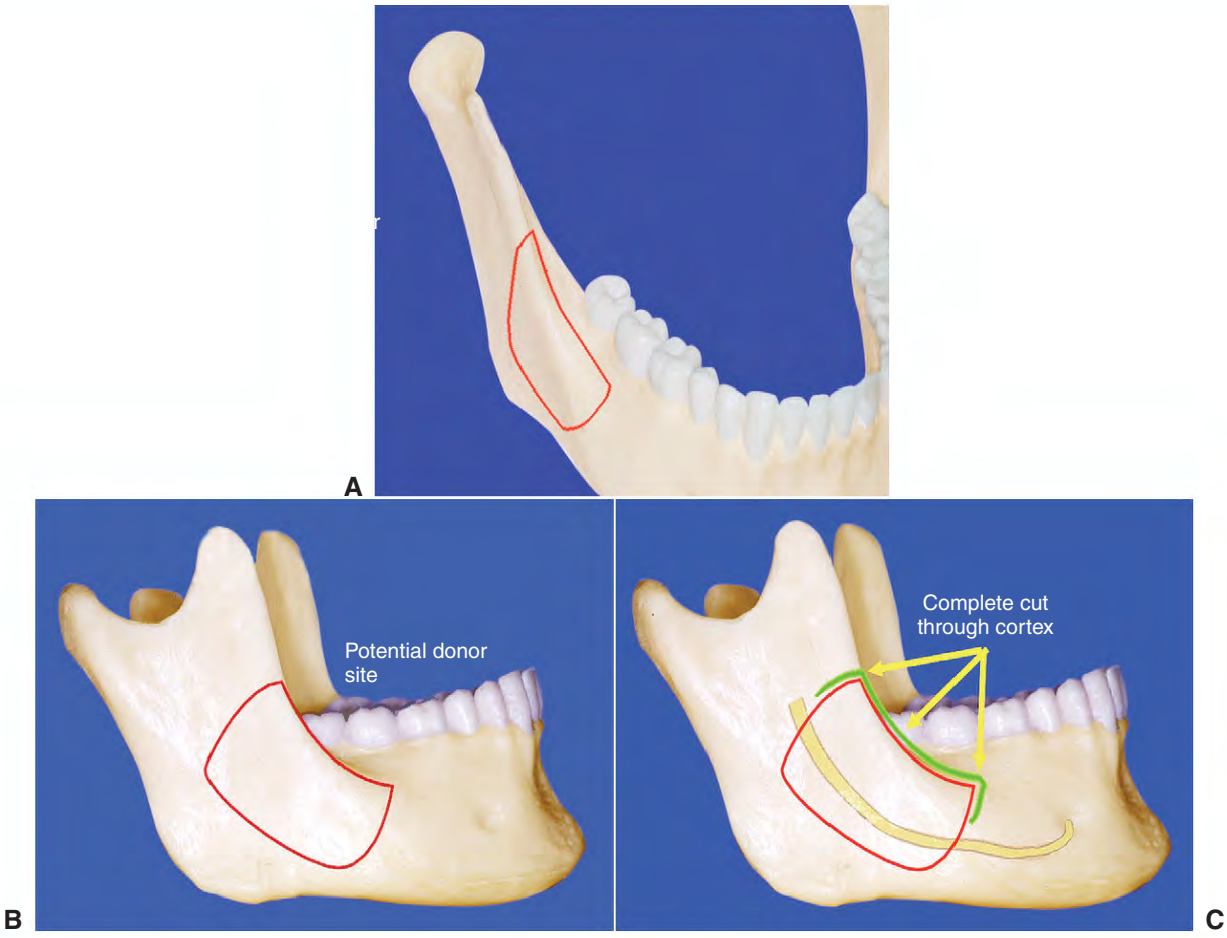


Figure 39-23 **A,** The ramus donor site is lateral to the molars (buccal shelf region) and extends up the ascending ramus. **B,** The ramus donor site uses the outer cortical bone of the ramus and posterior body of the mandible. **C,** The top portion of the ramus donor block *(green line)* is usually above and lateral to the position of the inferior alveolar nerve complex.

but smaller in diameter than the head of the screw. This allows the fixation screw to slip through the block graft and thread into the host bone. The large head diameter of the screw compresses the block graft against the host bone while it threads into the host site for adequate immobilization (Figure 39-26).

The outer thread of the fixation screw is usually 1.4 to 2.0 mm in diameter with a V-shaped thread design. This allows the screw to thread into the host bone during the procedure, and as important, unthread from the host and donor bone after graft maturity. Unlike fracture fixation screws, these devices must be designed to be removed at graft maturity, because endosteal implants will be placed through their pathway. The head of the fixation screw should be 2.2 mm or more in diameter and flat so that it can compress the donor block graft against the host bone.

A lag fixation screw design should not be used. A lag screw design has smooth metal for 5 to 10 mm below the head, and the apical half of the screw has threads. When this device is placed through the block and fixates into the host bone, the concept works well (Figure 39-27). However, when the screw is removed before implant placement, the bone has grown around the smooth neck of the screw and the apical threads must tap the block while it is pulled out to remove the screw. This may pull the block off the host bone, because the interface is still weak between the host and donor.

The end of the screw should be pointed so that it may be self-tapping in softer bone types. A flat end on the screw is of benefit to minimize soft tissue inflammation if it penetrates the opposing cortical plate, but cannot advance in a fixation site that is under prepared in depth.

The surgeon should have two screw diameters available to fixate the block (i.e., 1.4 and 2.0 mm). The smaller diameter is usually first inserted. On occasion, the host bone is too soft or has been stripped when attempting to fixate the block with a small-diameter

WWW.HIGHDENT.IR

همیار دندانسازان

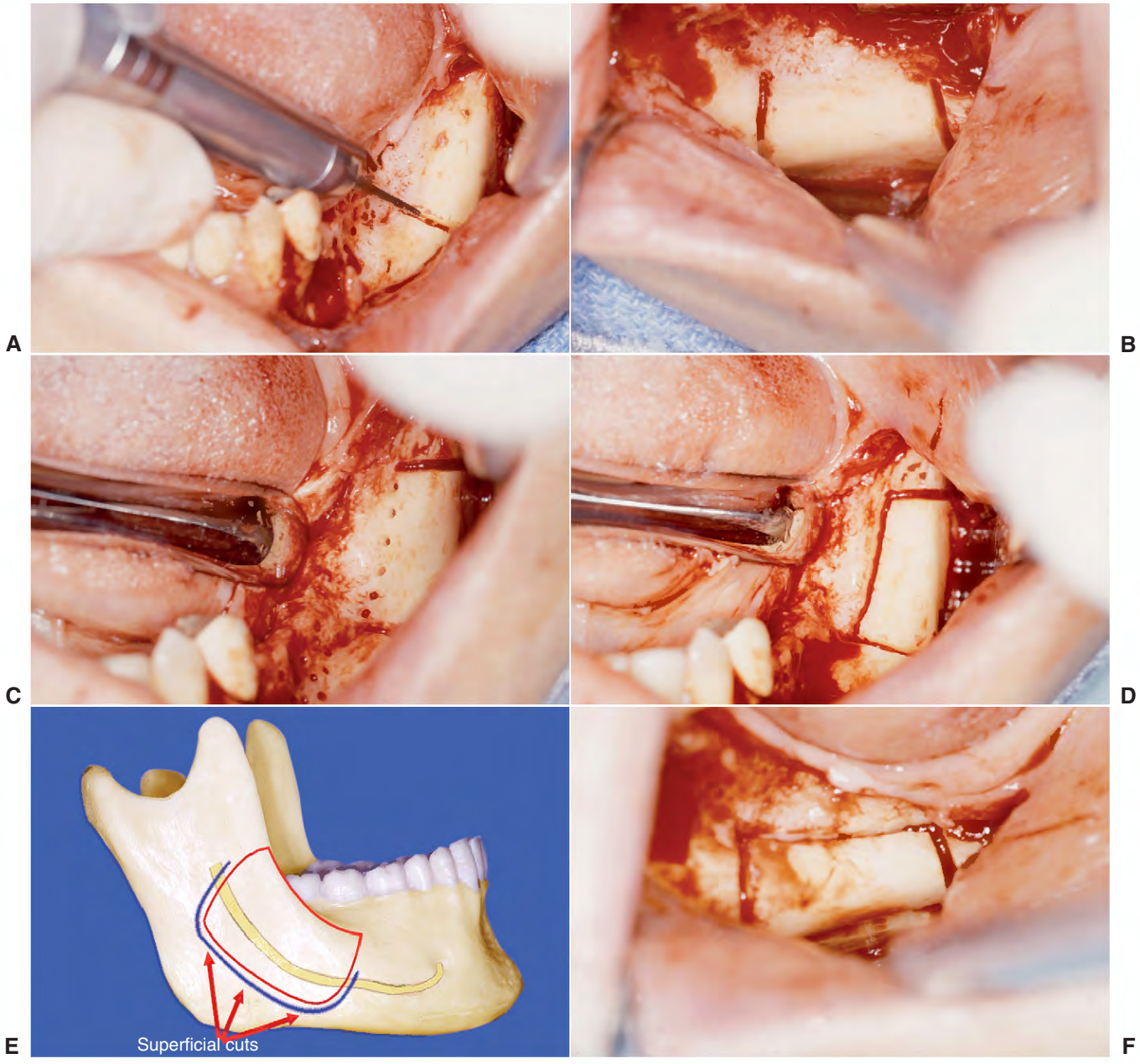


Figure 39-24 **A,** The anterior vertical cut is made with a straight hand piece and a fissure bur or oscillating saw. The position is often at the mid first molar region. The cuts 5 mm above the inferior alveolar nerve (IAN) complex are full thickness. **B,** The posterior vertical cut is often above and lateral to the lingual and IAN and therefore may be made full thickness along the lateral ramus to the superior block margin. The width of the bone block on the superior margin is designed with a small round bur and ranges from 3 to 6 mm in width from the lateral border. **C,** The superior osteotomy is made through the cortical bone and may extend to within 5 mm of the posterior IAN. The horizontal dimension of the ramus block determines the width of the donor site and is related to the amount of bone needed and the anatomy of the donor site. **D,** A fissure bur connects the pilot holes of the horizontal cut. This cut is through the cortical plate and may proceed to 2 mm above the IAN. **E,** The inferior portion of the ramus block is lateral to the IAN. These bone cuts are only thought the cortical bone and do not extend into the trabecular spaces. **F,** The ramus retractor creates a pouch under the masseter muscle and the round bur scratches the bone at the desired depth of the bone block. *Continued*

screw. A larger-diameter screw permits the first to be removed and the same site used to secure the block. Another advantage of two different diameters at surgery is the larger-diameter drill prepares screw sites in the block graft. As a result, the smaller-diameter screws pass through the holes for the lag effect. The smaller-diameter drills prepare the host bone for the RAP and the holes to receive the smaller-diameter screw.

After the graft is mortised to the host bone, two screws or more secure the block. When only one screw fixture is used, the block may rotate or become mobile during initial healing. A high-speed Lindemann bur or

WWW.HIGHDENT.IR

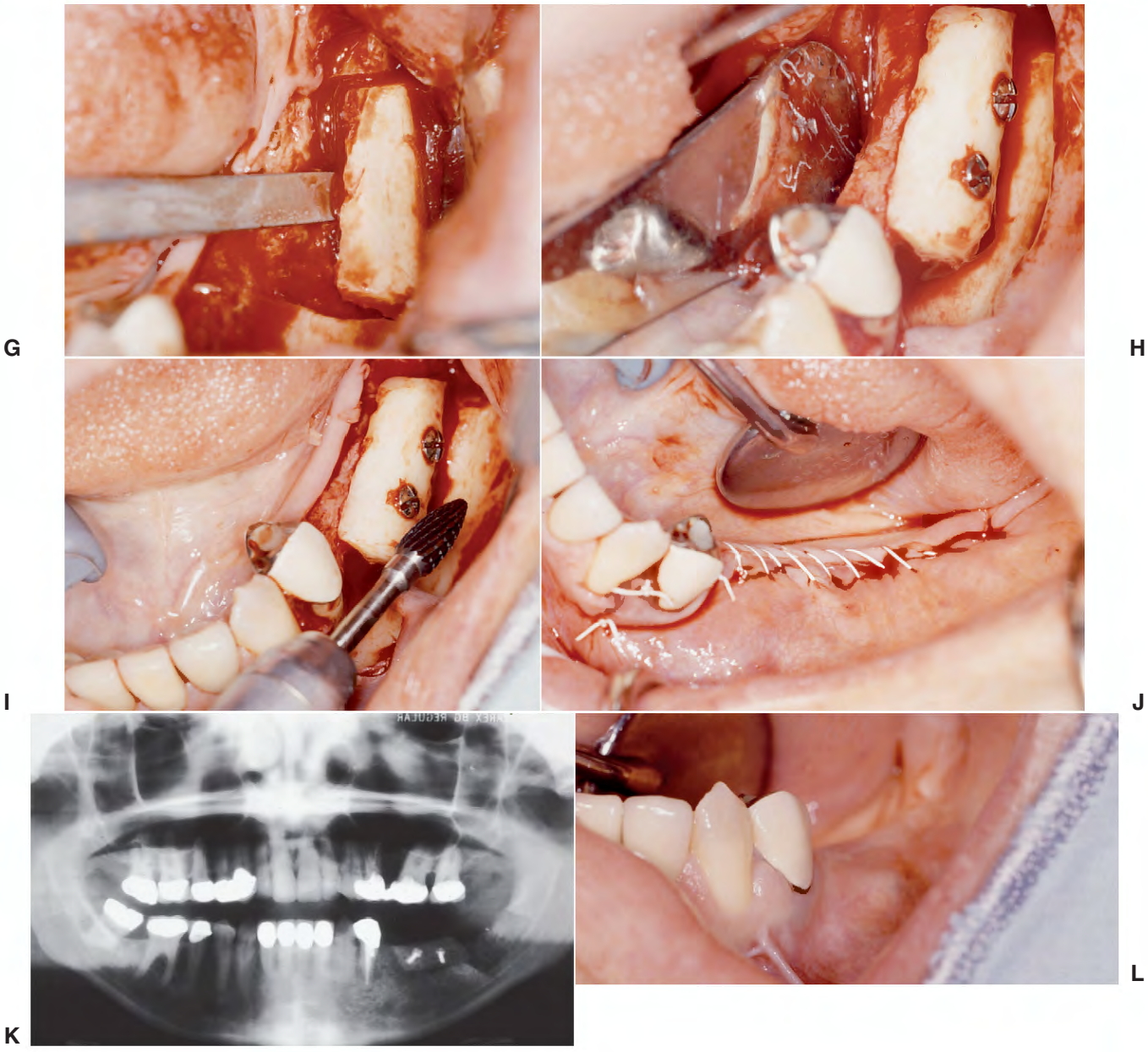


Figure 39-24, cont'd **G,** A bone chisel is placed into the superior cut and greenstick fractures the block off the ramus. **H,** The ramus donor block is fixated to the posterior mandible with two fixation screws. **I,** The block is recontoured in situ. **J,** Primary closure without tension covers the graft site. **K,** An immediate postoperative panoramic radiograph of the donor and host site in the posterior mandible. **L,** Soft tissue healing after 5 months.

Continued

carbides are then used to recontour the bone block after it is fixated in place. It is faster and easier to recontour the block after it is fixated than to attempt to do this in your hand. Any sharp corners or edges are smoothed with the burs.

When an augmentation for height is required, the entire graft should be composed of block graft. Particulate grafts are most predictable for increase of width and when walls of bone are present around the material. A bone graft for height is more prone to incision line opening. Therefore soft tissue–borne provisional restorations should not be used the first month after the graft and then sparingly, only for esthetics.

Trabecular bone harvested from the donor site can be used to fill small discrepancies around block grafts. A mixture of platelet-rich plasma (PRP) and fine particle autograft, allograft, or alloplast may also be used as a filler to provide an additional source of platelet-derived growth factor and transforming growth factor beta.[133-140] Although transforming growth factor beta and platelet-derived growth factor are not osteoinductive, they are capable of promoting bone regeneration in the presence

WWW.HIGHDENT.IR

همیار دندانسازان

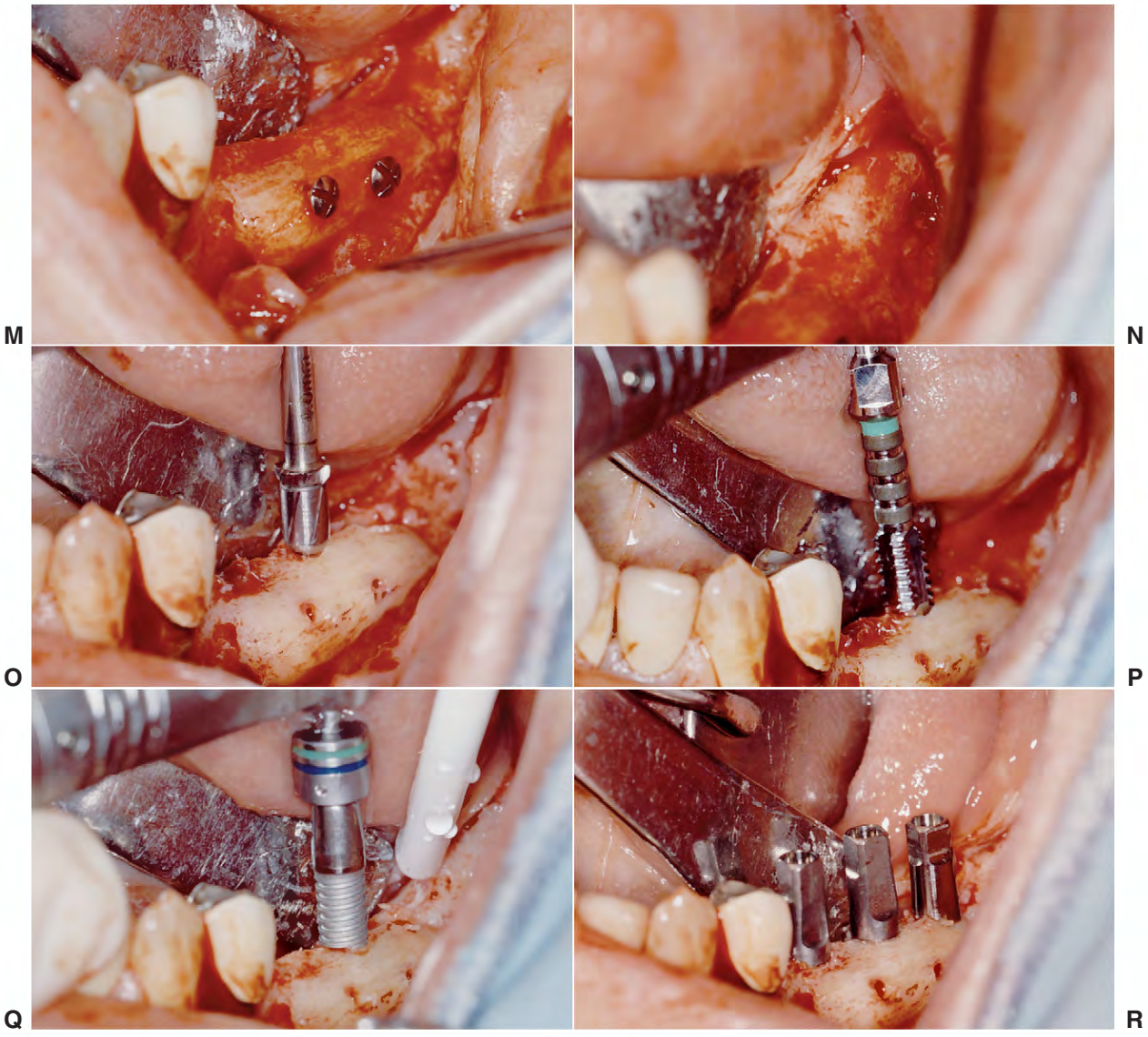


Figure 39-24, cont'd **M,** Reentry into the graft site demonstrates minimal resorption of the graft. **N,** The ramus region has filled with bone, although the external oblique is not as distinct. **O,** The implant osteotomy includes a crest module bur to prepare the bone, which is cortical in nature. **P,** The implant site is tapped to reduce the risk of block fracture from the host site. **Q,** Threaded implants (BioHorizons) are placed into the graft site. **R,** Three endosteal implants are inserted to replace the second premolar and two molars.

of graft materials. In addition to the biological advantages of growth factor amplification, the PRP acts as a fibrin glue to provide adherence of the graft particles for ease of placement around the graft site.[138]

Although it appears that barrier membranes may help minimize autograft resorption,[53,88,141,142] they are not routinely necessary with cortical block bone grafts, which completely fill the graft site and already show minimal volume loss. Not only is the routine use of a barrier membrane questionable, but it also adds additional costs and may lead to complications if early exposure occurs. It also adds unnecessary volume to the graft site, which may increase tension on the overlying soft tissue and increase the risk of incision line opening.

However, if the size of the block graft is inadequate to fill the entire space of the graft site or the harvest is more particulate or trabecular in nature, a barrier membrane should be used. When the host site is larger than two teeth and the block bone graft is inadequate in length, the block should be divided into two pieces. Two pieces are often easier to recontour and fit into position. As important, they may be separated by 5 to 8 mm. In this way, the space between the blocks acts as walls of host bone for the particulate graft. In other words, the particular graft has block graft walls on the

WWW.HIGHDENT.IR

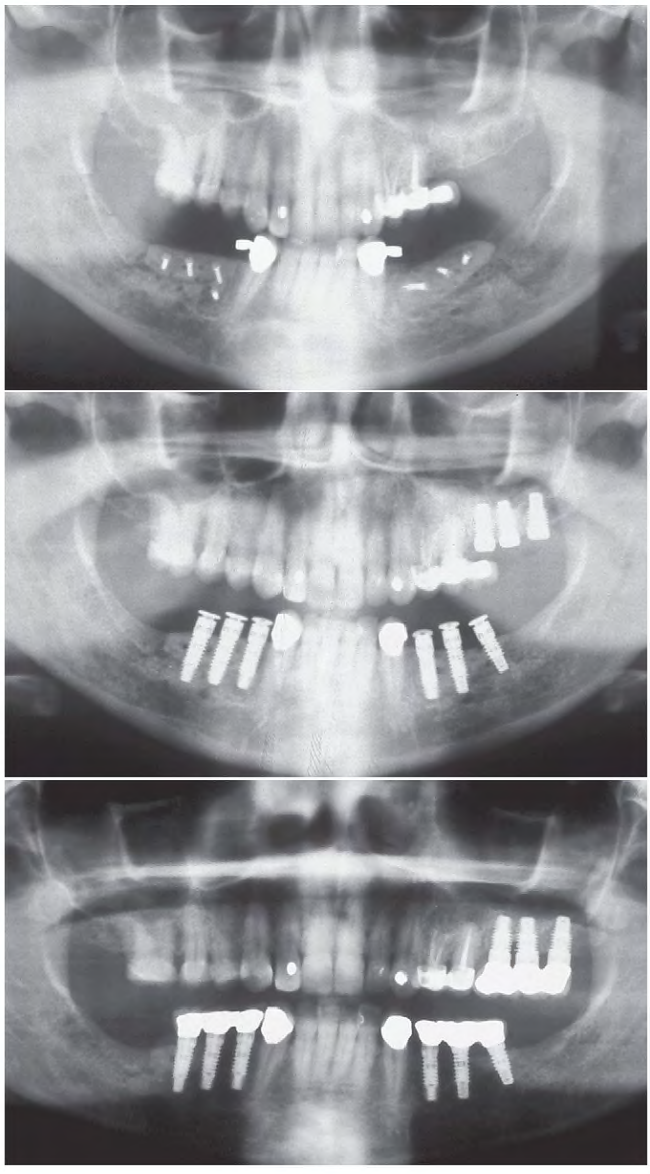


Figure 39-25 **A,** Immediate postoperative panoramic radiograph of block grafted bilateral ramus. **B,** After healing of the block bone grafts, implants are inserted into the posterior edentulous regions. **C,** The quadrants are restored with splinted crown for the posterior dentition.

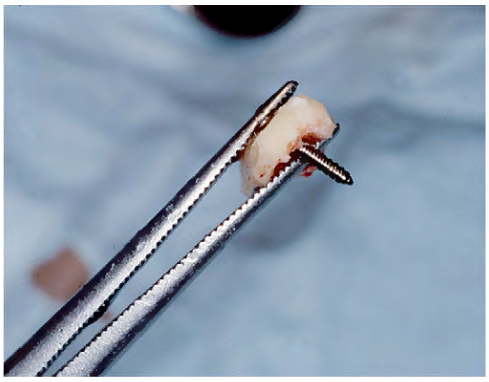


Figure 39-26 A lag screw approach is used to fixate the block. The screw slides through the block, and the screw head is larger than the hole in the block.

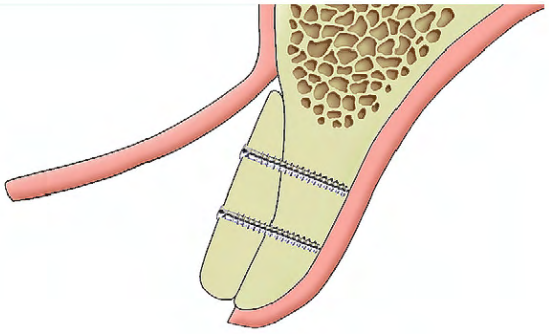


Figure 39-27 The screw should fixate each bone block. The screws should engage the lingual plate of the host site.

mesial and distal and host bone on the lateral and apical region. Whenever particulate bone is used as a portion of the graft, a resorbable barrier membrane such as acellular dermal matrix should be used over the particulate graft (Figure 39-28).

Studies on the size of particulate autologous bone grafts have shown that smaller particles resorb more quickly and completely than larger graft particles.[132,133] Therefore the autologous particles are usually 3 × 5 mm. When inadequate autologous particles are available, a layered approach is indicated. The autograft is layered over the host bone, the next layer is a mixture of 30% demineralized freeze-dried bone (DFDB) fibers (Grafton), 70% mineralized HA (e.g., FDB, xenograft), and PRP. The top layer is a collagen barrier membrane. A barrier membrane may be used to help contain and stabilize the particulate graft, allow bony regeneration of any remaining space, and minimize the overall volume loss.

Although it is imperative that the soft tissue flap margins be well approximated, the sutures must not be pulled too tightly or tissue ischemia may result. Because the early strength of the healing wound is not achieved for several days, the sutures must be capable of maintaining closure for 2 weeks. If the bone augmentation is large and advancement was more difficult, it is best to use nonabsorbable sutures such as Gore-Tex or Prolene, which maintain their tensile strength for several weeks, and allow them to stay in place 3 weeks. Mucosal dehiscence and premature exposure of the autologous bone transplant are the most common causes of graft failure.* Therefore great care should be taken to properly close the surgical site. Usually horizontal or vertical mattress sutures combined with continuous sutures can achieve proper closure with minimal tension.

*References 22, 25, 38, 59, 61, 143, 144.

WWW.HIGHDENT.IR

همیار دندانسازان

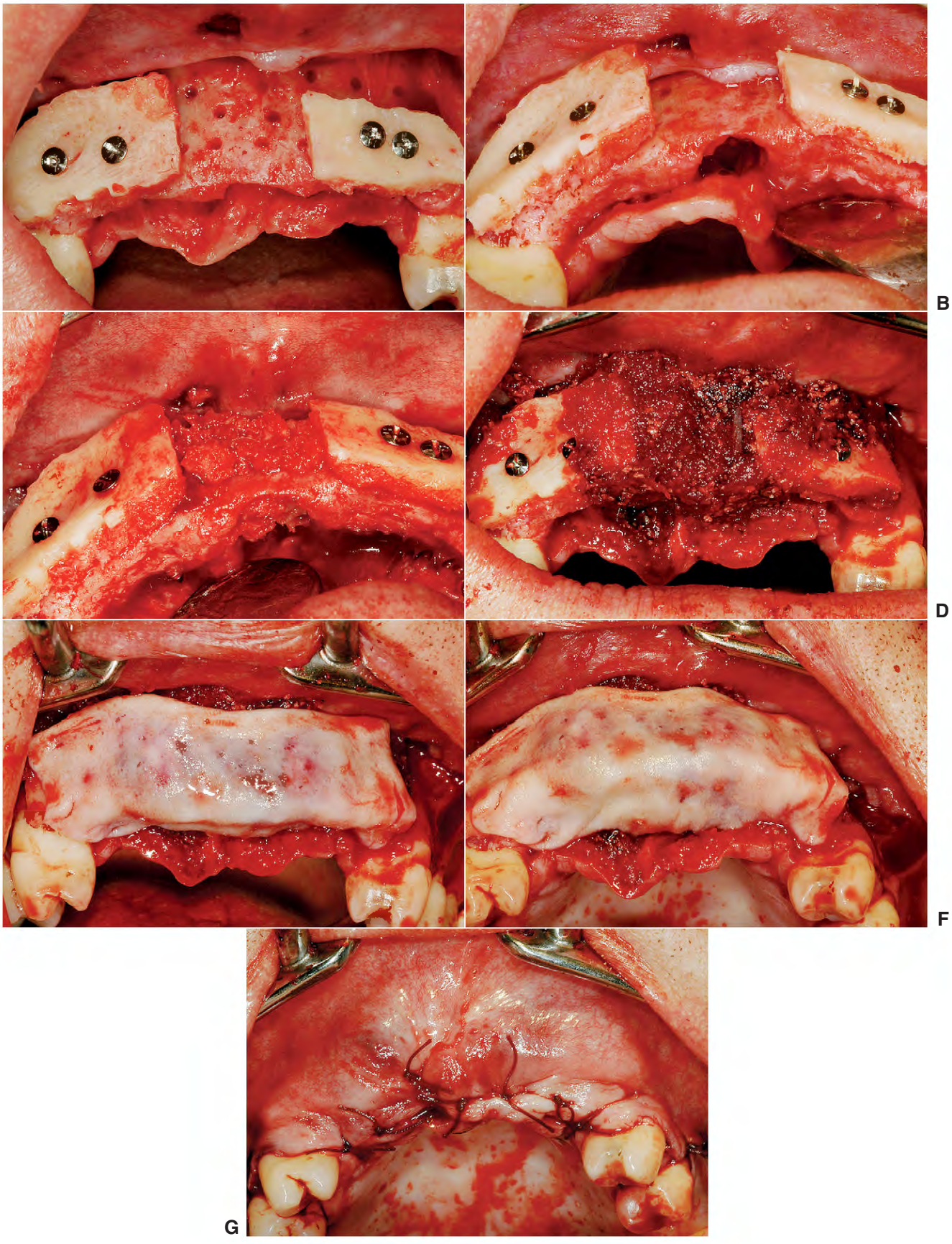


Figure 39-28 **A,** The blocks are positioned in key implant sites. **B,** The lingual tissues are reflected and the incisive foramen evaluated for size and position when a central incisor implant is indicated. **C,** A particulate autograft is placed on host bone between the blocks and into the incisive canal site. **D,** The second layer of the particulate graft is a mixture of 30% demineralized freeze-dried bone fibers, 70% mineralized bone (i.e., freeze-dried bone) and platelet-rich plasma. **E,** A barrier membrane is used when a particulate graft is placed. **F,** The barrier membrane is positioned under the palatal flap. **G,** Soft tissue closure, without tension, covers the bone graft.

WWW.HIGHDENT.IR

Postoperative Instructions

An increase in incision line opening risk has been associated with postoperative smoking and diabetes.[26,145] Patients should stop smoking at least 3 days before surgery and until the incision has healed. It is imperative that the graft be immobilized during healing. Removable soft tissue–borne prostheses should not be worn or should be adjusted to prevent graft loading. The flange area of a removable prosthesis is completely removed, and the edentulous ridge area is generously relieved, which requires the patient to use denture adhesive for prosthesis retention. The patient is instructed to use the provisional removable prosthesis for cosmetic appearance rather than function.

Careful postoperative follow-up is necessary to inspect the bone graft region and eliminate pressure areas from an overlying prosthesis. More favorable provisional solutions are tooth-borne fixed or removable partial dentures, resin-bonded bridges, or denture teeth bonded to the adjacent dentition.[146] The use of transitional implants to support a fixed-interim prosthesis during the healing phase may be considered for patients less tolerant of removable provisional restorations.[147,148]

A comparison of intraoral donor sites for onlay grafting before implant placement was reported by Misch.[44] The volume of the symphyseal donor grafts was almost twice as great as ramus sites (1.74 cm^3 versus 0.9 cm^3). The ramus was primarily a cortical graft, whereas the symphysis block was cortical/trabecular. Dehiscence of the donor site occurred in 10.7% of anterior mandibles, and no incision line opening was found in the posterior mandible or ramus. Temporary nerve paresthesia was reported to be 9.6% in symphyseal graft patients, whereas no sensory complications were observed in ramus graft patients. Altered feeling of the lower anterior teeth affected 29% of anterior mandibles, and no ramus donor block patients reported changes in their posterior teeth. Bone quality was most often D1 for the ramus (11 of 19 cases) and D2 for symphyseal block patients (19/31). There was no difference in the amount of resorption on reentry, no difference in success rates, and no difference in implant survival. Therefore when possible, the ramus site is the first option, because the local consequences of the graft harvest are less (Table 39-1).

Complications: Symphysis Donor Site

The most common concern of patients is the postoperative appearance of their chin. No complications were found on young cleft palate patients treated with augmentation of the symphyseal donor site.[36,37] Ptosis of the chin has not occurred and can be prevented by avoiding degloving of the facial and inferior aspects of the mandible and exposing the lingual aspect.[149] When this approach is used to harvest the symphysis, use of pressure bandages for 3 days is in order.

Although radiographic evidence of incomplete bony regeneration has been reported in elderly patients, it did not result in any discernible profile changes.[39] No alteration in chin contour has been observed clinically or radiographically when grafting the donor area.[38,41,111,150] Although reentry to the symphysis after 4 to 6 months for additional bone harvest has been successful, it is recommended that extraoral donor sites be selected for larger graft requirements rather than making the patient undergo repeated surgeries and extended healing periods between each bone graft.

Although a 5-mm border below the apices of the anterior tooth roots is recommended,[38,41,151] the contents of the incisive canal that innervate the teeth may still be disturbed. Altered sensation of the lower anterior teeth is a relatively common postoperative symptom of the symphysis graft patients.[44,62,150-156] They describe a dullness in sensation of the incisors, which usually resolves within 6 months. Studies showed a significant

Table 39-1 Comparison of Mandibular Donor Sites

CRITERIA	SYMPHYSIS	RAMUS
Surgical access	Good	Fair to good
Patient cosmetic concerns	High	Low
Graft shape	Thicker block	Thinner veneer
Graft morphology	Corticocancellous	Cortical
Graft size (cm^3)	>1	<1
Graft resorption	Minimal	Minimal
Healed bone quality	D1, D2	D1, D2
Donor site complications		
Postop pain/edema	Moderate	Minimal to moderate
Neurosensory: teeth	Common (temporary)	Uncommon
Neurosensory: tissue	Common (temporary)	Uncommon
Incision dehiscence	Occasional (vestibular)	Uncommon

WWW.HIGHDENT.IR

همیار دندانسازان

loss of sensitivity that could last well into 12 months. A prospective study showed 20% reduced nerve sensation and 40% superficial impairment.[153,154]

Hoppenreijs et al.[151] examined the lower anterior teeth in children treated with mandibular symphyseal bone grafts and found a high frequency of pulp canal obliteration (12%) and negative pulpal reaction (16%). Reports on tooth vitality after genioplasty or subapical osteotomy also reveal that a substantial percentage (20% to 25%) of teeth test nonvital.[156-158] However, unless clinical signs of pulpal necrosis become apparent (discoloration, radiographic change), endodontic therapy is not indicated.[159] Even if discoloration of a tooth is discovered, it may be closely followed because return of vitality has been observed.[160] Although the author has not observed the need for endodontic therapy, patients should be aware of the risk of pulpal damage.

Complications: Ramus Donor Site

Compared with the symphysis region, the ramus donor site is associated with fewer postoperative complications.[19,44,150,156] Patients have also shown less concern with bone removal from the ramus area, and augmentation of this donor site has been unnecessary. Although vestibular incision dehiscence has occurred with chin grafts, it has not been found with posterior donor sites. Patients are less able to discern neurosensory disturbances in the posterior buccal soft tissues compared with the lower lip and chin. Although the incision along the external oblique ridge could possibly damage the buccal nerve, reports of postoperative sensory loss in the buccal mucosa are less frequent and will most likely go unnoticed by the patient.[161,162] In contrast to the teeth superior to the symphysis donor site, patients have reported minimal altered sensation in their molar teeth.[44,150]

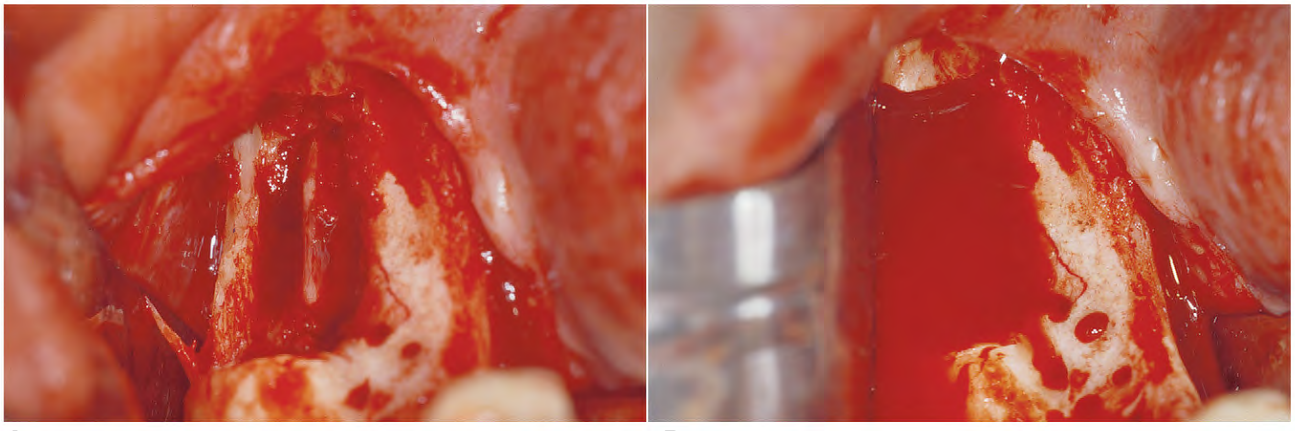
Damage to the neurovascular bundle could also occur during sectioning of the graft. Bone chisels should parallel the lateral surface of the ramus. If the inferior ramus cut is below the level of the inferior alveolar canal, graft separation should not be completed until it can be verified that the neurovascular bundle is not trapped within the graft. Although nerve injury to the inferior alveolar nerve is low, patients should be aware of this risk. On occasion, the IAN is identified and directly observed when the block graft is removed from the ramus (Figure 39-29). When this occurs, Decadron 4 mg, 1 cc, may be placed directly on the nerve for 30 seconds to reduce inflammation and edema. A second dose of 4 mg, 1 cc then is applied for an additional 30 seconds. A collagen sponge may be placed over the site, but HA or graft material is not indicated.

Complication: Host Site

The most common host site complication is graft dehiscence and incision line opening. If graft dehiscence occurs, the wound is allowed to heal by secondary intention after 2 to 4 weeks. The block graft may then be recontoured with a diamond bur to reduce the bulk of exposed bone. The bone above the margins of the tissue is ground off. This procedure is repeated every 2 to 4 weeks until the site is closed (Figure 39-30). No attempt should be made to advance a soft tissue flap to recover the graft for at least 6 to 8 weeks. If the graft becomes mobile, the mobile piece of bone should be removed.

Implant Placement

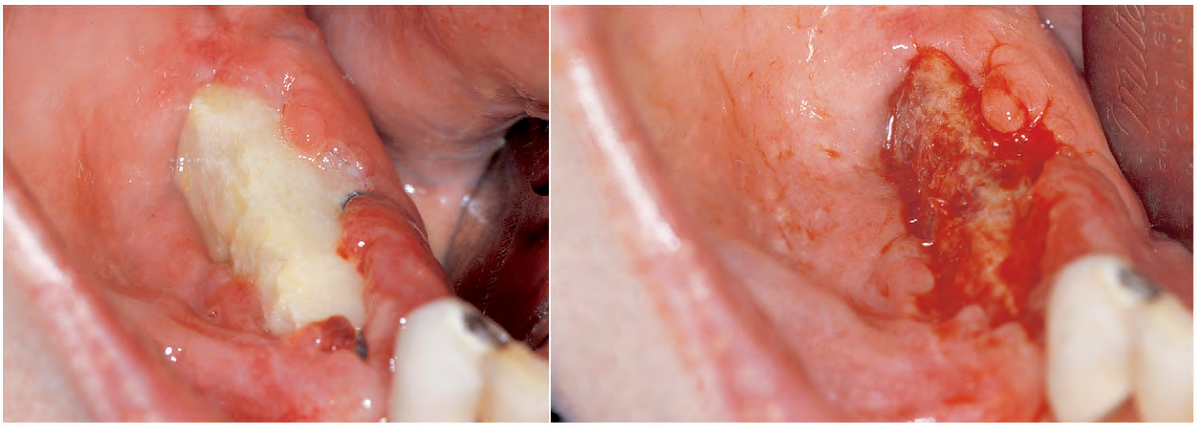
Sufficient healing time should be allowed for graft incorporation, but implants should be placed early enough to take advantage of their bone-preserving stimulus.[32,39,163] Intraoral block grafts are allowed to heal for a minimum of 4 months for maxillary recipient sites and 5 to 6 months for mandibular sites. Compared with the recommended 6- to 9-month healing period for bone grafts of endochondral origin, a 4-month healing period has been found to be adequate in clinical studies on mandibular bone grafts.[38,58] The



A B

Figure 39-29 **A,** On occasion the inferior alveolar nerve complex can be identified after the block is removed. **B,** When this occurs, Decadron 4 mg (1 cc) may be used directly on the nerve to reduce inflammation.

WWW.HIGHDENT.IR



A B

Figure 39-30 **A,** Incision line opening or block dehiscence is a complication of block bone grafts. **B,** The soft tissue should not be resutured over the graft that dehisces during the first few months. Instead, the block is recontoured so the bone above the tissue margins is removed. The area is left to heal by secondary intention.

shorter healing time was based on the hypothesis that membranous bone grafts revascularize earlier than endochondral grafts.[72,164] The rich vascularity of the maxillary cortex in comparison to the mandible allows a more rapid angiogenesis of the onlay graft. The longer healing period for mandibular sites is also to ensure an adequate union between the graft and denser cortex of the host bone. This is critical because the implant osteotomy and insertion are along the graft-host junction. Particulate onlay grafts should be allowed to consolidate and heal for as long as 6 to 9 months unless autologous growth factors have been added to the graft.

As mandibular donor grafts exhibit minimal resorption, predictable gains in bone volume allow implant placement in most planned sites. A staged treatment plan with implant placement following graft healing is the preferred method of reconstruction. Reports on simultaneous implant insertion during bone graft placement have revealed complications such as block graft fracture, wound dehiscence with exposure of implants and graft, and a higher implant failure rate compared with a staged approach.[38-40,46,55,59] In addition, diminished bone contact has been found around titanium implants placed simultaneously with autologous grafts.[12,30,86,165] A staged surgery permits implant placement for ideal prosthetic alignment without the concern of graft fixation or remodeling. Staging the implant placement also provides an improved vascularity of the transplanted bone as the exposed surface area is increased and unimpeded by an inert biomaterial.[53,166] It also allows for any unanticipated increase in graft resorption and should provide a more stable foundation. The implant-bone interface should be improved, as the implant surface is in close contact with the already incorporated bone graft.

The insertion of implants into healed bone grafts as a secondary procedure is similar to their use in jaws that have not been grafted. Autologous bone grafts offer an improved quality of bone at earlier healing times compared with allogeneic bone grafts or guided bone regeneration techniques.[38,58,88,167-169] The density of healed block mandibular bone grafts has been found to be D1 to D2 regardless of the original quality of the recipient site.[38,44,58] An appropriate drilling sequence for dense bone and tapping may be necessary for atraumatic implant placement. The implant surgery activates bone formation and induces interfacial remodeling with bone maintenance, even in unloaded conditions.[170,171] After integration, a progressive bone loading of the implants is recommended.[172] Additional graft resorption after implant insertion has not been noted radiographically on loaded cases.[173-176]

SUMMARY

Autologous bone grafts continue to be the gold standard for jaw reconstruction. Bone harvested from the maxillofacial region offers several advantages in the reconstruction of the residual ridge for implant placement. Intraoral donor sites only require one operational field, which decreases the surgical and anesthetic time. Larger block-type grafts may be harvested from the mandibular symphysis, body, or ramus area. Particulate autograft may be harvested from the maxillary tuberosity, extraosseous tori, ridge osteoplasty, extraction sites, implant osteotomy, and bone collection devices. These grafts require a short healing period and exhibit minimal resorption while maintaining their dense quality. The morbidity of graft harvest is low, and complications usually result in only temporary

WWW.HIGHDENT.IR

همیار دندانسازان

debilitation. The use of these techniques allows the placement of implants in ideal positions for optimal esthetics and functional support.

References

1. Misch CE, Dietsh F: Bone grafting materials in implant dentistry, *Implant Dent* 2:158-167, 1993.
2. Buser D, Bragger U, Lang NP et al: Regeneration and enlargement of jaw bone using guided tissue regeneration, *Clin Oral Implant Res* 1:22-32, 1990.
3. Lekholm U, Becker W, Dahlin C et al: The role of early vs. late removal of GTAM membranes on bone formation around oral implants placed in immediate extraction sockets: an experimental study in dogs, *Clin Oral Implant Res* 4:121-129, 1993.
4. Palmer RM, Floyd PD, Palmer PJ et al: Healing of implant dehiscence defects with and without expanded polytetrafluoroethylene membranes: a controlled clinical and histological study, *Clin Oral Implant Res* 5:98-104, 1994.
5. Jovanovic SA, Schenk RK, Orsini M et al: Supracrestal bone formation around dental implants: an experimental dog study, *Int J Oral Maxillofac Implants* 10:23-31, 1995.
6. Pinholt EM, Haanaes HR, Donath K et al: Titanium implant insertion into dog alveolar ridges augmented by allogenic material, *Clin Oral Implant Res* 5:213-219, 1994.
7. Becker W, Lekholm U, Dahlin C et al: The effect of clinical loading on bone regenerated by GTAM barriers: a study in dogs, *Int J Oral Maxillofac Implants* 9:305-313, 1994.
8. Lang NP, Hammerle CHF, Bragger U et al: Guided tissue regeneration in jawbone defects prior to implant placement, *Clin Oral Implant Res* 5:92-97, 1994.
9. Simion M, Baldoni M, Rossi P et al: A comparative study of the effectiveness of e-PTFE membranes with and without early exposure during the healing period, *Int J Periodont Rest Dent* 14:167-180, 1994.
10. Simion M, Trisi P, Piatelli A: Vertical ridge augmentation using a membrane technique associated with osseointegrated implants, *Int J Periodont Rest Dent* 14:497-511, 1994.
11. Fritz ME, Malmquist J, Koth D et al: The use of guided bone regeneration to fill large mandibular defects in monkeys: a pilot study, *Int J Oral Maxillofac Implants* 9:644-652, 1994.
12. Becker W, Schenk R, Higuchi K et al: Variations in bone regeneration adjacent to implants augmented with barrier membranes alone or with demineralized freeze-dried bone or autologous grafts: a study in dogs, *Int J Oral Maxillofac Implants* 10:143-154, 1995.
13. Caplanis N, Sigurdsson TJ, Rohrer MD et al: Effect of allogeneic, freeze-dried, demineralized bone matrix on guided bone regeneration in supra-alveolar peri-implant defects in dogs, *Int J Oral Maxillofac Implants* 12:634-642, 1997.
14. Hammack BL, Enneking WF: Comparative vascularization of autogenous and homogenous bone transplants, *J Bone Joint Surg* 42A:811, 1960.
15. Male AJ, Gasser J, Fonseca RJ et al: Comparison of onlay autologous and allogenic bone grafts to the maxilla in primates, *J Oral Maxillofac Surg* 42:487-499, 1983.
16. Boyne PJ: Performance of bone grafts in reconstructive surgery. In Williams EF, editor: *Biocompatibility of tissue analogs*, vol 2, Boca Raton, Fla, 1985, CRC Press.
17. Burchardt H: Biology of bone transplantation, *Orthop Clin North Am* 18:187-195, 1987.
18. Marx RE: Biology of bone grafts, *Oral Maxillofac Surg Knowl Update*: 1:3-17, 1994.
19. Cordaro L, Amade DS, Cordaro M: Clinical results of alveolar ridge augmentation with mandibular block bone grafts in partially edentulous patients prior to implant placement, *Clin Oral Implants Res* 13:103-111, 2002.
20. Misch CE: Use of mandibular ramus as a donor site for onlay bone grafting, *J Oral Implantol* 26:42-49, 2000.
21. Proussaefs P, Lozada J. Kleinman A, Roaher MD: The use of ramus autogenous block grafts for vertical alveolar ridge augmentation and implant placement: a pilot study, *Int J Oral Maxillofac Implants* 17:238-248, 2002.
22. Proussaefs P, Lozada J: The use of intraorally harvested autogenous block grafts for vertical alveolar ridge augmentation: a human study, *Int J Periodontics Rest Dent* 25:351-363, 2005.
23. Proussaefs P: Clinical and histologic evaluation of the use of mandibular tori as donor site for mandibular block autografts: report of three cases, *Int J Periodont Res Dent* 26:43-51, 2006.
24. Schwartz-Arad D, Levin L: Multitier technique for bone augmentation using intraoral autogenous bone blocks, *Implant Dent* 16:5-12, 2007.
25. Schwartz-Arad D, Levin L: Intraoral autogenous block onlay bone grafting for extensive reconstruction of atrophic maxillary alveolar ridges, *J Periodontol* 76:636-641, 2005.
26. Schwartz-Arad D, Levin L, Sigal L: Surgical success of intraoral autogenous block onlay bone grafting for alveolar ridge augmentation. *Implant Dent* 14:131-138, 2005.
27. Garg AK, Morales MJ, Navarro I et al: Autogenous mandibular bone grafts in the treatment of the resorbed maxillary anterior alveolar ridge: rationale and approach, *Implant Dent* 7:167-176, 1998.
28. Cranin AN, Katzap M, Demirdjan E et al: Autogenous bone ridge augmentation using the mandibular symphysis as a donor, *J Oral Implantol* 27:43-47, 2001.
29. Brånemark PI, Lindstrom J, Hallen O et al: Reconstruction of the defective mandible, *Scand J Plast Reconstr Surg* 9:116-128, 1975.
30. Breine U, Brånemark PI: Reconstruction of alveolar jaw bone, *Scand J Plast Reconstr Surg* 14:23-48, 1980.
31. Keller EE, van Roekel NB, Desjardins RP et al: Prosthetic-surgical reconstruction of the severely resorbed maxilla with iliac bone grafting and tissue-integrated prostheses, *Int J Oral Maxillofac Implants* 2:155-165, 1987.
32. Listrom RD, Symington JM: Osseointegrated dental implants in conjunction with bone grafts, *Int J Oral Maxillofac Surg* 17:116-118, 1988.
33. Kahnberg KE, Nystrom L, Bartholdsson L: Combined use of bone grafts and Brånemark fixtures in the treatment of severely resorbed maxillae, *Int J Oral Maxillofac Implants* 4:297-304, 1989.
34. Misch CE, Dietsh F: Endosteal implants and iliac crest grafts to restore severely resorbed totally edentulous maxillae: a retrospective study, *J Oral Implantol* 20:100-110, 1994.
35. Joshi A, Kostakis GC: An investigation of post-operative morbidity following iliac crest graft harvesting, *Br Dent J* 196:167-171, 2004.

WWW.HIGHDENT.IR

36. Sindet-Pedersen S, Enemark H: Reconstruction of alveolar clefts with mandibular or iliac crest bone grafts: a comparative study, *J Oral Maxillofac Surg* 48:554-558, 1990.
37. Borstlap WA, Heidbuchel KLWM, Freihofer HPM et al: Early secondary bone grafting of alveolar cleft defects: a comparison between chin and rib grafts, *J Cranio Max Fac Surg* 18:201-205, 1990.
38. Misch CM, Misch CE, Resnik R et al: Reconstruction of maxillary alveolar defects with mandibular symphysis grafts for dental implants: a preliminary procedural report, *Int J Oral Maxillofac Implants* 7:360-366, 1992.
39. Jensen J, Sindet-Pedersen S: Autogenous mandibular bone grafts and osseointegrated implants for reconstruction of the severely atrophied maxilla: a preliminary report, *J Oral Maxillofac Surg* 49:1277-1287, 1991.
40. Jensen J, Sindet-Pedersen S, Oliver AJ: Varying treatment strategies for reconstruction of maxillary atrophy with implants: results in 98 patients, *J Oral Maxillofac Surg* 52:210-216, 1994.
41. Misch CM, Misch CE: The repair of localized severe ridge defects for implant placement using mandibular bone grafts, *Implant Dent* 4:261-267, 1995.
42. Misch CM: Ridge augmentation using mandibular ramus bone grafts for the placement of dental implants: presentation of a technique, *Prac Periodont Aesth Dent* 8:127-135, 1996.
43. Aalam AA, Nowzari H: Mandibular cortical bone grafts part 1: anatomy, healing process, and influencing factors, *Compend Contin Educ Dent* 28:206-212, 2007.
44. Misch CM: Comparison of intraoral donor sites for onlay grafting prior to implant placement, *Int J Oral Maxillofac Implants* 12:767-776, 1997.
45. Misch CM: Enhance maxillary implant sites through symphysis bone graft, *Dent Implant Update* 2:101-104, 1991.
46. Smith JD, Abramson M: Membranous vs. endochondral bone autografts, *Arch Otolaryngol* 99:203, 1974.
47. Zins JE, Whitaker LA: Membranous vs endochondral bone autografts: implications for craniofacial reconstruction, *Plast Reconstruct Surg* 72:778, 1983.
48. Kusiak JF, Zins JE, Whitaker LA: The early revascularization of membranous bone, *Plast Reconstr Surg* 76:510-514, 1985.
49. Lin KY, Bartlett SP, Yaremchuk MJ et al: The effect of rigid fixation on the survival of onlay bone grafts: an experimental study, *Plast Reconstruct Surg* 86:449-456, 1990.
50. Rabie ABM, Dan Z, Samman N: Ultrastructural identification of cells involved in the healing of intramembranous and endochondral bones, *Int J Oral Maxillofac Surg* 25:383-388, 1996.
51. Miller NA, Penaud J, Kohler C et al: Regeneration of bone graft donor sites, *Clin Oral Implants Res* 10:326-330, 1999.
52. Avery JK: Development of cartilages and bones of the facial skeleton. In Avery JK, editor: *Oral development and histology,* New York, 1994, Thieme Medical Publishers.
53. Marx RE: The science of reconstruction. In Bell WH, editor: *Modern practice in orthognathic and reconstructive surgery,* Philadelphia, 1992, WB Saunders.
54. Shirota T, Ohno K, Motohashi M et al: Histologic and microradiologic comparison of block and particulate cancellous bone and marrow grafts in reconstructed mandibles being considered for dental implant placement, *J Oral Maxillofac Surg* 54:15-20, 1996.
55. Lu M, Rabie AB: Quantitative assessment of early healing of intramembranous and endochondral autogenous bone grafts using micro-computed tomography and Q-win image analyzer, *Int J Oral Maxillofac Surg* 33:369-376, 2004.
56. Lu M, Rabie AB: Macroarchitecture of rabbit mandibular defects grafted with intramembranous or endochondral bone shown by micro-computed tomography, *Br J Oral Maxillofac Surg* 41:385-391, 2003.
57. Ozaki W, Buchman SR: Volume maintenance of onlay bone grafts in the craniofacial skeleton: microarchitecture versus embryologic origin, *Plast Reconstr Surg* 102:291-299, 1998.
58. Glowacki J: In discussion of Kusiak JF, Zins JE, Whitaker LA: The early revascularization of membranous bone, *Plast Reconstr Surg* 76:515, 1985.
59. Koole R, Bosker H, Noorman van der Dussen F: Secondary autogenous bone grafting in cleft patients comparing mandibular (ectomesenchymal) and iliac crest (mesenchymal) grafts, *J Cranio Max Fac Surg* 17:28-30, 1989.
60. Rosenthal AH, Buchman SR: Volume maintenance of inlay bone grafts in the craniofacial skeleton, *Plast Reconstr Surg* 112:802-811, 2003.
61. Sharawy M, El-Shazly D, Um IW et al: Fate of allogeneic cranial bone grafted to rabbit mandible (abstract 1015), *J Dent Res* 75: 1996.
62. Takikawa S, Bauer TW, Kambic H et al: Comparative evaluation of the osteoinductivity of two formulations of human demineralized bone matrix, *J Biomed Mater Res A* 65:37-42, 2003.
63. Martin GJ, Boden SD, Titus L et al: New formulations of demineralized bone matrix as a more effective bone graft alternative in experimental posterior lateral lumbar spine arthrodesis, *Spine* 24:637, 1999.
64. Finkleman RD, Eason AL, Rakijian DR et al: Elevated IGF-II and TGF-B concentrations in human calvarial bone: potential mechanism for increased graft survival and resistance to osteoporosis, *Plast Reconstr Surg* 93:732-738, 1994.
65. Hardesty RA, Marsh JL: Craniofacial onlay bone grafting: a prospective evaluation of graft morphology, orientation and embryonic origin, *Plast Reconstr Surg* 85:5-14, 1990.
66. Manson PN: Facial bone healing and bone grafts: a review of clinical physiology, *Clin Plast Surg* 21:331-348, 1994.
67. Alberius P, Gordh M, Linberg L et al: Influence of surrounding soft tissues on onlay bone graft incorporation, *Oral Surg Oral Med Oral Pathol* 82:22-33, 1996.
68. Lew D, Marino AA, Startzell JM et al: A comparative study of osseointegration of titanium implants in corticocancellous block and corticocancellous chip grafts in canine ilium, *J Oral Maxillofac Surg* 52:952-958, 1994.
69. Wong RW, Rabie AB: A quantitative assessment of the healing of intramembranous and endochondral autogenous bone grafts, *Eur J Orthod* 21:119-126, 1999.
70. Buser D, Dula K, Belser UC et al: Localized ridge augmentation using guided bone regeneration. II. Surgical procedure in the mandible, *Int J Periodont Rest Dent* 15:11-29, 1995.
71. Roberts WE, Garetto LP, DeCastro RA: Remodeling of devitalized bone threatens periosteal margin integrity of endosseous titanium implants with threaded or smooth surfaces: indications for provisional loading

WWW.HIGHDENT.IR

همیار دندانسازان

and axially directed occlusion, *J Ind Dent Assoc* 68:19-24, 1989.
72. Schenk RK: Bone regeneration: biologic basis. In Buser D, Dahlin C, Schenk RK, editors: *Guided bone regeneration in implant dentistry,* Chicago, 1994, Quintessence.
73. Buser D, Dula K, Hirt HP et al: Lateral ridge augmentation using autografts and barrier membranes: a clinical study with 40 partially edentulous patients, *J Oral Maxillofac Surg* 54:420-432, 1996.
74. Misch CE: Density of bone: effect on treatment plans, surgical approach, healing and progressive bone loading, *Int J Oral Implantol* 6:23-31, 1990.
75. Sennerby L, Thomsen P, Ericson LE: A morphometric and biomechanic comparison of titanium implants inserted in rabbit cortical and cancellous bone, *Int J Oral Maxillofac Implants* 7:62-71, 1992.
76. Bidez M, Misch CE: Issues in bone mechanics related to oral implants, *Implant Dent* 1:289-294, 1992.
77. Collins TA: Onlay bone grafting in combination with Brånemark implants, *Oral Maxillofac Surg Clin North Am* 3:893-902, 1991.
78. ten Bruggenkate CM, Kraaijenhagen HA, van der Kwast WAM et al: Autogenous maxillary bone grafts in conjunction with placement of I.T.I. endosseous implants: a preliminary report, *Int J Oral Maxillofac Surg* 21:81-84, 1992.
79. Rosenquist JB, Nystrom E: Occlusion of the incisal canal with bone chips, *Int J Oral Maxillfac Surg* 21:210-211, 1992.
80. Bahat O, Fontanesi RV, Preston J: Reconstruction of the hard and soft tissues for optimal placement of osseointegrated implants, *Int J Periodont Rest Dent* 13:255-275, 1993.
81. Collins TA, Nunn W: Autogenous veneer grafting for improved esthetics with dental implants, *Compend Contin Educ Dent* 15:370-376, 1994.
82. Becker W, Becker BE, Polizzi G et al: Autogenous bone grafting of bone defects adjacent to implants placed into immediate extraction sockets in patients: a prospective study, *Int J Oral Maxillofac Implants* 9:389-396, 1994.
83. Becker W, Becker BE, Caffesse R: A comparison of demineralized freeze-dried bone and autologous bone to induce bone formation in human extraction sockets, *J Periodontol* 65:1128-1133, 1994.
84. Raghoebar GM, Batenburg RHK, Vissink A et al: Augmentation of localized defects of the anterior maxillary ridge with autogenous bone before insertion of implants, *J Oral Maxillofac Surg* 54:1180-1185, 1996.
85. Friberg B: Bone augmentation at single-tooth implants using mandibular grafts: a one-stage surgical procedure, *Int J Periodont Rest Dent* 15:437-445, 1995.
86. Lustmann J, Lewinstein I: Interpositional bone grafting technique to widen narrow maxillary ridge, *Int J Oral Maxillofac Implants* 10:568-577, 1995.
87. Lazzara R: Transplantation of a preosseointegrated implant from the mental area to a maxillary sinus graft, *Int J Periodont Rest Dent* 15:539-547, 1995.
88. Williamson RA: Rehabilitation of the resorbed maxilla and mandible using autogenous bone grafts and osseointegrated implants, *Int J Oral Maxillofac Implants* 11:476-488, 1996.
89. Triplett RG, Schow S: Autologous bone grafts and endosseous implants: complementary techniques, *J Oral Maxillofac Surg* 54:486-494, 1996.
90. Boyne PJ: Bone grafts: materials. In Boyne PJ, editor: *Osseous reconstruction of the maxilla and the mandible: surgical techniques using titanium mesh and bone mineral,* Chicago, 1996, Quintessence.
91. von Arx T, Hardt N, Wallkamm B: The TIME technique: a new method for localized alveolar ridge augmentation prior to placement of dental implants, *Int J Oral Maxillofac Implants* 11:387-394, 1996.
92. Chiapasco M, Zaniboni M, Rimondini L: Autogenous onlay bone grafts vs. alveolar distraction osteogenesis for the correction of vertically deficient edentulous ridges: a 2-4 year prospective study on humans, *Clin Oral Implant Res* 18:432-440, 2007.
93. Garber DA, Belser UC: Restoration-driven implant placement with restoration-generated site development, *Compend Contin Educ Dent* 16:796-804, 1995.
94. Mecall RA, Rosenfeld AL: The influence of residual ridge resorption patterns on fixture placement and tooth position in the partially edentulous patient. Part III. Presurgical assessment of ridge augmentation requirements, *Int J Periodont Rest Dent* 16:323-337, 1996.
95. Rosenfeld AL, Mecall RA: The use of interactive computed tomography to predict the esthetic and functional demands of implant-supported prostheses, *Compend Contin Educ Dent* 17:1125-1144, 1996.
96. Sarment DP, Misch CE: Scannographic templates for novel preimplant planning methods, *Int Mag Oral Implant* 4:16-22, 2002.
97. Albrektsson T: Repair of bone grafts: a vital microscopic and histologic investigation in the rabbit, *Scand J Plast Reconstr Surg* 14:1-12, 1980.
98. Whitaker LA: Biological boundaries: a concept in facial skeletal restructuring, *Clin Plast Surg* 16:1-10, 1989.
99. Nyman S, Lang NP, Buser D et al: Bone regeneration adjacent to titanium dental implants using guided tissue regeneration. A report of two cases, *Int J Oral Maxillofac Implants* 5:9-14, 1990.
100. Park HD, Min CK, Kwak HH et al: Topography of the outer mandibular symphyseal region with reference to the autogenous bone graft, *Int J Oral Maxillofac Surg* 33:781-785, 2004.
101. Montazem A, Valauri DV, St-Hilaire H et al: The mandibular symphysis as a donor site in maxillofacial bone grafting: a qualitative anatomic study, *J Oral Maxillofac Surg* 58:1368-1371, 2000.
102. Hirsch JM, Ericsson I: Maxillary sinus augmentation using mandibular bone grafts and simultaneous installation of implants: a surgical technique, *Clin Oral Implant Res* 2: 91-96, 1991.
103. Raghoebar GM, Brouwer TJ, Reintsema H et al: Augmentation of the maxillary sinus floor with autogenous bone for the placement of endosseous implants: a preliminary report, *J Oral Maxillofac Surg* 51:1198-1203, 1993.
104. Lundgren S, Moy P, Johansson C et al: Augmentation of the maxillary sinus floor with particulated mandible: a histologic and histomorphometric study, *Int J Oral Maxillofac Implants* 11:760-766, 1996.
105. Buhr W, Coulon JP: Limits of the mandibular symphysis as a donor site for bone grafts in early secondary cleft palate osteoplasty, *Int J Oral Maxillofac Surg* 25:389-393, 1996.
106. Cranin AN, Katzap M, Demirdjan E et al: Autogenous bone ridge augmentation using the mandibular symphysis as a donor, *J Oral Implantol* 27:43-47, 2001.
107. Pikos MA: Block autografts for localized ridge augmentation: part II. The posterior mandible, *Implant Dent* 9: 67-75, 2000.

WWW.HIGHDENT.IR

108. Cutright B, Quillopa N, Schubert W: An anthropometric study of the key foramina for maxillofacial surgery, *J Oral Maxillofac Surg* 61:354-357, 2003.
109. Gapski R, Wang HL, Misch CE: Management of incision design in symphysis graft procedures: a review of the literature, *J Oral Implant* 26:13243-13142, 2001.
110. Kaufman E, Wang PD: Localized vertical maxillary ridge augmentation using symphyseal bone cores: a technique and case report, *Int J Oral Maxillofac Implants* 18:293-298, 2003.
111. Papay FA, Morales L, Ahmed OF et al: Comparison of ossification of demineralized bone, hydroxylapatite, gelfoam and bone wax in cranial defect repair, *J Craniofac Surg* 7:347-351, 1996.
112. Rajchel J, Ellis E, Fonseca RJ: The anatomical location of the mandibular canal: its relationship to the sagittal ramus osteotomy, *Int J Adult Orthod Orthognath Surg* 1:37, 1986.
113. Smith BR, Rajchel JL: II. Anatomic considerations in mandibular ramus osteotomies. In Bell WH, editor: *Modern practice in orthognathic and reconstructive surgery,* Philadelphia, 1992, WB Saunders.
114. Hall HD: Intraoral surgery. In Bell WH, editor: *Modern practice in orthognathic and reconstructive surgery,* Philadelphia, 1992, WB Saunders.
115. Gungormus M, Yavuz MS: The ascending ramus of the mandible as a donor site in maxillofacial bone grafting, *J Oral Maxillofac Surg* 60:1316-1318, 2002.
116. Wood RM, Moore DL: Grafting of the maxillary sinus with intraorally harvested autogenous bone prior to implant placement, *Int J Oral Maxillofac Implants* 3:209-214, 1988.
117. Wheeler S, Holmes RE, Calhoun CJ: Six-year clinical and histologic study of sinus-lift grafts, *Int J Oral Maxillofac Implants* 11:26-34, 1996.
118. Berry RL, Edwards RC, Paxton MC: Nasal augmentation using the mandibular coronoid as an autogenous graft: report of case, *J Oral Maxillofac Surg* 52:633-638, 1994.
119. McLoughlin PM, Hopper C, Bowley NB: Hyperplasia of the mandibular coronoid process: an analysis of 31 cases and a review of the literature, *J Oral Maxillofac Surg* 53:250-255, 1995.
120. Koury ME: Complications of mandibular fractures. In Kaban LB, Pogrel MA, Perrott DH, editors: *Complications in oral and maxillofacial surgery,* Philadelphia, 1997, WB Saunders.
121. Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. Part I. Surgical procedures to correct mandibular prognathism and reshaping of the chin, *Oral Surg* 10:677, 1957.
122. DalPont G: Retromolar osteotomy for correction of prognathism, *J Oral Surg* 19:42, 1961.
123. Hunsuck EE: Modified intraoral splitting technique for correction of mandibular prognathism, *J Oral Surg* 26: 250, 1968.
124. Epker BN: Modifications in the sagittal osteotomy of the mandible, *J Oral Surg* 35:157, 1977.
125. Bell WH, Schendel SA: Biologic basis for modification of the sagittal ramus split operation, *J Oral Surg* 34:362, 1977.
126. Heggie AAC: The use of mandibular buccal cortical grafts in bimaxillary surgery, *J Oral Maxillofac Surg* 51:1282-1283, 1993.
127. Jensen J, Reiche-Fischel O, Sindet-Pedersen S: Autogenous mandibular bone grafts for molar augmentation, *J Oral Maxillofac Surg* 53:88-90, 1995.
128. Marx RE, Snyder RM, Kline SN: Cellular survival of human marrow during placement of marrow cancellous bone grafts, *J Oral Surg* 37:712-718, 1979.
129. Steiner M, Ramp WK: Short-term storage of freshly harvested bone, *J Oral Maxillofac Surg* 46:868-871, 1988.
130. Thompson N, Casson JA: Experimental onlay bone grafts to the jaws: a preliminary study in dogs, *Plast Reconstr Surg* 46:341-349, 1970.
131. Knize D: The influence of periosteum and calcitonin on onlay bone graft survival, *Plast Reconstr Surg* 53:190-199, 1974.
132. Fonseca RJ, Clark PJ, Burkes EJ et al: Revascularization and healing of onlay particulate autologous bone grafts in primates, *J Oral Surg* 38:572-577, 1980.
133. Dado DV, Izquierdo R: Absorption of onlay bone grafts in immature rabbits: membranous versus endochondral bone and bone struts versus paste, *Ann Plast Surg* 23: 39-48, 1989.
134. Canalis E, McCarthy TL, Centrella M: Effects of platelet-derived growth factor on bone formation in vitro, *J Cell Physiol* 140:530, 1989.
135. Peirce GF, Mustoe TA, Altrock BW et al: Role of platelet derived growth factor in wound healing, *J Cell Biochem* 45:319, 1991.
136. Lynch SE, Ruiz de Castilla G, William RC et al: The effects of short term application of a combination of platelet-derived and insulin-like growth factors on periodontal wound healing, *J Periodontol* 62:458-467, 1991.
137. Lynch SE, Buser D, Hernandez RA et al: Effects of the platelet-derived growth factor/insulin-like growth factor-I combination on bone regeneration around titanium dental implants. Results of a pilot study in beagle dogs, *J Periodontol* 62:710-716, 1991.
138. Strauss JE, Marx RE, Carlson ER: Platelet rich plasma: a source of growth factors for bony reconstruction of the jaws, *J Oral Maxillofac Surg* 54(suppl 3):37, 1996.
139. Rudkin GH, Miller TA: Growth factors in surgery, *Plast Reconstr Surg* 97:469-476, 1996.
140. Hollinger J, Wong ME: The integrated processes of hard tissue regeneration with special emphasis on fracture healing, *Oral Surg Oral Med Oral Pathol* 82:594-606, 1996.
141. Jensen OT: Guided bone graft augmentation. In Buser D, Dahlin C, Schenk RK, editors: *Guided bone regeneration in implant dentistry,* Chicago, 1994, Quintessence.
142. Jensen OT, Greer RO, Johnson L et al: Vertical guided bone-graft augmentation in a new canine mandibular model, *Int J Oral Maxillofac Implants* 10:335-344, 1995.
143. Tolman DE: Reconstructive procedures with endosseous implants in grafted bone: a review of the literature, *Int J Oral Maxillofac Implants* 10:275-294, 1995.
144. Pikos MA: Atrophic posterior mandible reconstruction utilizing mandibular block autografts, risk management, *Int J Oral Maxillofac Implants* 18:765-766, 2003.
145. Jones JK, Triplett RG: The relationship of smoking to impaired intraoral wound healing, *J Oral Maxillofac Surg* 50:237-239, 1992.
146. Misch CM: The extracted tooth pontic-provisional replacement during bone graft and implant healing, *Pract Periodont Aesth Dent* 10:711-718, 1998.
147. Schnitman PA, Wohrle PS, Rubenstein JE: Immediate fixed interim prostheses supported by two-stage threaded implants. Methodology and results, *J Oral Implantol* 16:96-105, 1990.
148. Salama H, Rose LF, Salama M et al: Immediate loading of bilaterally splinted titanium root-form implants in

WWW.HIGHDENT.IR

همیار دندانسازان

fixed prosthodontics—a technique reexamined: two case reports, *Int J Periodont Rest Dent* 15:345-361, 1995.
149. Rubens BC, West RA: Ptosis of the chin and lip incompetence: consequences of lost mentalis muscle support, *J Oral Maxillofac Surg* 4:359-366, 1989.
150. Silva FM, Cortez AL, Moreira RW et al: Complications of intraoral donor site for bone grafting prior to implant placement. *Implant Dent* 15:420-426, 2006.
151. Hoppenreijs TJM, Nijdam ES, Freihofer HPM: The chin as a donor site in early secondary osteoplasty: a retrospective clinical and radiological evaluation, *J Craniomaxillofac Surg* 20:199-124, 1992.
152. Joshi A: An investigation of post-operative morbidity following chin graft surgery, *Br Dent J* 196:215-218, 2004.
153. von Arx T, Hafliger J, Chappuis V: Neurosensory disturbances following bone harvesting in the symphysis: a prospective clinical study, *Clin Oral Implants Res* 16:432-439, 2005.
154. Nkenke E, Schultz-Mosgau S, Radespiel-Troger M et al: Morbidity of harvesting of chin grafts: a prospective study, *Clin Oral Implants* 12:495-502, 2001.
155. Johnson JV, Hinds EC: Evaluation of teeth vitality after subapical osteotomy, *J Oral Surg* 27:256, 1969.
156. Clavero J, Lundgren S: Ramus or chin grafts for maxillary sinus inlay and local onlay augmentation: comparison of donor site morbidity and complications, *Clin Implant Dent Relat Res* 5:154-160, 2003.
157. Pepersack WJ: Tooth vitality after alveolar segmental osteotomy, *J Maxillofac Surg* 1:85-91, 1973.
158. Lindquist CC, Obeid G: Complications of genioplasty done alone or in combination with sagittal split-ramus osteotomy, *Oral Surg* 66:13-16, 1988.
159. Hutchinson D, Macgregor AJ: Tooth survival following various methods of subapical osteotomy, *Int J Oral Surg* 1:81-86, 1972.
160. Morrison AD: Prognosis of darkened teeth following orthognathic surgery, *J Oral Maxillofac Surg* 55(Suppl):82, 1997.
161. Hendy CW, Smith KG, Robinson PP: Surgical anatomy of the buccal nerve, *Br J Oral Maxillofac Surg* 34:457-460, 1996.
162. Nkenke E, Radespiel-Troger M, Wiltfang J et al: Morbidity of harvesting of retromolar bone grafts: a prospective study, *Clin Oral Implants Res* 13:513-521, 2002.
163. Datillo DJ, Misch CM, Arena S: Interface analysis of hydroxyapatite-coated implants in a human vascularized iliac bone graft, *Int J Oral Maxillofac Implants* 10:405-409, 1995.
164. Phillips JH, Rahn BA: Fixation effects on membranous and endochondral onlay bone graft revascularization and bone deposition, *Plast Reconstr Surg* 85:891-897, 1990.
165. Nystrom E, Kahnberg K-E, Albrektsson T: Treatment of the severely resorbed maxillae with bone graft and titanium implants: histologic review of autopsy specimens, *Int J Oral Maxillofac Implants* 8:167-172, 1993.
166. Buser D, Dula K, Belser U et al: Localized ridge augmentation using guided bone regeneration. I. Surgical procedure in the maxilla, *Int J Periodont Rest Dent* 13:29-45, 1993.
167. Simion M, Dahlin C, Trisi P et al: Qualitative and quantitative comparative study on different filling materials used in bone tissue regeneration: a controlled clinical study, *Int J Periodont Rest Dent* 14:199-215, 1994.
168. Simion M, Fontana F: Autogenous and xenogeneic bone grafts for the bone regeneration: a literature review, *Minerva Stomatol* 53:191-206, 2004.
169. Pansegrau KJ, Friedrich KL, Lew D et al: A comparative study of osseointegration of titanium implants in autogenous and freeze-dried bone grafts, *J Oral Maxillofac Surg* 56:1067-1073, 1998.
170. Roberts EW: Bone tissue interface, *J Dent Educ* 52:804-809, 1988.
171. Chen J, Chen K, Garetto LP et al: Mechanical response to functional and therapeutic loading of a retromolar endosseous implant used for orthodontic anchorage to mesially translate mandibular molars, *Implant Dent* 4:246-258, 1995.
172. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, *Postgrad Dent* 2: 3-16, 1995.
173. Hall MB: Marginal bone loss around Brånemark fixtures in bone grafts used for augmentation, *J Oral Maxillofac Surg* 48:117, 1990.
174. Ozkan Y, Ozcan M, Varol A et al: Resonance frequency analysis assessment of implant stability in labial onlay grafted posterior mandibles: a pilot clinical study, *Int J Oral Maxillofac Implants* 22:235-242, 2007.
175. Sjostrom M, Lundgren S, Milson H et al: Monitoring of implant stability in grafted bone using resonance frequency analysis. A clinical study from implant placement to 6 months of loading, *Int J Oral Maxillofac Surg* 34:45-51, 2005.
176. Sethi A, Kaus T: Ridge augmentation using mandibular block bone grafts: preliminary results of an ongoing prospective study, *Int J Oral Maxillofac Implants* 16:378-388, 2001.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 40

Extraoral Autogenous Donor Bone Grafts for Endosteal Implants: Ilium and Tibia

Carl E. Misch, Francine Misch-Dietsh, Miles L. Singer, Mathew Lyman

In ridges with abundant bone, all factors converge to provide an ideal long-term prognosis for the implant-supported prosthesis, including favorable ridge relationships. These conditions allow long and wide endosteal implants in adequate numbers, favorable crown/implant ratio, adequate bone density, and favorable orientation of occlusal forces toward the support system. Patients with these advantages usually have less-demanding needs than when unfavorable factors include advanced bone atrophy, as well as the dire need for prosthetic support and retention. When confronted with cases of extreme alveolar and basal bone resorption or discontinuities, most conventional treatment modalities fall short of the intended result. Instead, autogenous bone grafts from extraoral origin, in conjunction with implants, are often indicated to restore these patients.[1-16] In addition, patients with moderate resorptive conditions and ideal prosthetic goals may also require augmentation.

The usual goal of autogenous bone grafts is to provide sufficient available bone to permit the placement of endosteal implants in compromised maxillae and mandibles or to meet the prosthetic and/or esthetic needs and desires of the patient with moderate resorption. Autogenous grafts permit the placement of endosteal implants of increased surface area, height, width, number, and improved location. The type of final prosthesis can be upgraded to a fixed prosthesis with more natural appearance by enhancement of the soft tissue support and possible correction of ridge relationships. The placement of the implants in ideal locations improves stress distribution patterns, interarch relationships, and esthetics of the final prosthesis. As a result, benefits to the patient include implant longevity and improved prosthetics.

HISTORY

Throughout the evolution of oral implantology, several approaches have been proposed to address the problems of the severely resorbed jaw. The lack of support for traditional endosteal implant designs has led to the development of the subperiosteal implant, resting on the remaining bone of the ramus and symphysis in the mandible.[17-18] The endosteal ramus frame implant design also takes advantage of these areas.[19] In the maxilla, extension to the pterygoid plates, maxillary tuberosity, and lateral aspect of the zygomatic process are attempted with endosteal and subperiosteal implants.[20,21] However, the extremely atrophic conditions and the resultant poor biomechanics of the implant system have led to many complications (Figure 40-1). The violation of fundamental prosthodontic principles in the final restoration, as insufficient implant support

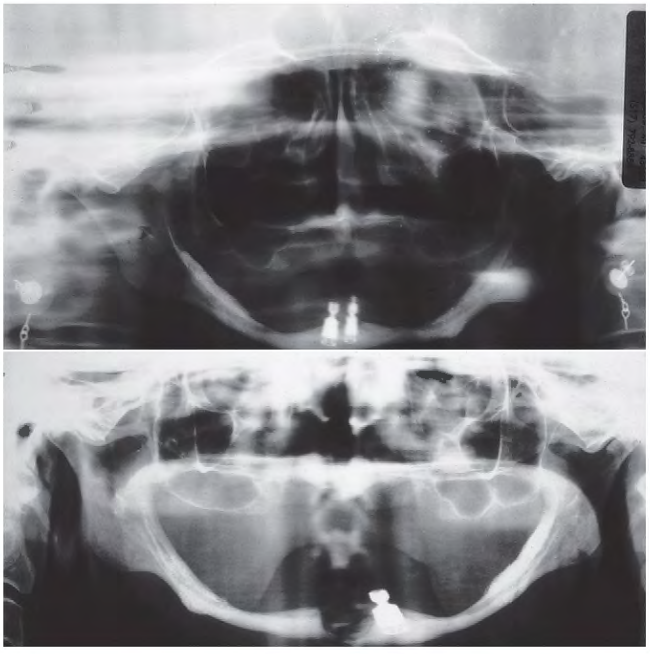


Figure 40-1 **A,** Advanced atrophy of the jaws provides reduced implant surface area, large crown heights, and poor soft tissue support. **B,** Implant failure in patients with advanced atrophy may lead to fracture of the mandible or oral antral and nasal fistulae in the maxilla.

WWW.HIGHDENT.IR

همیار دندانسازان

(in size, number, and location) and excessive number of pontics, has encouraged the development of more predictable procedures.

The concept of jaw reconstruction with varied autogenous and alloplastic materials has been used extensively for many years in craniofacial reconstruction.[22-25] However, jaw reconstruction with autogenous bone alone has been characterized by rapid, advanced bone resorption during the 3 to 5 years after the procedure.[26-29] In addition, augmentation of atrophic ridges for conventional complete dentures still does not return a patient to normal mastication and comfort (Figure 40-2). On the other hand, reports of implants in conjunction with autogenous grafts have been reported in the literature, with improved success in relation to prosthetic support and long-term maintenance of the graft. The implants stimulate and maintain the grafted bone and prevent the rapid bone resorption found with autogenous grafts for complete denture support.[1,16,30,31]

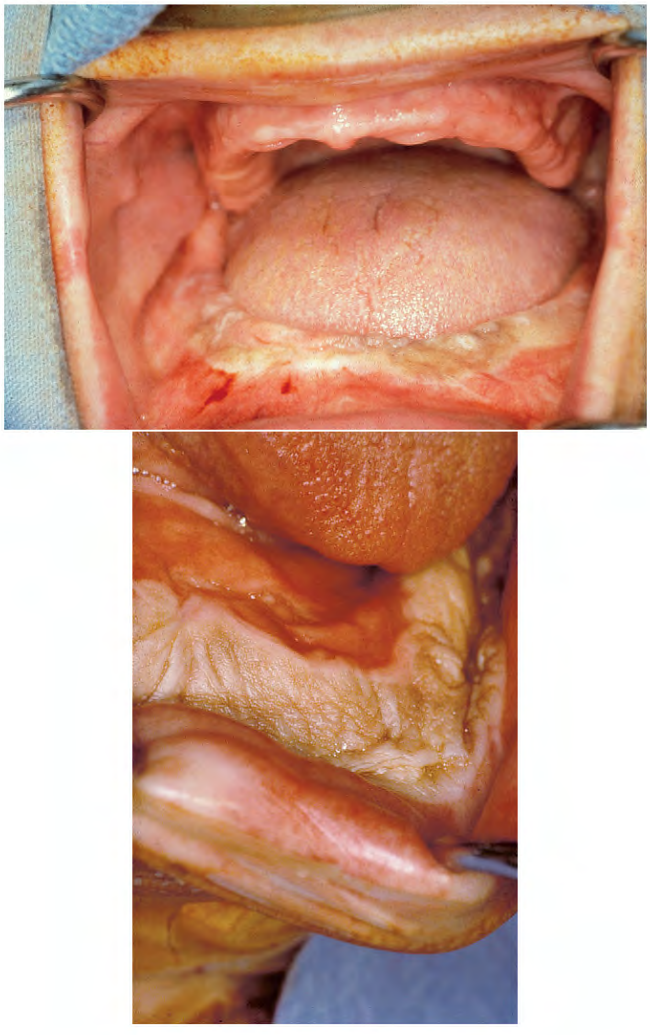


Figure 40-2 **A,** Iliac crest bone grafts with skin grafts to an edentulous mandible initially improve the support for a denture. **B,** The autograft resorbs rapidly over a 3- to 5-year period, leaving loose tissue and poorer denture support than the initial poor condition before the autograft.

When the placement of endosteal implants is desired and advantageous for prosthodontic support, it is beneficial to reconstruct the anatomy of the atrophic jaw with bone or a material that will resorb and be replaced by living bone. The posterior maxilla can be augmented predictably by sinus grafting to permit the placement of endosteal implants, using synthetic bone substitutes, combined with demineralized freeze-dried bone (DFDB) and intraorally harvested autogenous bone.[32,33] No other region of the mouth permits as predictable augmentation for endosteal implants in the severe atrophic state. The subantral (SA) area created for sinus grafts presents many features that are favorable for bone formation. The graft is surrounded by bony walls in a well-vascularized region and protected from loads during healing. Other areas of the mouth that require ridge augmentation more often require onlay autogenous bone grafts to predictably gain bone volume. As a consequence, in most regions of the mouth, the treatment of choice for moderate to severe atrophic conditions is reconstruction with autogenous bone grafting, followed by oral rehabilitation with implant-supported prostheses.

This chapter primarily addresses the iliac crest cortical and trabecular block grafts for onlay augmentation in completely edentulous patients, as well as tibia trabecular bone grafts for barrier membrane procedures. Smaller atrophic regions may be augmented with intraorally harvested bone. An extraoral origin for autogenous bone grafts is necessary for large atrophic regions of the jaws. The preferred extraoral donor sites of autogenous bone include the iliac crest, tibia, cranium, and, to a lesser degree, the rib and fibula.

Treatment Planning

Indications

The prosthetic goal and the patient's needs and desires are the primary determinants for implant-related treatment option selection. Demanding esthetics or functional desires and expectations of the patient may mandate the augmentation of the bony structures, even when other treatment alternatives are available. For example, a patient with moderate atrophy may be a candidate for an implant-supported overdenture. However, when the patient's needs and/or desires require a fixed prosthesis, bone grafting for additional implant number, size, and position may be required. Therefore, when any discrepancy of hard tissues exists that cannot render predictable implant prosthesis treatment, the implant dentist has three options:

1. Select a traditional prosthodontic rehabilitation without implants.
2. Modify the patient's mind by lowering expectations, and plan a prosthesis requiring less implant support.

WWW.HIGHDENT.IR

3. "Improve" the available bone to the amount and form needed to achieve the expected result.

Indications for autogenous grafts for the placement of endosteal implants are presented using the Misch-Judy available bone and Misch prosthetic options classifications.[33a,33b] In addition, bone Division E has been added to the original four divisions to encompass those conditions in which trauma, pathology, surgery, or genetics have resulted in a discontinuity defect of the jaw.

General Considerations

Autogenous bone grafts are osteogenic and able to form bone in the absence of undifferentiated mesenchymal cells. In addition, the autograft may form bone by osteoinduction, osteoconduction, and a barrier membrane approach. In addition, it appears that no age-related change exists in the viability of bone-derived cells.[34] Fresh, autogenous, cancellous bone provides the survival of the maximum number of transplanted bone and pluripotential cells of the marrow.[23] For large grafts, it is usually harvested from the ilium, which presents a high population of pluripotential cells and can deliver the greatest amount of trabecular bone.[35-37] Proponents of the use of solely trabecular bone argue that when harvested from iliac crest, it allows implant placement earlier.[38] Fonseca and Davis[37] recommended the addition of chips from the lateral cortex to increase the bone morphogenetic protein (BMP) concentration. The addition of platelet-rich plasma (PRP)[35,39,40] over the grafted bone attempts to further stimulate the second phase of bone formation and may enhance and stimulate remodeling and incorporation of the graft. However, trabecular grafts are usually maintained in place by the use of cribs.[36] More importantly, the resorption rate is faster than the bone formation rate, and the volume of bone obtained is highly variable. In addition, the quality of bone formed is often poorer compared with other graft options.

An advantage of a trabecular graft is the solid block form, which permits contouring and adaptation of the graft to the recipient bed anatomy.[41] The block graft maintains greater volume of bone compared with particulate onlay grafts, because resorption of the cortical bone is delayed and rigid fixation of the block to the atrophic ridge is possible. The cortical bone on the outside of the graft also acts like a barrier membrane similar to membranes used in guided bone regeneration (GBR).[42] The fibrous tissue is prevented from invading the graft site, and this provides additional time for angiogenesis and new bone formation below the cortical layer. The rigidity and stability of the graft can be achieved by the use of fixation screws to stabilize the graft, eliminating the need for wiring, pins, or the use of a crib.[2,3] The quality of the bone is greater and increases strength, modulus of elasticity, and initial bone-implant contact percent (BIC). A review of the literature of bone grafts and the placement of implants reported similar success rates for block and particulate grafts to the maxilla and mandible,[6] and both procedures are widely reported.

ILIAC CREST CORTICAL AND TRABECULAR BLOCK GRAFTS

According to several publications, autogenous bone harvested from the ilium is the donor site of choice for large-defect bone grafts to the jaws.[1-16,36,43-53] The primary advantage of the iliac crest graft is its large volume. The outer portion of the graft may be primarily cortical, with abundant trabecular bone underneath. The volume of bone harvested permits shaping of two thirds of a mandible or maxilla or filling of larger bony voids.

Relative ease of access and harvesting from the ilium have made it a safe and well-accepted procedure. However, rapid bone resorption of 30% to 90% of the iliac crest bone grafts has been reported when conventional dentures are placed on top of the reconstruction.[29] As a consequence, other treatment options have often been considered, such as zygomatic and pterygoid implants in the maxilla or 7- to 9-mm implants in an anterior mandible. However, the placement of implants into the grafted bone has dramatically modified this resorption rate. Once the implants are placed and in function, the rate of bone resorption is similar to host bone of similar quantity and density in similar conditions.[5,53-55]

In 1972, Kratochvil and Boyne[30] originally reported the iliac crest bone graft procedure with the concomitant use of implants with subperiosteal implants (Figure 40-3). In the past decades, an increasing number of reports have discussed concomitant use of grafted bone from the iliac crest with endosteal root form implants placed simultaneously or in a second step.[1-16,53-75] However, the reported success rates of autogenous bone grafts and dental implants are variable (Table 40-1).

In the early years of reports, implant failure was higher in grafted bone than native host bone. Breine and Brånemark[3] placed implants in 18 atrophic jaws in conjunction with autogenous trabecular bone. Only 25% of the 129 implants remained integrated in 14 atrophic maxillae and four mandibles. Most jaws required additional implants, and 20% of the maxillary jaws never achieved permanent bridge stability. Additional implants placed after graft maturity had 73% survival. In 1987, Keller et al.[58] reported on five patients with 28 implants and bone grafting, with four failing to fixate (85% survival) and four patients with 76% survival (16 of 21 implants) when placed after Le Fort I osteotomy and grafts. They later reported on these patients and 20 additional grafted maxillae after a 2-year evaluation, with an overall implant survival

WWW.HIGHDENT.IR

همیار دندانسازان

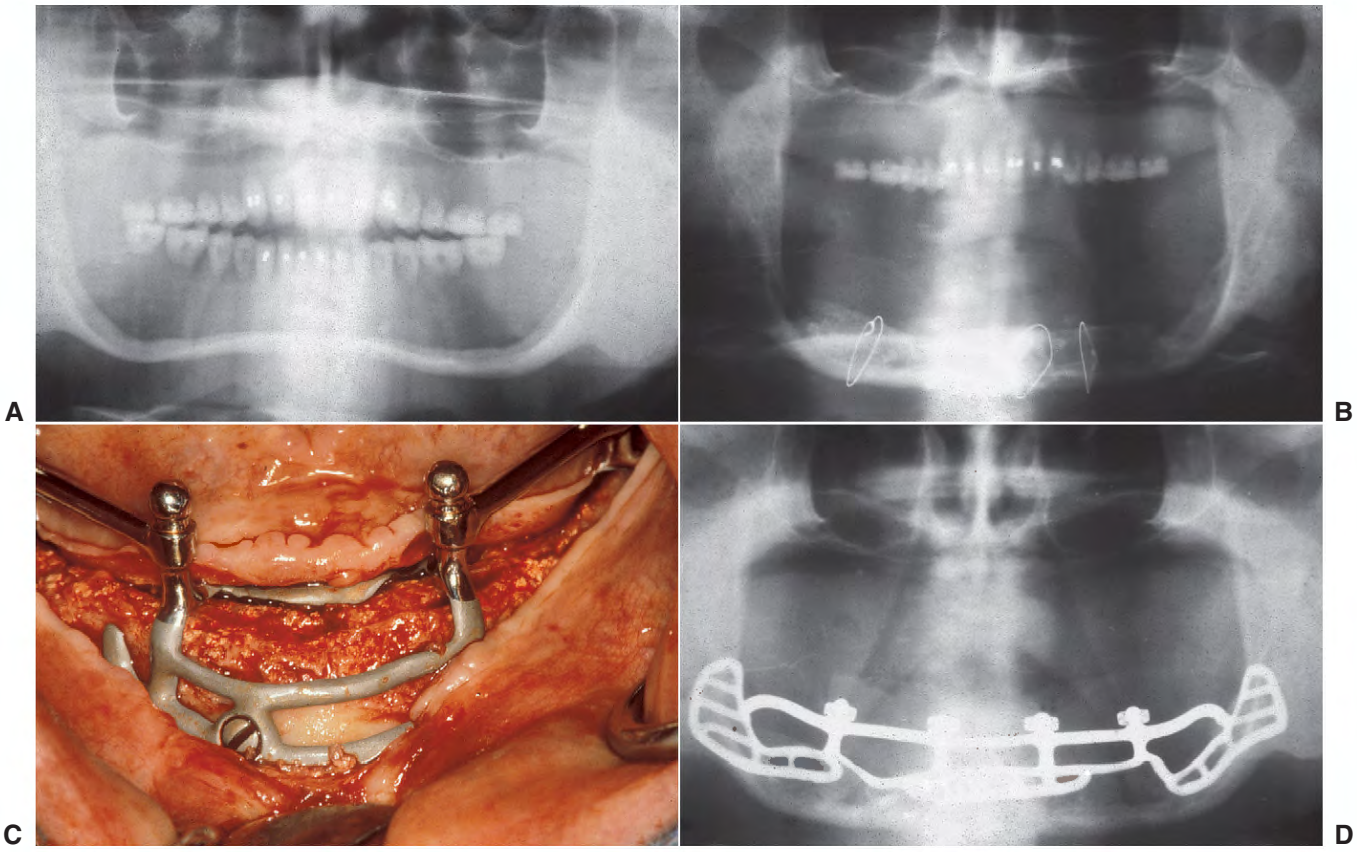


Figure 40-3 **A,** A panoramic radiograph of a severely resorbed mandible, measuring less than 3 mm in height. **B,** A postoperative panoramic radiograph of an iliac crest bone graft wired around the atrophic mandible (1983). **C,** Reentry after 6 months into the iliac crest bone graft and a subperiosteal implant inserted (1983). **D,** Postoperative panoramic radiograph of the iliac crest graft after maturity and the subperiosteal implant (1987).

Table 40-1 Iliac Crest Grafts and Endosteal Implants

STUDY	NUMBER OF ARCHES	IMMEDIATE IMPLANTS (% SURVIVAL)	DELAYED IMPLANTS (% SURVIVAL)
Breine and Brånemark[3] (1980)	18	25	73
Breine and Brånemark[3] (1980)	9	54.6	60
Keller et al.[58] (1987)	9	85	76
Listrom and Symington[44] (1988)	22	85	77
Kahnberg et al.[60] (1989)	10	85	–
Hall[53] (1990)	10	–	100
Adell et al.[57] (1990)	23	75.3	31
Jensen et al.[56] (1990)	4	–	41
Keller and Tolman[59] (1992)	7	87.5	–
Misch[5] (1994)	20	90	99
Nystrom et al.[62] (1996)	20	88.3	–
Triplett et al.[64] (1996)	99	83.6	90.4
Neyt et al.[65] (1997)	17	–	92.7
Schliephake et al.[66] (1997)	137	67.8	–
Stellingsma et al.[15] (1998)	10	–	100
Misch[55] (1999)	97	95.6	99.08
Umstadt et al.[14] (1999)	40	87	66
Raghoebar et al.[12] (2003)	10	95.6	–
Van der Meij et al.[13] (2005)	17	88.2	–
Verhoeven et al.[8] (2006)	13	100	–
Clayman[9] (2006)	8	–	83
Sjostrom et al.[10] (2007)	25	–	90

WWW.HIGHDENT.IR

of 77.4%. Keller and Tolman[59] reported on seven mandibles with iliac crest grafts and 32 simultaneous implant placements, resulting in 87.5% survival rate. In 1988, Listrom and Symington[44] inserted implants in 12 grafted arches, with 85% initial implant survival, as well as a secondary procedure in 10 patients with 43 implants and 77% implant survival. Kahnberg et al.[60] reported on 10 patients in 1989 having received 57 implants simultaneously, with a graft from the iliac crest and eight implants lost (85% survival). Complications included graft exposure in three patients (30%). The use of a splint to protect the graft was reported to significantly decrease this complication.

Results by the same authors were updated in 1995, with 20 arches grafted with simultaneous implant placement and an 88.3% survival rate.[54] In 1997, Schliephake et al.[66] reported on the 5-year follow-up of 137 grafted atrophic maxillae and a cumulative survival rate of 67.8% for implants placed at the time of grafting.

In 1990, Jensen et al.[56] also reported problems with iliac bone grafts and stated the procedure should still be considered in an evaluation stage. Twenty-nine implants were placed in four patients who received iliac crest grafts. Twelve remained fixated and in function, or 41% implant survival. In a study of 23 consecutive grafted maxillae over a 10-year period, Adell et al.[57] reported on 124 fixtures placed simultaneously with the grafts and 16 implants added in a second stage. The survival rate of fixtures placed with the graft was 75.3% at 4 years after grafting. However, 16 implants placed at a secondary procedure resulted in nine implant failures (31% implant survival).

On the other hand, several reports in the literature indicate implant survival in grafted bone is similar to native bone. Hall[53] reported 100% implant success after graft maturity in 10 patients and also reported bone loss similar to nongrafted bone sites where implants were inserted. Tidwell et al.[63] reported on combined sinus and iliac onlay grafts in 48 maxillary patients and found 6.4% implant failure in the sinus and 7.8% implant failure in the anterior region. In 1996, Triplett et al.[64] reported on 129 grafts in 99 patients (including ilium, cranium, and symphyseal grafts). Survival rates for the implants placed at the time of grafting were 83.6% and 90.4% for implants placed in a delayed approach. In 2003, Raghoebar et al.[12] reported on the early loading of 68 implants inserted in a single-stage approach, with iliac crest grafts to 10 maxillae and a 95.6% survival rate after 1 year.

The ideal timing for implant placement in autogenous bone grafts from extraoral origin remains a controversial issue. In 1997, Neyt et al.[65] reported on 17 atrophic maxillae restored with sinus and iliac crest grafts and implants and an overall 92.7% survival rate. All implants were placed in a delayed fashion. In 1999, Umstadt et al.[14] performed 40 iliac crest grafts in a prospective study of 176 implants and reported 87% and 66% survival rate for implants placed in a immediate and delayed fashion, respectively. In 1998, Stellingsma et al.[15] reported 100% success for 40 implants placed in a delayed fashion in 10 augmented mandibles of female patients.

In 2005, Van der Meij et al.[13] also selected an immediate implant placement approach for 17 atrophic mandibles augmented with the iliac crest and restored with two implant overdentures (34 implants). The average length of the study was 4.3 years, with an 88.2% implant success rate. In 2006, Verhoeven et al.[8] reported 100% implant survival rate when placed at the same time as the bone graft in 13 atrophic mandibles, after 10 years. However, 51% of the bone graft had resorbed, mostly during the first year after the bone graft. In 2006, Clayman[9] reported on eight grafted maxillae with delayed placement of 34 implants, with an 83% success rate. Sjostrom et al.[10] elected to place all implants in a delayed fashion. They grafted 25 maxillae that were restored with 192 implants. At the 3-year follow-up, 90% were successful with stable bone levels.

In conclusion, recommendations in the literature range from immediate placement to delays of 4 to 12 months postoperatively. Requirements for the immediate-placement success include the anchorage of immediate implants in native bone, with a minimum of 1.5-mm thickness of graft bone covering the implant. Several other reports underline that implants anchored in graft alone fail.[68-70] Graft maturation is the primary advantage of delayed implant placement, along with the possibility to address eventual postoperative complications and a more precise placement of the implants for the intended prosthesis. Timing of implant placement should, however, attempt to take advantage of the bone-preserving stimulus.[44,71-73] It is also suggested that the implant and graft success may be different whether a maxilla or mandible is the host site.

A retrospective case series study was published in 1994 by Misch and updated in 1999.[55] This report is further updated in this chapter. Patients received iliac crest block grafts for implant-supported prostheses from 1984 to 2005 and were monitored until 2007 (Table 40-2). A total of 182 patients and 221 arches received autogenous bone grafts from the iliac crest to the maxilla (133) and mandible (88). Participants included 146 women and 36 men in the study group. Thirty-three women and six men received grafts in

Table 40-2 Illiac Crest Grafts (1984 to 2005)

	PATIENTS	ARCHES
Male patients	36	42
Female patients	146	179
TOTAL	182	221

WWW.HIGHDENT.IR

همیار دندانسازان

both the maxilla and the mandible. Six arches in the maxilla were lost to recall and dropped (two patients died before treatment was completed, three patients elected not to continue treatment for economic and personal reasons, and one patient developed an uncontrolled disease and elected not to continue treatment [not reentered]). One arch in the mandible was dropped because the patient elected not to continue treatment for economic reasons (same patient as maxilla). These seven arches were dropped from the study.

A total of four arches failed (all female patients and all maxillae). Therefore a total of 210 arches were restored (123 maxillae, 87 mandibles), with a total success rate of the graft of 95% (92.5% and 98.9% for maxillae and mandibles, respectively) (Table 40-3).

Ninety-one implants were placed to stabilize the block graft, of which 44 were in the mandible and 64 in the maxilla (Table 40-4). Six months later, the implants were evaluated by direct observation for rigid fixation and quantity of bone around the implant. Three implants were mobile, and three other implants exhibited rigid fixation; however, two had poor placement, and one had more than one third of crestal bone loss. Therefore six implants were removed (five of the six were in the maxilla), with 97.7% of mandibular immediate implants placed maintained, whereas 92.2% of maxillary immediate implants were maintained (overall survival 94.4%). After 1993, no implants were placed concomitantly with the grafts to fixate the graft. In some selected cases, however, some were placed in the tuberosity or vomer to support the transitional prosthesis.

Five to 8 months after the autogenous bone graft, 1256 endosteal implants were placed in a delayed fashion. The maxillae received 876 implants, and the mandibles received 380 endosteal implants. Thirty-seven implants did not achieve rigid fixation and were removed at the stage II (uncovery) surgery. Two additional implants were lost after 2 years in function in the posterior maxilla. The survival of the implants placed in a delayed fashion in autogenous bone graft sites was 96.9% in the maxilla and 96.8% in the mandible, with an overall survival rate of 96.9% (see Table 40-2).

In conclusion, a total of 1364 (940 maxillary and 424 mandibular) implants were placed in either an immediate or delayed fashion, with an overall survival rate of 96.7% (96.6% and 96.9% for maxilla and mandible, respectively) and higher survival rates for delayed-placement implants.

The causes of the 45 implant losses included poor implant placement at the time of grafting, parafunction and trauma from the overlying removable prosthesis opposing natural teeth during healing, incision line opening after the autogenous graft (two), and one late failure caused by advanced uncontrolled bone loss. No "sleepers" were counted in this study. Sleepers in the maxilla were used primarily in the tuberosity to help support and retain the denture and protect the bone graft until implant uncovery. The patients in this report have been in function with their final restoration from as long as 238 months (19 years, 9 months) to as little as 12 months, with more than half of the arches in function for more than 12 years. The amount of crestal bone loss observed during the time frame of this study on implants placed after graft maturity after the first year of loading is similar to that for implants placed in full arches that did not receive a graft.

Several authors have advocated the placement of implants after graft bone maturation for 5 to 10 months. A delayed implant placement after graft maturity of 5 to 10 months is also strongly encouraged for several reasons. Incision line opening is a common compli-

Table 40-3 Iliac Crest Graft and Endosteal Implants (1984 to 2005)

	TREATMENT	FAILED	DROPPED	RESTORED (% SUCCESS)
Maxilla	133	4*	6†	123 (92.5)
Mandible	88	—	1‡	87 (98.9)
TOTAL	221	4	7	210 (95.0)

*Four failed (all female patients).
†Six dropped: two deceased, three lost to recall or dropped, one uncontrolled disease.
‡One dropped: local to recall.

Table 40-4 Iliac Crest Grafts with Endosteal Implant-Supported Prostheses (1984 to 2005)

	Placed			Lost			Survival %		
	TOTAL	MAXILLA	MANDIBLE	TOTAL	MAXILLA	MANDIBLE	TOTAL	MAXILLA	MANDIBLE
Immediate	108	64	44	6	5	1	94.4	95.3	97.7
Delayed	1256	876	380	39	27	12	96.9	96.9	96.8
TOTAL	1364	940	424	45	32	13	96.7	96.6	96.9

WWW.HIGHDENT.IR

cation with larger bone grafts. Implants exposed along with the surrounding graft are more likely to fail or exhibit crestal bone loss. In addition, because the first 5 to 10 months are a primary time of consolidation and/or remodeling of the graft and modeling of new bone, the contours and volumes of the bone block exhibit the greatest changes.[61,74,75] Functional loading of the implants allows this resorption to fall to the value of physiologic involution. The delayed approach permits the placement of implants in optimized bone volume, and it even allows regrafting in regions compromised after initial graft healing. Prosthetic and surgical templates may be used with greater accuracy for delayed implant placement, with precise information on the bony topography for surgical template fabrication. As a result, implants are positioned more ideally for the intended prosthesis.

Procedure

Maxillary Bone Grafts

The iliac crest graft technique to the maxillae has evolved over the last 25 years. Different approaches such as onlay grafting or intrapositional graft with Le Fort I osteotomies have been proposed. Originally, implants were often placed at the same time as the bone graft and positioned anterior to the maxillary sinuses.[3,44,45,56,57] Because the maxilla resorbs to the medial and the mandible resorbs toward the facial as basal bone remains, implants placed at the time of the bone graft are too lingual in the maxilla and too facial in the mandible (Figure 40-4). Poor implant position, increased risk of implants in compromised areas of incision line opening, poor anteroposterior (A-P) implant distance, and less-controlled crestal bone loss during bone revascularization suggested several modifications of the technique.

The maxillary sinus bone graft is one of the most predictable regions of the mouth to form large volumes of bone, even when the graft site is primarily alloplasts and/or allografts. As a result, sinus grafts as part of the iliac crest bone graft approach have been strongly suggested by the authors. This permits the placement of more distal implants of large dimension and surface area for improved biomechanical distribution of stress and strain to the bone. However, one complication of simultaneous sinus and onlay grafts is an increased risk of postoperative infection within the first 2 months after sinus grafting, which occurs in approximately 3% of procedures. The infection compromises the sinus graft and may spread to the onlay portion of the graft. Although relatively low, this risk can be eliminated by performing the sinus graft with primarily alloplasts and allografts 3 to 4 months before the iliac crest graft. In this way, the sinus membrane and ostium complications can be addressed before the iliac crest graft. During the onlay block graft surgery, the inferior one third to one half of the sinus graft may be removed and replaced with autogenous bone. Because the sinus membrane is not manipulated, the risk of infection is minimized.

Another main surgical advantage of early sinus grafting is the opportunity to enhance the soft tissue quality and quantity in the anterior region for primary coverage of the onlay graft. A transitional augmentation may be performed in the premaxilla. A full-thickness incision is made 5 mm to the palatal aspect of the crest from canine to canine. A mucoperiosteal flap exposes the facial region of the ridge. A Vicryl-encapsulated dense hydroxyapatite (HA) (PermaRidge) or AlloDerm acellular tissue graft is placed in the premaxilla region. The mesh of dense HA or acellular tissue acts as a transitional augmentation during soft tissue healing. At the iliac crest graft surgery, the HA "sausage" is removed before positioning the iliac block of bone. The primary

Figure 40-4 **A,** The maxilla resorbs toward the palate in the anterior and posterior region. The mandible becomes wider because the basal bone is lateral to the alveolar bone. Implants secured into basal bone and positioned at the time of iliac crest graft placement are therefore too lingual in the maxilla and too facial in the mandible. **B,** Implants placed at the time of iliac crest augmentation are more facial than the position of the teeth. The overdenture bar is cantilevered to the lingual in the anterior and is in the vestibule of the lip.

WWW.HIGHDENT.IR

همیار دندانسازان

advantage is the improved tissue thickness available to allow primary closure over the graft without tension.

A third reason to perform a sinus graft 4 months before the iliac crest block bone graft is related to fixation. It is sometimes difficult to secure the onlay graft to the thin residual ridge, and primary graft stability is paramount to the success of the procedure. Wires around the nasal spine, piriform rim, and/or zygomatic arches may be required when this occurs. The advantage of sinus grafting several months before the onlay graft is the easier placement of fixation screws and the ability to fixate the graft in the anterior and posterior regions. This reduces the risk of block mobility, especially when opposing mandibular anterior teeth. A fourth reason to perform a sinus graft 4 months or more before the iliac crest graft is that the implant may be inserted at the time of the sinus graft into the maxillary tuberosity and palatal incline of the premaxilla vomer bone. At the iliac crest graft surgery, the implants may be uncovered and used as retentive devices for the transitional prosthesis while the block bone graft matures.

SURGICAL APPROACH: HARVEST OF THE ILIAC CREST TRICORTICAL BLOCK

Anterior Iliac Crest Graft

The iliac crest is widely accepted as the donor site of choice of bone for reconstruction of large defects of the maxillla and mandible. A tricortical bone block provides excellent fixation for implants until the bone graft is fully incorporated. In addition, the ilium provides a generous amount of cancellous bone.

The subcrestal window technique has been described by Behairy and Al-Sebai.[76] This provides a biocortical full-thickness graft while sparing an 8- to 10-mm bridge of iliac crest proximally. However, the harvest of the tricortical bone block is primarily used because of the excellent ridge of cortical bone provided by the crest. The procedure for harvest is described along with the reconstruction technique to prevent cosmetic deformity. This technique also limits hematoma formation caused by exposed cancellous bone. Herniation of abdominal contents is a well-known[77-82] complication of full-thickness iliac crest bone grafting. The technique described may limit this complication.[83] With proper technique, the harvest of tricortical bone graft from the anterior iliac crest can be performed with good results.

Procedure for Harvest of Iliac Crest

Place the patient supine and administer general anesthetic, with placement of a nasotracheal airway to allow free access to the oropharynx. The oral surgery is typically performed after harvest because of space constraints around the patient. If only a one-sided harvest is planned, then the surgeon should place a bump under that side. In selecting the side for harvest, one consideration is the side preferred for sleeping. If bone is to be harvested bilaterally, then no bump is used. Primarily, three different harvest sites of the ilium exist (Figure 40-5). The anterior iliac crest is preferred, because the posterior harvest is usually limited to one or two cortical plates and a tricortical bone harvest is desired. In addition, bone width at the crest of the ilium is often greater in the anterior region compared with the midcrestal anatomy. A 7- to 8-cm transverse incision is made either proximal or distal to the iliac crest. An incision directly over prominence of the iliac crest is avoided[84] because of likely irritation from clothing or seat belts. The incision must remain at least 2 cm lateral to the anterior superior iliac spine (ASIS) to avoid damage to the lateral femoral cutaneous nerve (LFCN), which can rarely course through this area. Electrocautery is used for hemostasis and to dissect through the subcutaneous tissues to the fascia overlying the ilium.

Beginning at least 2 cm lateral to the ASIS, the relatively avascular interval between the abdominal musculature superiorly and the abductor musculature inferiorly is identified and incised with electrocautery directly over the ilium.[85] Subperiosteal dissection by means of electrocautery and a Cobb elevator is used to expose the inner and outer tables of the iliac crest, clearing away the iliacus muscle medially and gluteal muscle at the outer table. It is imperative to remain in a subperiosteal plane during this exposure to avoid damaging neurovascular structures on the inner table. This minimizes muscular bleeding and thus hematoma formation. Staying within this plane also decreases postoperative pain and creates a thick periosteal and facial layer for adequate repair. A self-retaining retractor provides excellent visualization at this point.

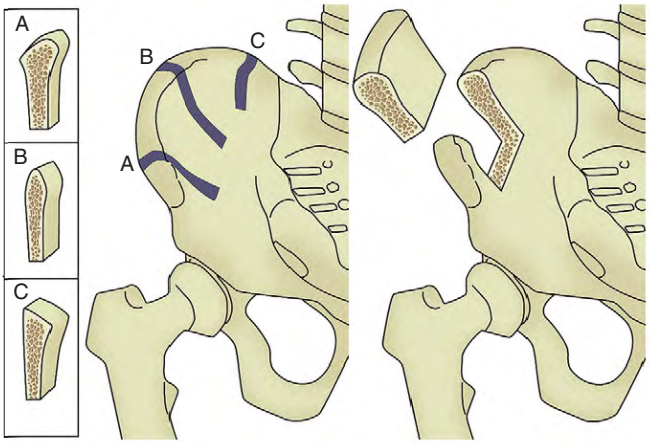


Figure 40-5 The iliac rest may be harvested from the anterior crest *(A)*, the midcrest *(B)*, or the posterior region *(C)*. The anterior site permits a thick tricortical harvest to augment the atrophic jaws.

WWW.HIGHDENT.IR

The ASIS is located and the iliac crest is cut sagittal at least 3 cm lateral, being careful to avoid wandering anteromedially. Attention to these details lessens the likelihood of ASIS avulsion by sartorius and tensor fascia latae muscles. The cut is made using the oscillating saw. The oscillating saw is preferred for sagittal cut as a result of biomechanical studies, which have shown that cutting the iliac crest with the osteotome has the effect of weakening graft strength.[86] A sizing guide is then taken (Figure 40-6), as determined by the oral surgeon preoperatively. It is placed on the inner table, and the size is marked directly onto the ilium. Care is taken to obtain a graft to match the sizing guide, because an inadequate graft lengthens the oral procedure considerably by requiring piecemeal reconstruction. A second sagittal cut is made with the oscillating saw. The area of the ilium where the bone thins considerably is located, and the bone is carefully osteotomized. This bone is then transferred to a basin with sterile saline until the oral surgeon begins contouring.

Technique for Reconstruction of the Iliac Crest

The wound is well irrigated with an antibiotic solution and suctioned dry. The pelvis is then inspected for fracture. A 0.062-inch unthreaded Kirschner wire is driven into one of the cut edges of the cancellous bone left exposed at the donor site. The Kirschner wire is measured, and a 3.5-mm cannulated cancellous screw is chosen so that about one third to half of the screw's length is left exposed, at least 1.0 to 1.5 cm (Figures 40-7 and 40-8). This cannulated screw is then driven over the Kirschner wire in usual fashion, and the Kirschner wire removed. Methylmethacrylate cement is mixed. Using the exact same procedure, a second screw is placed opposite the first (Figure 40-9). These screws are placed to act as rebar to stay the cement once it is placed.

Malleable retractors are now placed at the inner and outer tables, flush with the bone, to provide containment for the cement. Once the cement reaches the

Figure 40-6 A sizing guide is placed at the inner table to ensure adequate harvest for reconstruction. The ability to reconstruct the jaws is affected considerably, because inadequate bone harvest results in inadequate jaw augmentation or piecemeal reconstruction.

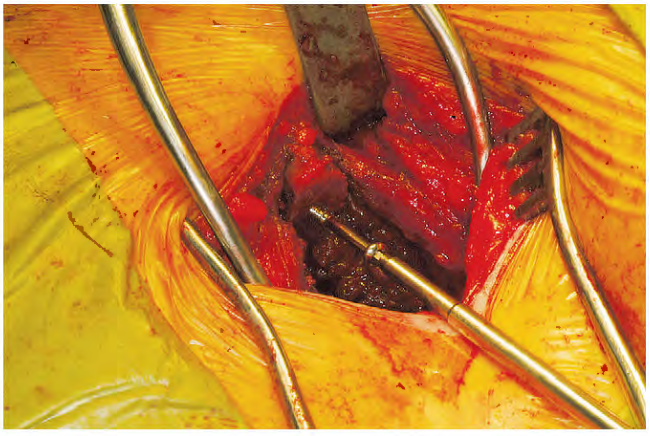


Figure 40-8 A cannulated screw is placed over a Kirschner wire and driven to a depth such that approximately one third of the screw is left exposed.

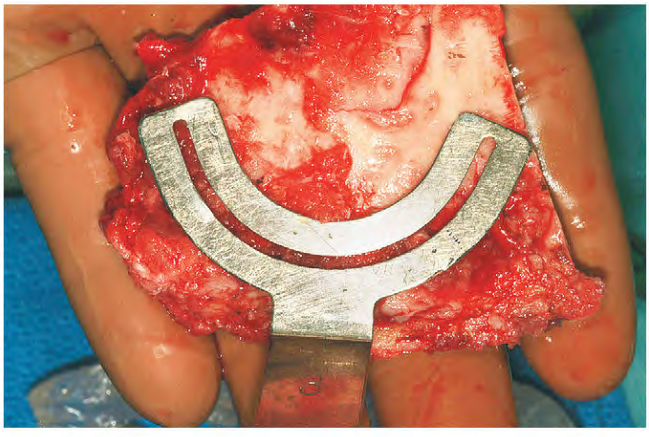


Figure 40-7 A measuring guide is used to determine and verify sufficient volume of the harvested block.

Figure 40-9 Screws are in place, and the site is prepared for bone cement reconstruction.

WWW.HIGHDENT.IR

همیار دندانسازان

consistency of putty, an appropriate amount is placed at the reconstruction site so that it mimics the bone block harvested. Great care is taken with the use of the malleable retractors and large osteotomes, if necessary, to contain the cement (Figure 40-10). As the cement hardens, the retractors are moved slightly to prevent adherence of the cement. The superior aspect of the cement is smoothed with the fingers so that the bone-cement interface is smooth and contoured to the normal anatomy. More cement is added if necessary (Figure 40-11). The wound is irrigated during hardening to prevent thermal damage. Once the cement is hardened, retractors are removed and the reconstruction is inspected for loose cement. Sharp edges are trimmed with a rongeur (Figure 40-12).

The wound is again irrigated. A Marcaine pain pump is placed below the fascia exiting away from the wound. The abdominal and abductor fascia is repaired carefully with No. 1 Vicryl. For subcuticular closure, 2-0 Vicryl is used; the skin is closed with 4-0 Monocryl. Benzoin tincture and Steri-Strips are applied liberally, and a sterile dressing is applied. Scrub is broken, and care is taken by the first team not to contaminate the back table, which will continue to be used during the oral reconstruction.

Figure 40-10 Malleable retractors are used to contain the bone cement.

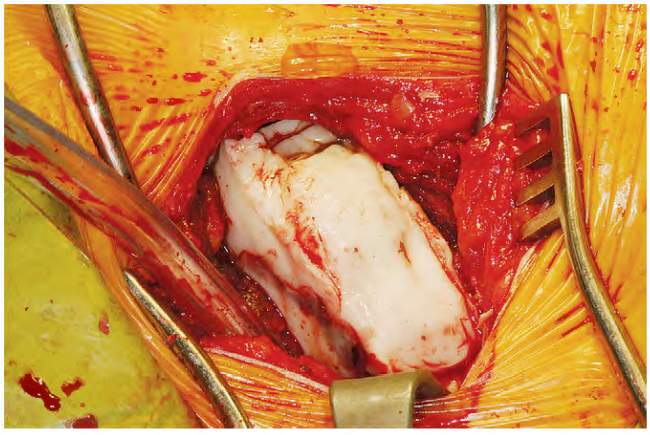


Figure 40-11 Reconstructed iliac crest.

Neurovascular Structures

In this approach the deep circumflex iliac artery is at risk as it courses approximately 2.5 cm inferior to the iliac crest on its internal margin in a tunnel along the transversus abdominis and iliac fasciae. The fourth lumbar and the iliolumbar arteries are also potentially at risk in this approach, because they parallel the crest as they transverse the iliacus and abdominal wall muscles. Subperiosteal dissection and careful retraction at the inner table will prevent damage and subsequent hematoma formation.

The LFCN typically crosses the anterior surface of the iliacus muscle before passing into the thigh, near the ASIS. Normally the nerve passes beneath the inguinal ligament and sartorius muscle; however, in as many as 10% of cases, it may pass over the iliac crest at a point as much as 2 cm lateral to the ASIS.[87,88] The technique, as described, prevents damage to the LFCM.

At the level of the anterior iliac crest, the ilioinguinal nerve passes over the transversus abdominis and internal oblique muscles. It then courses under the external oblique muscle, entering the inguinal canal to pass distally, supplying sensation to the genitalia and nearby skin. Great care should be taken to approach the inner table in the subperiosteal fashion and careful attention given when retracting or using the oscillating saw.[89]

The iliohypogastric nerve, at the level of the anterior iliac crest, passes lateral to the transversus abdominis (passing between that muscle and the obliquus internus abdominis). At this level, the nerve is located slightly more proximal than the ilioinguinal. Neuralgia of the iliohypogastric nerve can result as described previously for the ilioinguinal nerve.

The femoral nerve is also at risk when harvesting bone from the inner table. Located more distally than the ilioinguinal nerve, it is found in the iliac fossa as it

Figure 40-12 A rongeur is used to trim sharp edges.

WWW.HIGHDENT.IR

courses over the iliacus muscle, entering the thigh below the inguinal ligament.

Recipient Site Surgery

Division C–w

An incision is placed on the crest of the C–w ridge, and a full-thickness periosteal reflection exposes the moderately resorbed residual ridge. The posterior ilium has been shown to contain twice as much trabecular bone as the anterior ilium and also to result in less blood loss, morbidity, and no long-term gait disturbances. Therefore both approaches can be selected.[90] A block of one cortical plate and trabecular bone may be harvested from the ilium, or a tricortical harvest from the anterior ilium is made, which is then divided into two cortical and trabecular blocks. In either case, usually four blocks are made. Each bone block is contoured to the desired shape, and the trabecular component is compressed to increase the number of cells. The overlaying cortical plate of host bone is perforated with a small-diameter drill scored to enhance revascularization and to establish the regional acceleratory phenomenon (RAP).[91] The block bone graft is fixated with titanium screws to the lateral aspect of the recipient bone to restore the final ridge form in a more ideal position for future implant placement. The most usual placement is on the facial aspect of the C–w ridge. However, an Angle's Class III mandible may indicate lingual fixation of the bony block. The block graft may also be extended above the crest of the atrophic ridge to increase bone height, when the block graft is greater than 6 mm wide.

Once the block is fixed into position, bone marrow aspirant from the ilium harvest and additional trabecular bone harvested from the ilium are placed around the blocks and fill in any defects. This blend may also be placed under the block before fixation if any irregularity in the host bone exists. The tissues are approximated without tension with horizontal mattress sutures (Figure 40-13). Autogenous grafts of extraoral origin are often not necessary where only one or two segment of the maxilla necessitate bone grafting for width, because intraoral block grafts are often effective with multiple harvest sites.

Division C–h

The patient with a Division C–h ridge has moderate bone in height for endosteal implants and implant prosthetic treatment. In selected cases, shorter endosteal implants or a transosteal implant system can be used for a stable, removable restoration that may be an RP-5 or, with greater implant number, an RP-4 prosthesis. Patients with moderate desires can still be treated adequately by these implant modalities, provided the crown height and factors of stress are compatible with the planned prosthesis. However, for the more-demanding patient expecting a fixed restoration, the addition of autogenous bone is often necessary to obtain sufficient height and width of bone (especially in the maxilla). Most patients with C–h ridges requiring grafts are being treated for improved prosthodontic conditions in the maxillary arch.

The greater available bone after ridge augmentation permits the placement of ideal length and wider implants, improves the surface area of support, improves the stress distribution, recreates a more favorable crown/implant ratio, and returns the soft tissue contours to a more natural appearance. Most often, intraoral block grafts, even with multiple donor sites, are inadequate to augment a C–h edentulous ridge. Many C–h premaxilla treatment plans for a fixed restoration mandate augmentation with autogenous bone grafts before the placement of implants.

The initial incision for C–h available bone is in keratinized tissue and on the palatal aspect of the crest in the maxilla and the midcrest of the mandible, from second premolar to second premolar region. A mucoperiosteal reflection exposes the anterior section of the C–h host bone. A tunnel procedure is used to expose the lateral posterior maxilla or posterior mandible when onlay augmentation is desired.

The soft tissue is prepared for primary closure, without tension, before positioning the block of bone. An incision is made through the periosteum 3 to 5 mm above the height of the mucogingival junction and above the height of the vestibule. Soft tissue scissors are then introduced through the periosteum, and a blunt dissection creates a submucosal tunnel, which extends 10 to 15 mm toward the vermillion border of the lip. On soft tissue closure, the vestibule is reduced, but this is usually of no consequence because implants are used to support the restoration, rather than relying on the valve seal of a removable prosthesis.

The bone harvested from the donor site is often a full-thickness, tricortical block from the anterior ilium. One side of the cortex is thinned and contoured to fit the C–h host bone and perforated to allow revascularization of the trabecular component of the block. Surgical templates designed before surgery are used to help contour the block of bone.

Before the sinus graft, computed tomography (CT) reformatted images are indicated to evaluate the ridge topography. This CT process should also have a CT model fabricated of the resorbed maxilla or mandible. The harvested block may then use the CT model as a template to contour the arch and mating surface of the iliac crest block. When possible, rather than remove bone during the recontouring process, it is compressed to increase the cellular component of the graft.[92] Any bone removed from the block is stored in sterile saline to fill any voids or irregularities between the bone block and the recipient bone.[93]

The C–h edentulous available bone is usually augmented with a block graft on the crest of the atrophic

WWW.HIGHDENT.IR

همیار دندانسازان

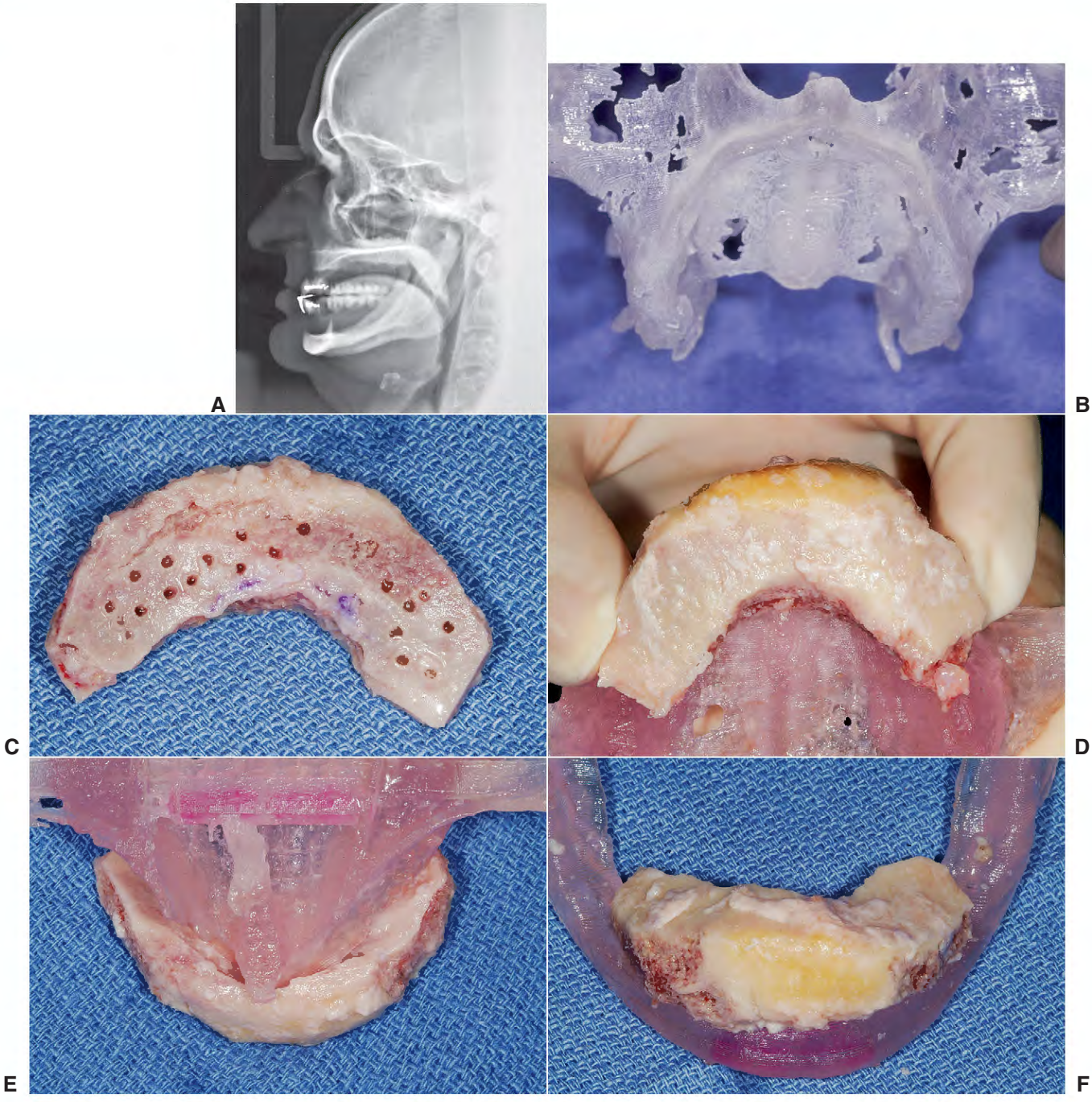


Figure 40-13 **A,** A lateral cephalometric radiograph of the Division C–w maxilla and Division D mandible. This film helps determine the position of the iliac crest block bone grafts. **B,** A computed tomography (CT) model of the Division C–w maxilla (palatal view). Note the thin premaxilla and premolar regions. **C,** The tricortical bone is prepared with holes through the cortical plate on the recipient bone surface side. The cortical plates are compressed together to compress the trabecular bone within. **D,** The block is shaped to fit the CT model of the C–w maxilla. **E,** The tricortical block is positioned more facial on the CT model to compensate for the facial bone loss. **F,** The tricortical iliac crest block is shaped to fit the CT model of the mandible.

Continued

bone. It often extends to the facial aspect of the ridge several millimeters in the maxilla, whereas it is positioned more lingually in the mandible. Cephalometric analysis may be required to predetermine ideal graft placement. Four to six titanium screws that are 2 mm in diameter and 12 to 18 mm long usually are used to fixate the block graft of autogenous bone. Intimate adaptation of the graft to the recipient bed and rigid fixation are paramount to graft success. An attempt is made to place the screws flush with the block graft.

Additional cortical and trabecular bone harvested from the recontouring of the block and stored in sterile saline[93] is made into particulate bone 3 × 5 × 5 mm in size and then compressed in a 5- to 10-mL

WWW.HIGHDENT.IR

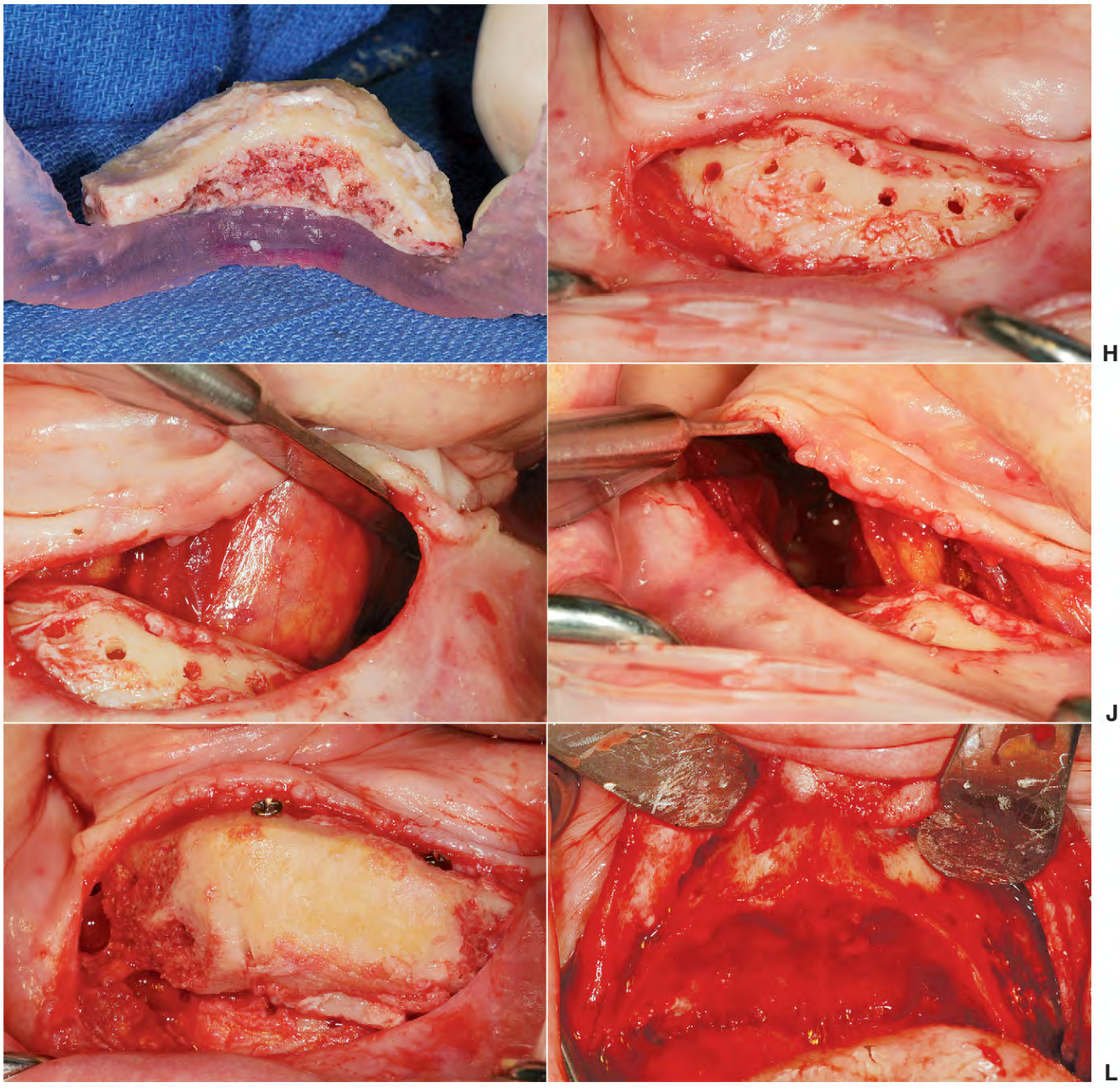


Figure 40-13, cont'd **G,** The bone is positioned more lingual, to compensate for the alveolar bone position before resorption. **H,** An incision is made on the crest of the ridge from canine to canine region. A full-thickness mucoperiosteal reflection is made. The anterior mandible is decorticated with a small-diameter (1.8 mm) drill. **I,** A subperiosteal tunnel is made in the posterior region. An elevator proceeds along the mylohyoid ridge to the retromolar pad, then facial along the lateral body of the mandible. **J,** A subperiosteal tunnel is made in the contralateral posterior region. The mental neurovascular structure is separated from the flap. **K,** The previously shaped bone block is rotated within the posterior tunnels and fixated to the mandible with 2-mm fixation screws between the mental foramina. **L,** The maxillary arch has a full-thickness mucoperiosteal reflection from tuberosity to tuberosity. The facial plate is decorticated. The sinus grafts (previously performed) are evaluated.

Continued

modified syringe.[92] It may be added ("injected") into the posterior regions (sinus graft and/or onlay tunnel technique) and on the labial regions above the block of autogenous bone. This creates further soft tissue support in the anterior and greater bone volume for future implant placement. The bone marrow aspirant (20 to 30 mL) is placed over the grafted site, especially on the mixed cortical and trabecular bone. The tissues are approximated with a nonresorbable suture (e.g., nylon or expanded polytetrafluoroethylene [e-PTFE]) to ensure adequate suture strength for the initial 3 weeks before the sutures are removed. Silk is not suggested because of its potential to wick bacteria under the soft tissue (Figure 40-14).

The endosteal implants are placed 5 to 10 months after the iliac crest graft, using a surgical template designed after the final graft is determined to optimize anterior implant placement. A staged implant placement

WWW.HIGHDENT.IR

همیار دندانسازان

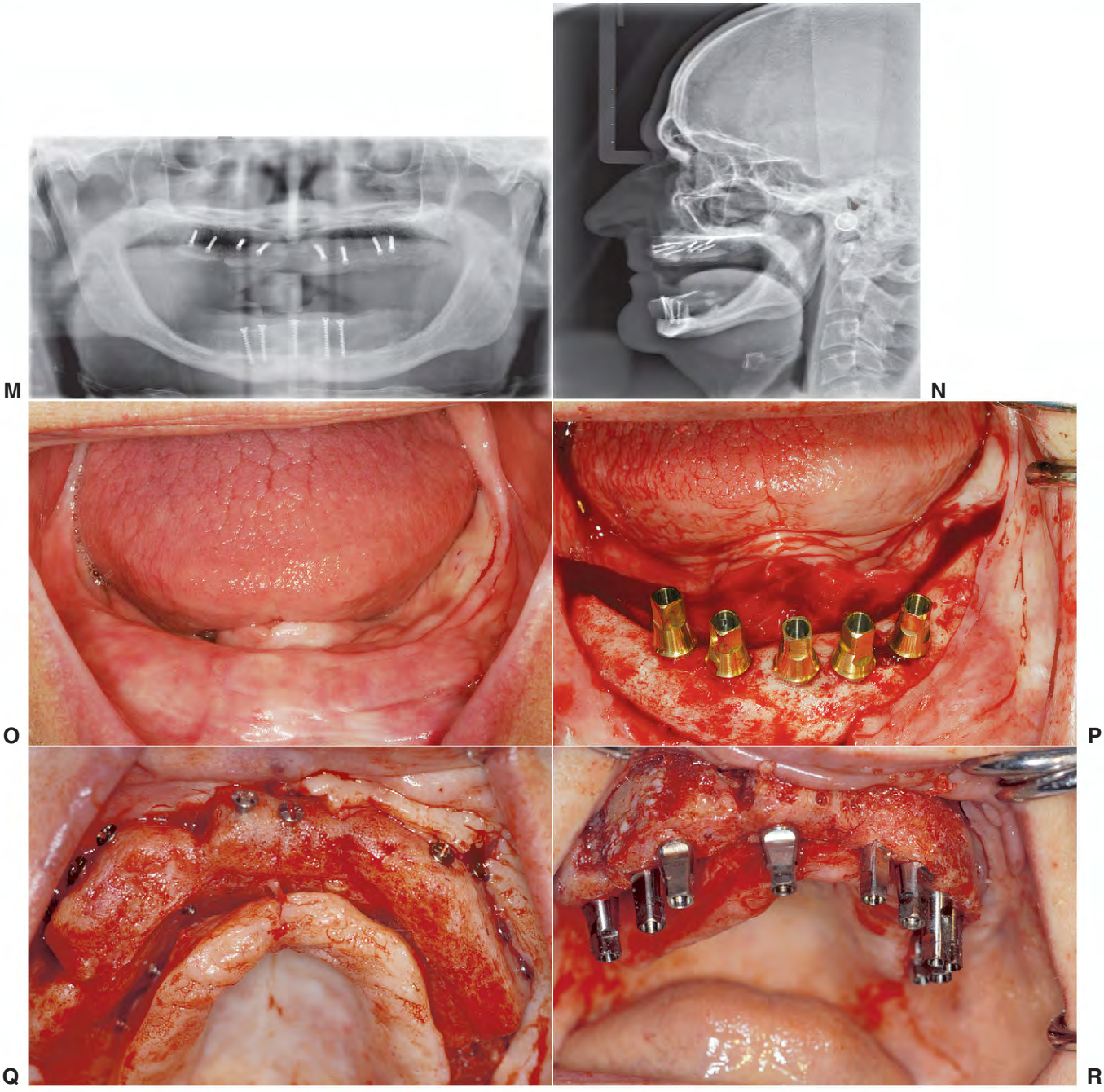


Figure 40-13, cont'd **M,** A postoperative panoramic radiograph of the iliac crest tricortical blocks fixated to the maxilla and mandible. **N,** A lateral cephalogram of the maxilla and mandible after graft fixation. **O,** An intraoral view of the augmented mandible. **P,** After 6 months of healing, five BioHorizons Maestro implants are positioned between the mental foramina. Two additional implants (one over each foramen) will also be inserted. **Q,** A 6-month reentry into the augmented maxilla. The block of bone was fixated with 2-mm screws through the C–w bone. **R,** The fixation screws were removed, and 10 BioHorizons Maestro implants were inserted in the bone block. *Continued*

reduces the risks associated with incision line opening, which may especially occur in maxillary patients when the opposing arch has only anterior natural teeth (Figure 40-15).

Division D

The completely edentulous Type I, Division D patient has a flat maxilla or pencil-thin mandible with dehiscent mandibular canals, occasionally accompanied by paresthesia of the lower lip. These patients require special considerations because of unfavorable biomechanical, surgical, prosthetic, and esthetic factors. Although these patients have greater requirements to satisfy their oral condition, the severe bone loss creates an unfavorable crown height (four times normal), lack of soft tissue support, poor bone quality from decreased blood supply, complexity of surgical procedure, poor tongue position, unfavorable ridge relationship, increased moment

WWW.HIGHDENT.IR

Figure 40-13, cont'd **S,** After 6 months, the maxillary transitional prosthesis is removed and abutments are added to the mandibular implants. **T,** An intraoral view of the maxilla and mandible after abutment preparation. **U,** Maxillary and mandibular fixed prostheses are both porcelain-metal. **V,** The maxillary prosthesis is cemented to the maxillary implants. **W,** The maxillary and mandibular prostheses in centric relation occlusion. **X,** The high smile lip position of the patient after iliac crest bone grafts, implants, and fixed prostheses.

forces, and unfavorable stress distribution patterns for predictable endosteal implant support (Figures 40-16 and 40-17).

Autogenous grafts represent the treatment of choice before nearly any implant restoration in the Division D maxilla or mandible. The risks associated with the aggressive placement of implants of any type in the severely atrophic mandible have been previously reported.[94,95] Regardless of past training in any surgical or prosthetic discipline, the clinician must remember that these patients require many unique surgical and prosthodontic treatment modifications. The patient's condition may worsen rather than improve if treatment plans are not specific and well coordinated. Implant survival of 90% in Division D conditions is more dangerous for these patients, because the 10% failure may result in atrophic mandibular fractures, oroantral fistulae, and more extensive complications, requiring many surgeries to restore them to their original poor Division D condition. Therefore augmentation is indicated for most patients before implant placement (Figure 40-18).

Exposure of the mandible should include the crest of the atrophic ridge between the mental foramen, the

WWW.HIGHDENT.IR

همیار دندانسازان

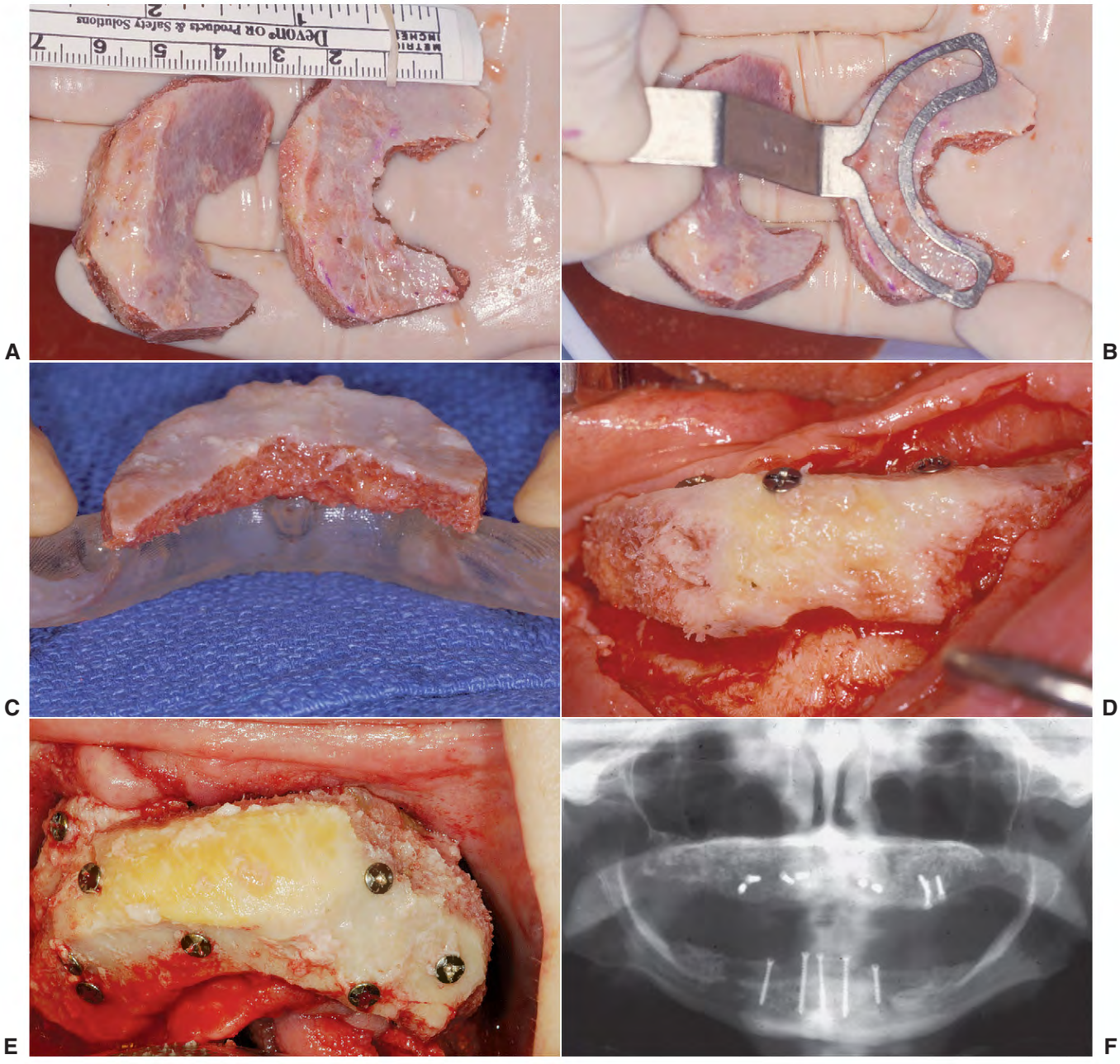


Figure 40-14 **A,** Bilateral, tricortical iliac crest bone grafts are harvested from the ilium. **B,** The bone is initially shaped to fit a bone harvest template (P.I. Brånemark, NobelPharma). **C,** The tricortical block is shaped to fit a computed tomography model of the mandible. **D,** The block of bone is contoured toward the lingual in relation to the original position of the alveolus. **E,** The tricortical block is fixated to the maxilla with 2-mm-diameter fixation screws. **F,** A postoperative panoramic radiograph of the iliac crest bone grafts to the maxilla and mandible. *Continued*

mandibular symphysis, mental foramina, and superior genial tubercles. A subperiosteal tunnel is created along the lingual aspect of the mandible, along the mylohyoid ridge and extending to the retromolar pad region. Even in severely atrophic mandibles with mandibular nerve dehiscence, bone covers the mandibular canal under the retromolar pad. As a result, a periosteal elevator within the retromolar lingual tunnel may be used to elevate the pad and then proceed forward, separating the overlying tissues off the mandibular neurovascular complex. The tunnel may then be expanded, exposing the external oblique (which is greatly reduced in height), lateral body, and ascending ramus lateral to the retromolar pad. Care is taken not to reflect muscle attachments of the mylohyoid, genioglossus, masseter, temporalis, and digastric muscles, because these provide the primary blood supply to the mandible and soft tissues that will revascularize the bone graft.

Division D mandibles often result in square-shaped arches, and anterior implants are more likely to be placed in a straight line. Mandibular nerve repositioning may be indicated when patients desire a fixed prosthesis,

WWW.HIGHDENT.IR

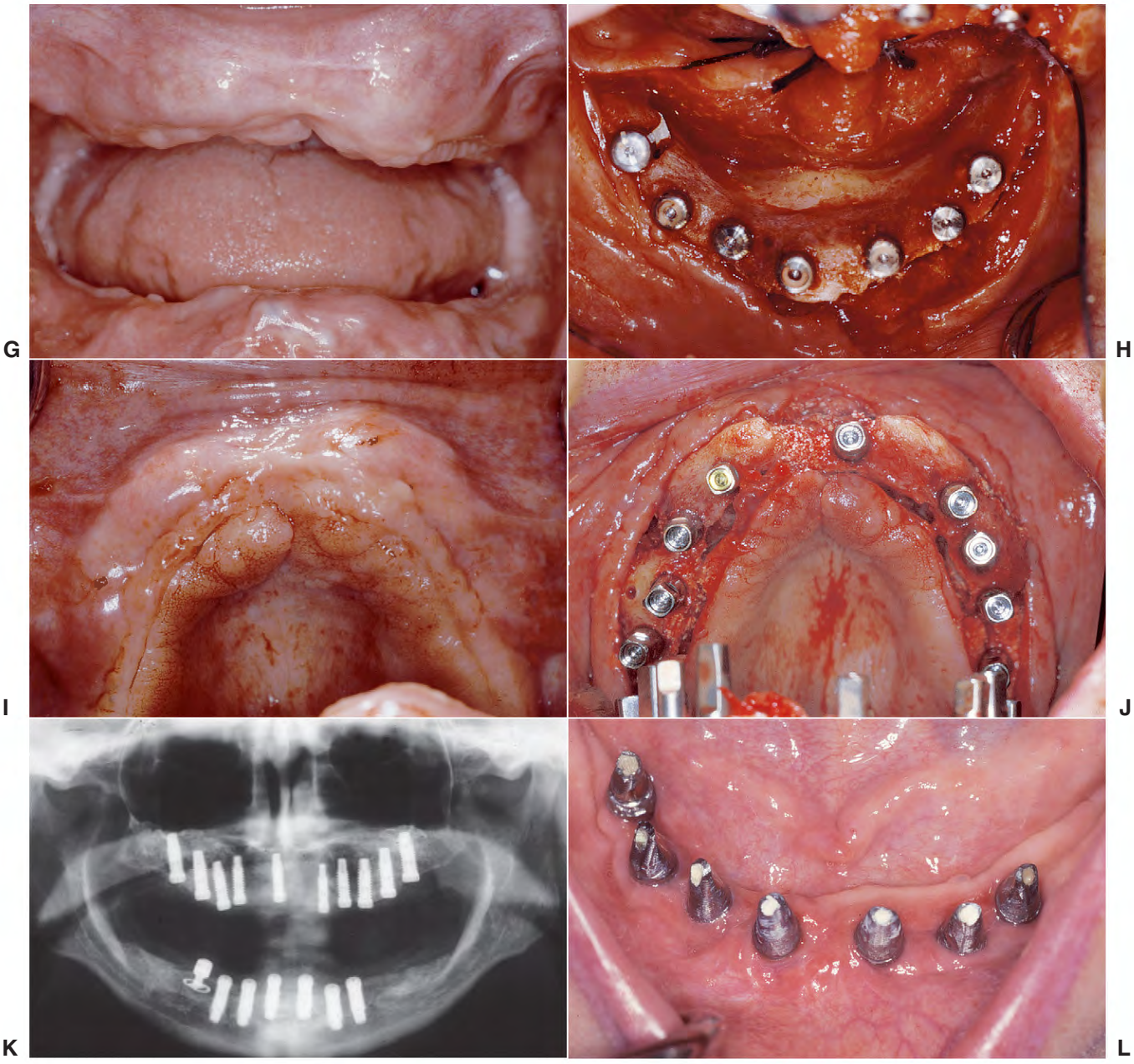


Figure 40-14, cont'd **G,** A 6-month postoperative intraoral view of the maxillary and mandibular arch after iliac crest grafts. **H,** Seven implants are inserted into the mandibular arch, 6 months after the iliac crest graft. **I,** Occlusal view of the maxillary arch 6 months after iliac crest graft. **J,** Ten maxillary implants are inserted into the maxillary arch, 6 months after the iliac crest graft. **K,** A postoperative radiograph of 10 implants in the maxillary arch and seven implants in the mandible arch. **L,** After 6 months, abutments are inserted into the mandibular implants. *Continued*

because additional implants and shorter cantilevered restorations result. Nerve repositioning is much less difficult in the Division D bone when the canal is dehiscent. In addition, many of these patients already have paresthesia of the mandibular nerve complex. Ease of surgery and previous history of paresthesia result in complications less often affecting the overall treatment. This improves the A-P distance for implant placement, because it places the most distal implant in the second premolar or first molar region. This dramatically decreases the length of the cantilever and modifies the arch form to an overall more favorable condition for long-term survival of the prosthesis.

As in the C–h patient, the crest of the ilium is sectioned so that the crest and bicortical inner and outer table are harvested. In this manner, the cortical bone may be shaped to augment the anterior segment of the atrophic arch width and height and extend at least to the first to second premolar region. A surgical template is selected that corresponds to the arch form. Once the block is contoured using hand rongeurs, the interior cortical bone of the block is thinned, decorticated with

WWW.HIGHDENT.IR

همیار دندانسازان

Figure 40-14, cont'd **M,** A porcelain-metal fixed restoration is fabricated and cemented on the seven implants. **N,** After 6 months from implant insertion, abutments are placed and prepared in the maxillary arch. **O,** The maxillary and mandibular fixed prostheses in centric relation occlusion. **P,** A panoramic radiograph of the maxillary and mandibular fixed prostheses. One implant in the posterior left maxilla was in a second molar position and was submerged rather than restored.

round drills, and compressed to fit the atrophic ridge. The trabecular portion of the block is compressed to increase the density of cells per area. The graft is placed and contoured with awareness of the final form for the proposed restoration.

Four to six fixation screws are placed through the block of bone and fixated into the atrophic recipient bone. A screw may be placed in the mandibular midline, which maintains width and density from the genial tubercles and muscle attachments. Additional screws may also be placed in the anterior region as required.

The host bone in the mandible is often D1 density, and care is taken to direct the fixation osteotomy to the center of the atrophic ridge. When possible, the inferior border is not completely penetrated to maintain cortical integrity for additional strength during the initial healing process. The grafted bone block is contoured for smooth borders, and any remaining bone harvested is ground in a bone mill or with hand rongeurs to a dimension of $3 \times 5 \times 5$ mm, compressed in a 5- to 10-mL syringe to increase the cells per volume, and placed into the distal periosteal tunnel. If additional volume is required, then 10 to 50 mL of bone marrow aspirant from the ilium and resorbable calcium phosphate ($CaPO_4$) may be added to the cortical and trabecular particulate mixture.

After 5 to 10 months, five to seven implants may be placed between the foramina in the mandible. Those endosteal implants may support a final fixed prosthesis, and no soft tissue support is required over the distal aspects of the autogenous graft. When adequate bone height is present above the mandibular canal, implants may also be inserted (Figure 40-19).

Maxillary Division D bone graft approaches have evolved over the years. Sinus grafts are usually performed before the iliac crest graft procedure (Figure 40-20). A full-thickness primary incision is carried from the anterior aspect of the arch to the distal aspect of the canines (similar to the C–h patient). Care is taken to avoid incision through the nasal mucosa, which may increase the risk of infection. A mucoperiosteal reflection exposes the posterior maxilla zygomatic processes, infra-orbital foramina, lateral and inferior piriform rims, nasal spine, incisive foramen, and anterior one third of the hard palate. A small rotary drill is used to perforate the crest of the maxilla (or slightly score it when the atrophic ridge is less than 5 mm in height) to enhance revascular-

WWW.HIGHDENT.IR

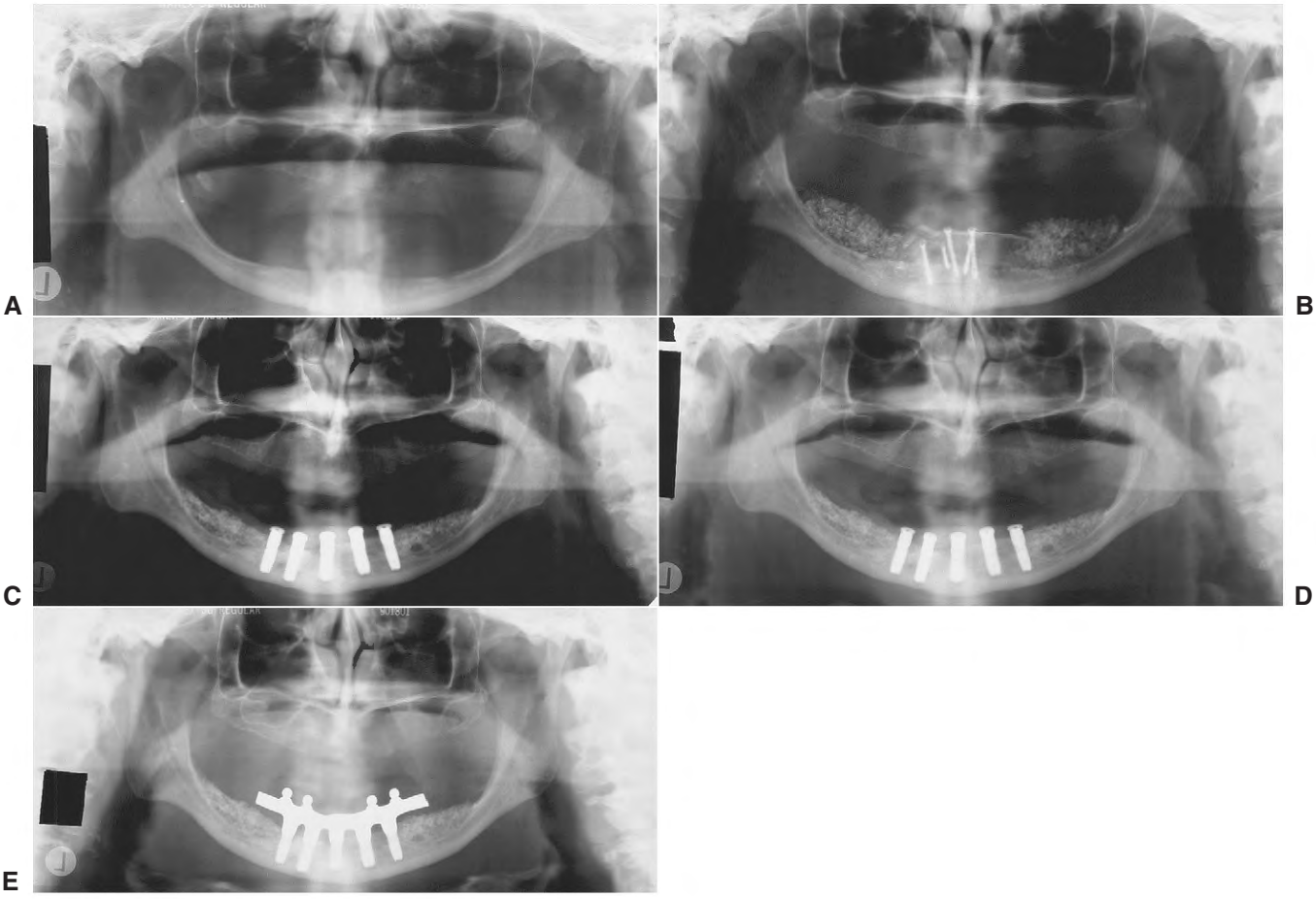


Figure 40-15 **A,** A panoramic radiograph of a C–h edentulous mandible. **B,** A postoperative radiograph of a C–h edentulous mandible after iliac crest bone graft. Particulate graft material, mixed with dense hydroxyapatite, is injected into the posterior mucoperiosteal tunnels before the tricortical block is fixated between the mental foramens. **C,** Six months after the iliac crest graft, five BioHorizons Maestro implants are inserted between the mental foramens as a one-stage surgical approach. **D,** Six months after the implant insertion, the iliac crest graft is now more mature (1 year after surgery). **E,** A cantilevered bar is attached to the five implants to support an RP-4 overdenture.

Figure 40-16 A Division D mandible has unfavorable crown height–ridge relationships and decreased available bone for implants.

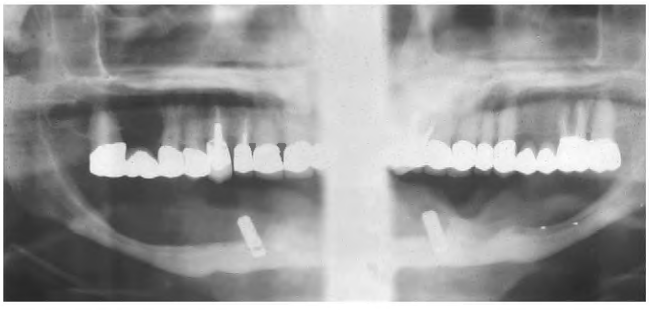


Figure 40-17 A panoramic radiograph of the maxillary dentition opposing a Division D mandible. The two mandibular implants are failing from the excessive force and inadequate support.

ization of the graft and provide the RAP, as is done for the preparation of all recipient beds for bone grafting.

The soft tissue is manipulated and expanded before positioning the block of bone. The technique is similar to that described for the C–h ridge. However, the submucosal tunnel may obliterate the entire vestibule to achieve primary closure. The particulate harvested bone, mixed along with bone aspirant and resorbable $CaPO_4$, may be used to augment the bottom portion of the SA region. The lower one third of the previous alloplastic graft in the sinus can be removed and replaced by particulate autogenous bone when necessary.

WWW.HIGHDENT.IR

همیار دندانسازان

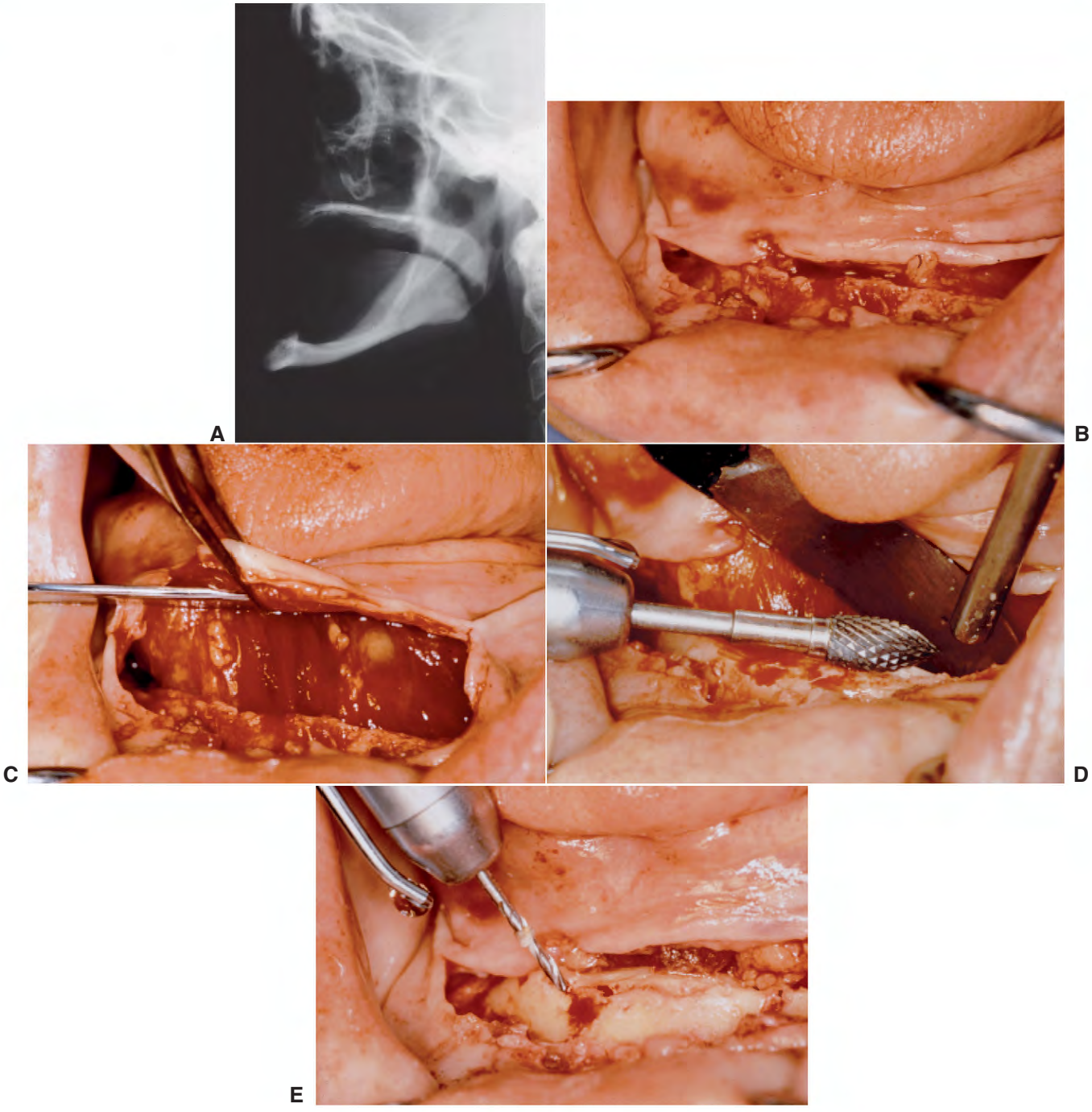


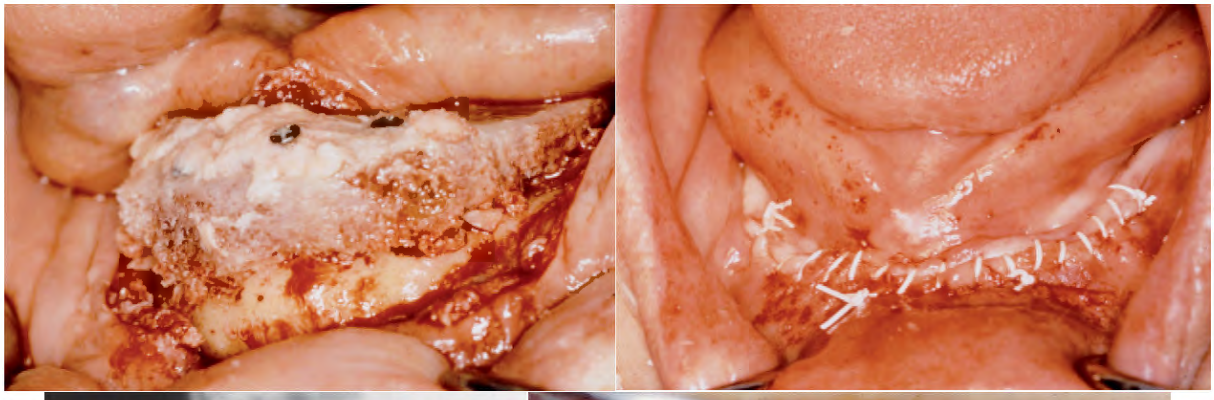
Figure 40-18 **A,** A lateral cephalometric radiograph of a Division D maxilla and mandible. **B,** An incision is made on the crest of the ridge between the mental foramens. **C,** A tunnel is made to the posterior region by sliding along the mylohyoid ridge of the mandible to the retromolar pad, then along the body of the mandible. The mental neurovascular contents are separated from the flap. **D,** The mandible is recontoured with a large carbide drill between the foramens to increase the regional acceleratory phenomenon. **E,** A small-diameter drill is used to penetrate the cortical plate.

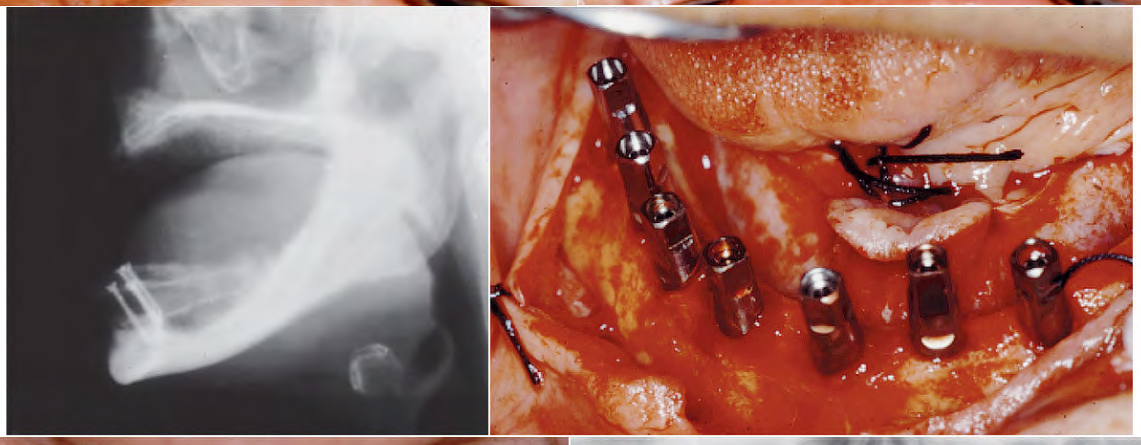
Continued

In the Division D premaxilla, subnasal elevation to gain approximately 2 to 4 mm in height may be indicated in the lateral piriform rim for canine implants or the inferior piriform rim for lateral or central incisor implants. Once this procedure is accomplished bilaterally, a template is selected to contour the premaxillary block graft. The harvested full-thickness block graft, which corresponds in size to the template, is shaped for proper contour with hand rongeurs. As for the C–h maxilla, a CT model can assist in contouring the corticotrabecular block to the recipient bone anatomy.

The block usually needs to be fixated 5 to 8 mm anterior to the residual ridge to improve the facial contour of the maxillary hard and soft tissues. The inferior

WWW.HIGHDENT.IR





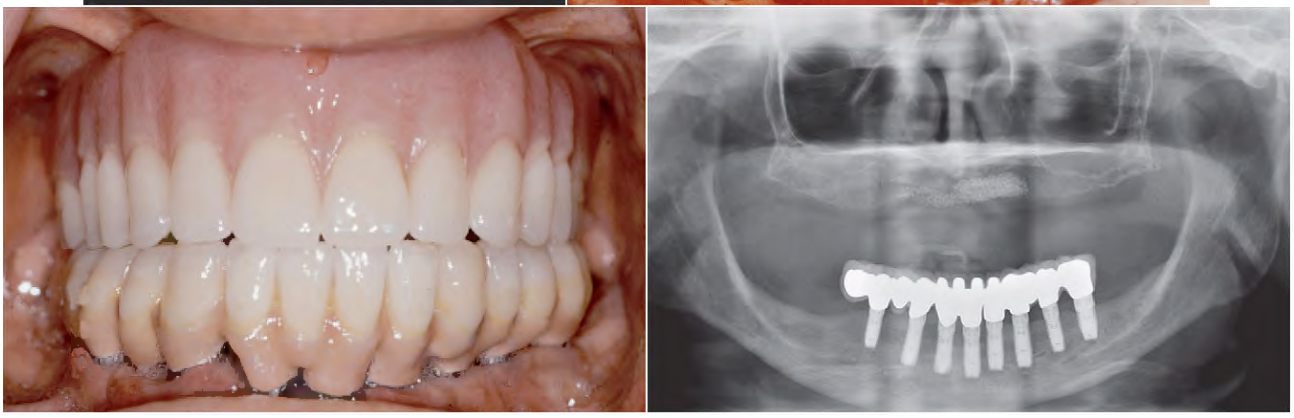


Figure 40-18, cont'd **F,** A tricortical block of bone is recontoured to fit the atrophic mandible. It is fixated with 2.0-mm-diameter screws between the mental foramina. **G,** Primary closure without tension is accomplished with the submucosal space soft tissue technique. Gore-Tex sutures are allowed to remain in place for 3 weeks. **H,** A lateral cephalogram 6 months after bone augmentation confirms an increase of 16 to 20 mm in height. **I,** Eight implants are inserted into the mandible, 6 months after the iliac crest graft. **J,** The mandibular porcelain-metal fixed prosthesis cemented to the mandibular implants opposing a complete denture. **K,** An 8-year postoperative radiograph of the eight implants and fixed prosthesis. The crestal bone levels are similar to implants in ungrafted bone.

cortical surface of the block is thinned, perforated, and compressed to increase the cellular component per area. A template of the denture is used to identify the canine to first premolar region in the block graft. A fixation screw is inserted in this region. Additional screws are added as needed to ensure absolute stability of the graft.

After block fixation, a rotary drill is used to smooth the contours of the corticotrabecular block graft, which is then covered with particulate bone and PRP. These materials are also used to fill the voids above the block positioned anteriorly. The soft tissues are approximated with interrupted horizontal mattress nylon or e-PTFE sutures without tension. After 5 to 10 months, a total of 7 to 10 implants may be placed in the anterior and posterior regions for an RP-4 or fixed (FP-3) prosthesis (Figures 40-21 and 40-22).

WWW.HIGHDENT.IR

همیار دندانسازان

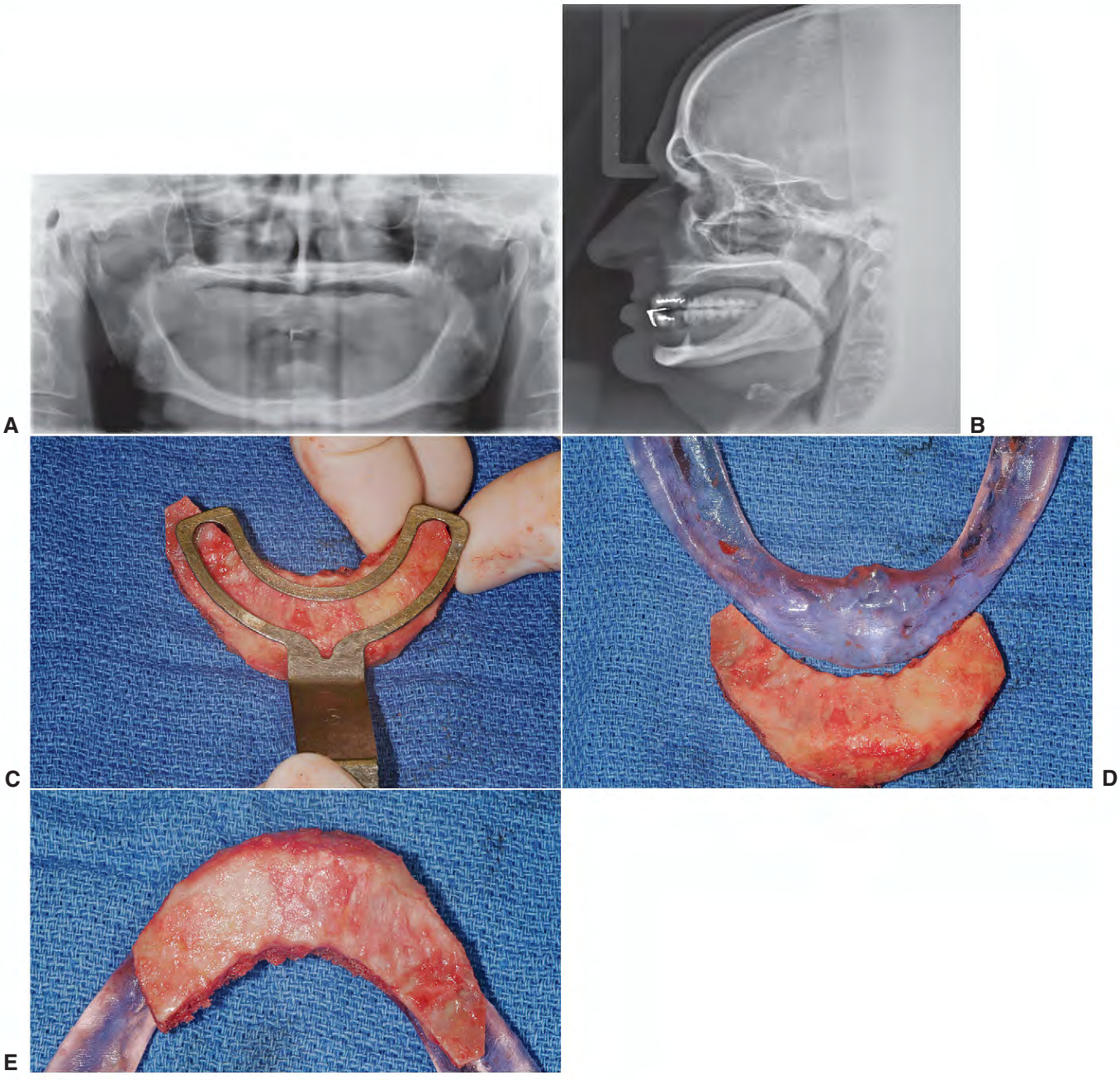


Figure 40-19 **A,** A panoramic radiograph of a completely edentulous maxilla and mandible. **B,** A lateral cephalogram of a patient with a Division D mandible. **C,** The bone is recontoured to the approximate shape of the surgical template **D,** The cortical block is shaped to fit the computed tomography model. **E,** The tricortical block projects toward the lingual aspect of the mandible. *Continued*

Iliac Crest Postoperative Analgesia

The postoperative pain and gait disturbances from harvesting bone from the iliac crest are significant surgical considerations. The use of an epidural catheter for infusion of local anesthetic into the iliac donor site has shown a dramatic decrease in postoperative pain and much earlier ambulation.[96] Bupivacaine is administered through a catheter every 6 to 12 hours as needed for pain relief. The catheter is usually removed on the second or third postoperative day.

Complications: Iliac Crest

A variety of complications have been associated with harvest of full-thickness iliac crest bone graft, including pain, herniation of abdominal contents, fracture, neuralgia, hematoma, seroma, infection, and cosmetic deformity.[79-83,97-103] Cosmesis is more often troublesome to the thin patient, who often exhibits proud contour of the pelvis.

Donor site pain is likely the most common complication after the iliac bone graft harvesting. A variety

WWW.HIGHDENT.IR

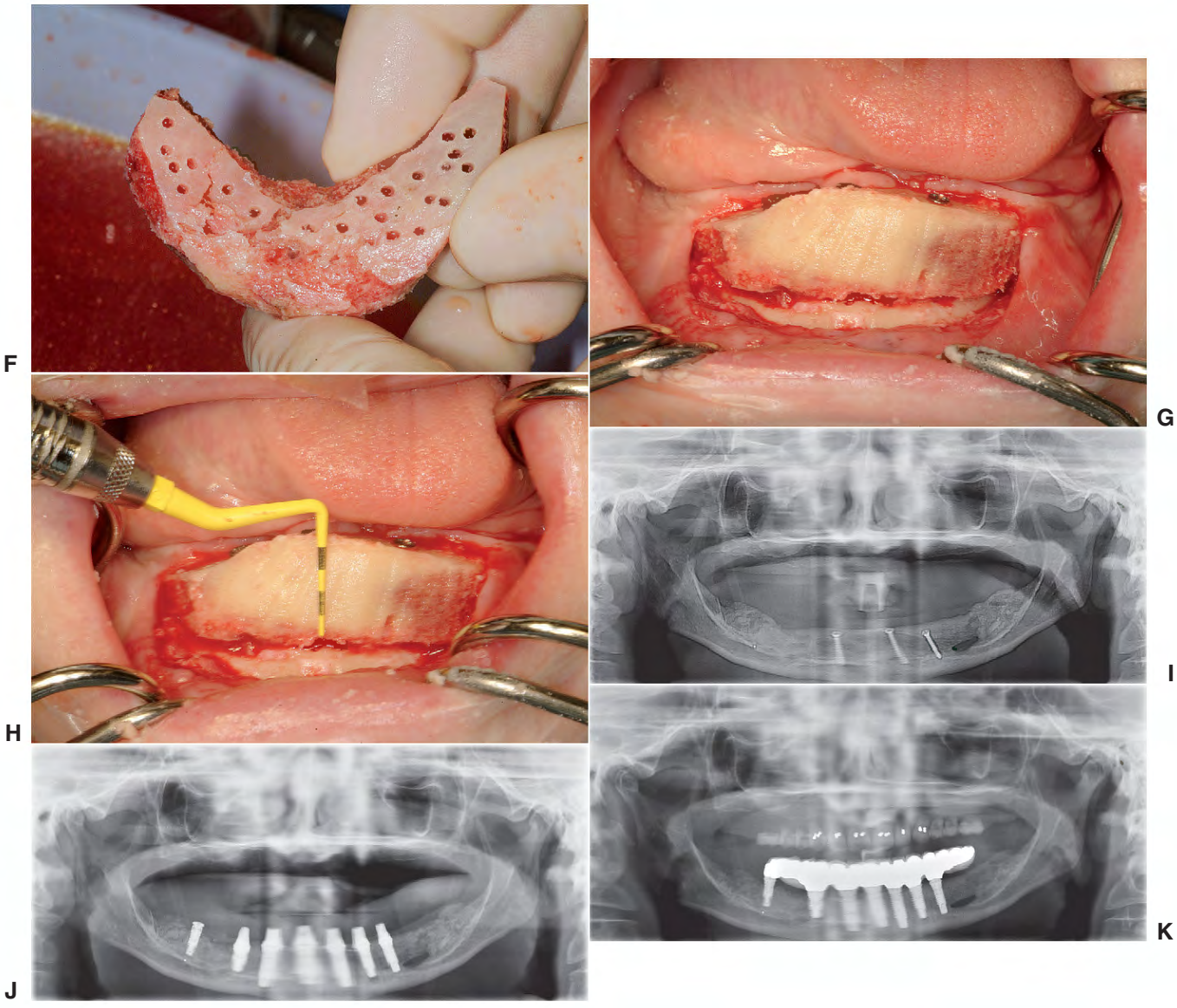


Figure 40-19, cont'd **F,** The bone surface of the bone block is decorticated with a small-diameter drill. **G,** The particulate bone is injected into the posterior tunnels, and the tricortical block is fixated with a 2-mm fixation screw placed between the mental foramina. **H,** The tricortical bone block measures 15 mm at the midline. **I,** A postoperative panoramic radiograph of the iliac crest block fixated by three fixation screws. **J,** Six months later, seven BioHorizons Maestro implants are inserted into the mandible, and an immediate restoration is delivered. The patient removes the maxillary denture at night. **K,** Six months later, a cemented porcelain-metal prosthesis is fabricated. A panoramic radiograph illustrates the seven implants and mandibular restoration.

of authors[98-100] have indicated donor site pain to occur in 15% to 29% of patients. Use of the avascular plane may limit such donor site pain.

Hematomas are more common for anterior structural harvests than for posterior harvests and can occur in as many as 10% of cases.[101] The presently described harvesting technique allows for closure without suction drainage, with no increased risk of hematoma because the methyl methacrylate reconstruction provides local tamponade, in the author's experience.

Herniation of abdominal contents can occur after full-thickness iliac crest bone harvest.[102] This is an extremely rare complication, however. During the subperiosteal exposure of the inner table, the abdominal and iliacus fascial and muscular attachments are detached. This weakens the ability of these structures to retain abdominal contents. The reconstruction technique described closes the defect left after harvest. Meticulous technique should be used in closing the fascia.

An improper incision at the harvesting site may cause neurologic problems, with injury to the LFCN. This complication can be avoided by making the incision medial or lateral to the crest and ensuring proper exposure of all structures. The lateral incision is preferred.

WWW.HIGHDENT.IR

همیار دندانسازان

Figure 40-20 **A,** A panoramic radiograph demonstrates a Division D maxilla opposing natural dentition. Two implants supporting an overdenture in the maxilla are fractured. **B,** Bilateral sinus grafts are performed before the hip graft. The panoramic radiograph demonstrates the bilateral grafts, the two implants are removed, and dense hydroxyapatite is positioned in the premaxilla to begin stretching the mucosa. **C,** A panoramic radiograph of an iliac crest bone graft fixated to the maxilla. **D,** Six months later, 11 BioHorizons Maestro implants are inserted into the maxillary bone graft with a one-stage approach. **E,** After 6 months the abutments are positioned into the integrated implants. **F,** A porcelain-metal fixed prosthesis is cemented onto the maxillary implants. **G,** A panoramic radiograph of the maxillary fixed restoration supported by 11 implants in iliac grafted bone.

Tibial Bone Grafts

The proximal tibial metaphysis provides an excellent source of trabecular bone for grafting.[103,104] This donor site offers a small cortical segment approximately 5 to 10 mm in diameter, 2 mm thick, and 10 to 40 mL of trabecular bone with low reported morbidity.[104-107] However, this area is contraindicated in children and adolescents because possible disruption of the epiphyseal growth center may occur.[104] In addition, the fat content of the marrow is sometimes greater than that found in the ilium.[108,109]

The tibial bone graft harvest is primarily trabecular bone and is therefore primarily used with a barrier membrane and GBR procedure. The layered approach

WWW.HIGHDENT.IR

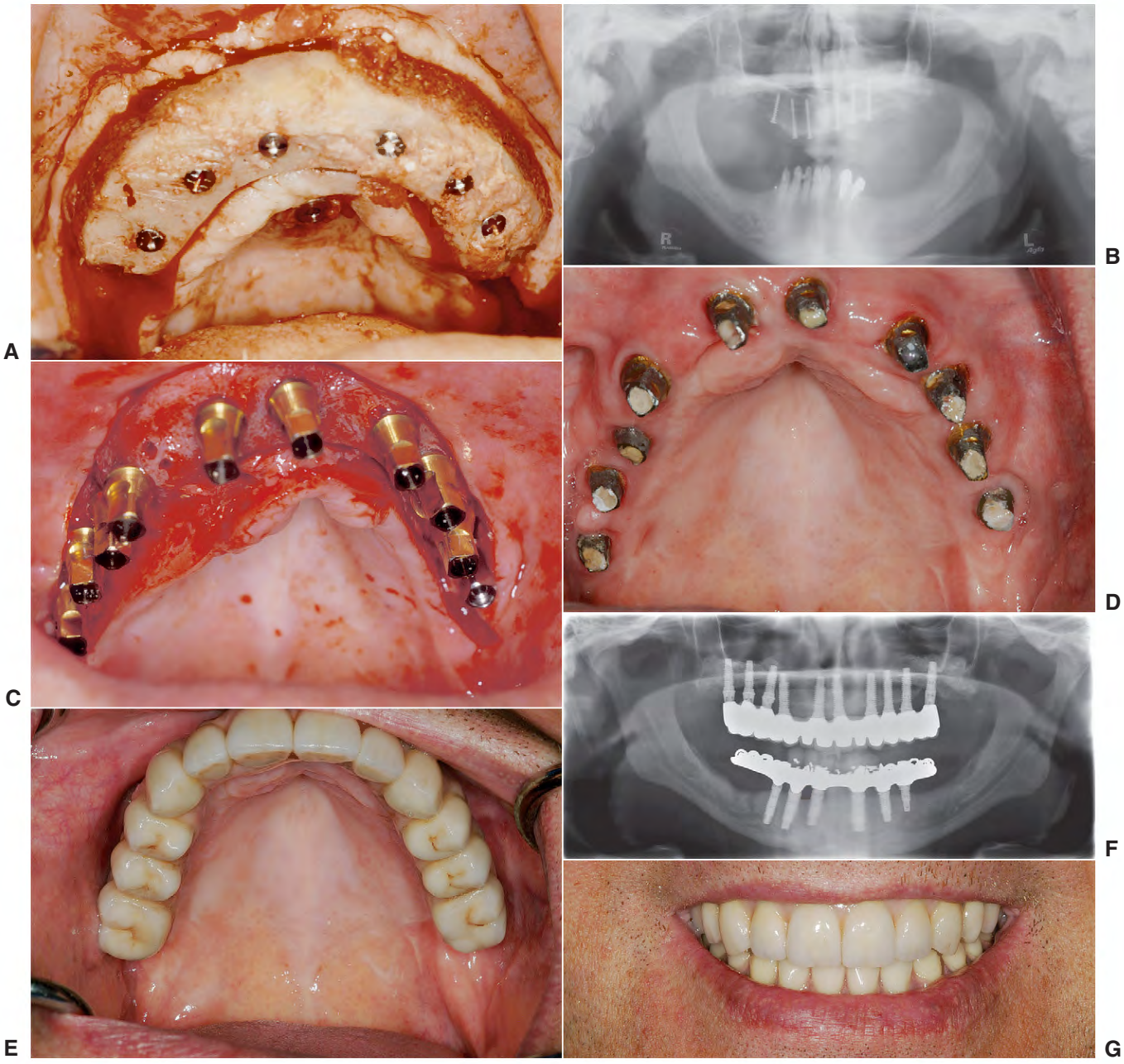


Figure 40-21 **A,** An iliac crest bone graft is fixated to a Division D maxilla with 2-mm-diameter fixation screws. **B,** A postoperative panoramic radiograph of the iliac crest bone graft fixated to the maxilla. **C,** Six months later, 10 BioHorizons Maestro implants are inserted into the bone block. **D,** After 6 months the implant abutments are prepared. **E,** The occlusal view of the maxillary fixed prosthesis cemented onto 10 implants. **F,** A panoramic radiograph of the 10 implants and fixed prosthesis in the maxilla. Six implants were also placed and restored in the mandible. **G,** A high smile line of the previous patient, with the final maxillary and mandibular fixed prostheses.

to GBR is addressed in Chapter 37. When ramus and/or symphysis donor sites do not provide sufficient volume, the tibia graft is an excellent source of trabecular bone.

The preparation of the recipient bed is similar to other grafting procedures, with proper aseptic preparation and skin disinfection with a chlorhexidine scrub. The surgeon gains access to the tibial plateau by a 2- to 3-cm oblique incision through the skin on the anterolateral aspect of the leg directly over Gerdy's tubercle, which is lateral to the tuberosity. Catone et al.[104] describe the incision as angled, with its cephalic limit superior and medial to the tibialis anterior muscle origin (below the articular surface of the tibial plateau) and extending lateral to the patellar ligament. Bleeding is controlled by electrocautery or preoperative placement of a tourniquet. After incising through the subcutaneous and fascial layers of the iliotibial tract, the periosteum is reflected to expose the cortex of the tibial metaphysis. The outer cortical bone and core of cancellous bone may be removed with a large 8- to 10-mm trephine bur

WWW.HIGHDENT.IR

همیار دندانسازان

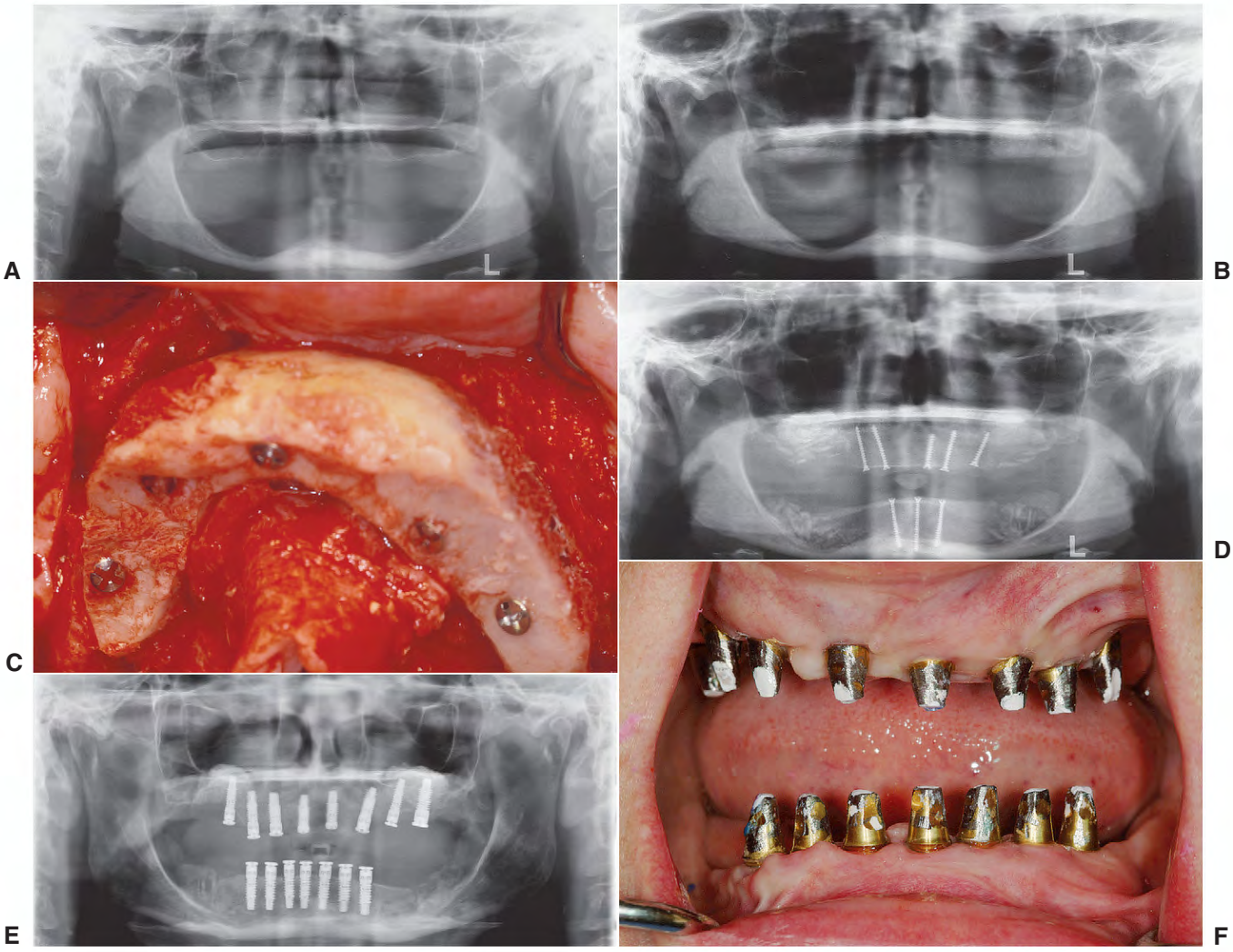


Figure 40-22 **A,** A panoramic radiograph of a Division D maxilla and mandible. **B,** A postoperative radiograph after bilateral sinus grafts. **C,** An intraoral view of the tricortical block graft fixated into resorbed maxilla. **D,** A postoperative radiograph of a tricortical block graft fixated to the maxilla and mandible. **E,** Six months later, eight maxillary implants are inserted and seven mandibular implants are inserted. All 15 implants have a one-stage surgical approach. **F,** After 6 months, abutments are inserted into the maxillary and mandibular implants and prepared for the final restoration. *Continued*

under copious sterile saline. On removal of the block, additional cancellous bone can be harvested with orthopedic curettes. An alternative approach is the use of a hollow, cylindrical, hand-driven Wagner instrument to procure rods of cancellous bone.[107] In most cases the cortical window is used in the oral reconstruction and is not replaced in the donor site. No drain is usually needed, and no fill of the donor site is necessary. Sometimes a hemostatic such as collagen may be inserted to assist in hemostasis.

The tibial wound is approximated in layers, with a resorbable suture and closed using a continuous subcuticular suture or staples. Antibiotic ointment is applied, and a pressure dressing is maintained for 48 hours. Patients may begin ambulation on the donor leg after the procedure but are advised to avoid full load bearing for several days. Possible complications reported are hematoma, postoperative pain, infections, and dehiscence, with incidence ranging from 1% to 4%[105] (Figures 40-23 and 40-24).

Division E

Discontinuity defects of the jaw (Division E) often require use of autogenous bone grafts to restore a patient to normal contour and function. Mandibular reconstruction with various types of materials has been attempted since the early 1800s, including allogenic, alloplastic, and autogenous materials.[110]

The cause of the discontinuity may be from trauma, disease, surgery, or genetics. Pathologic causes for discontinuity may include many conditions, of which carcinomas are the most common. The most common site for intraoral carcinomas is the lateral aspect of the tongue (20%); this affects mainly male patients. Carcinomas of the floor of the mouth account for 10% to 15% of all intraoral malignancies. Alcohol and tobacco

WWW.HIGHDENT.IR

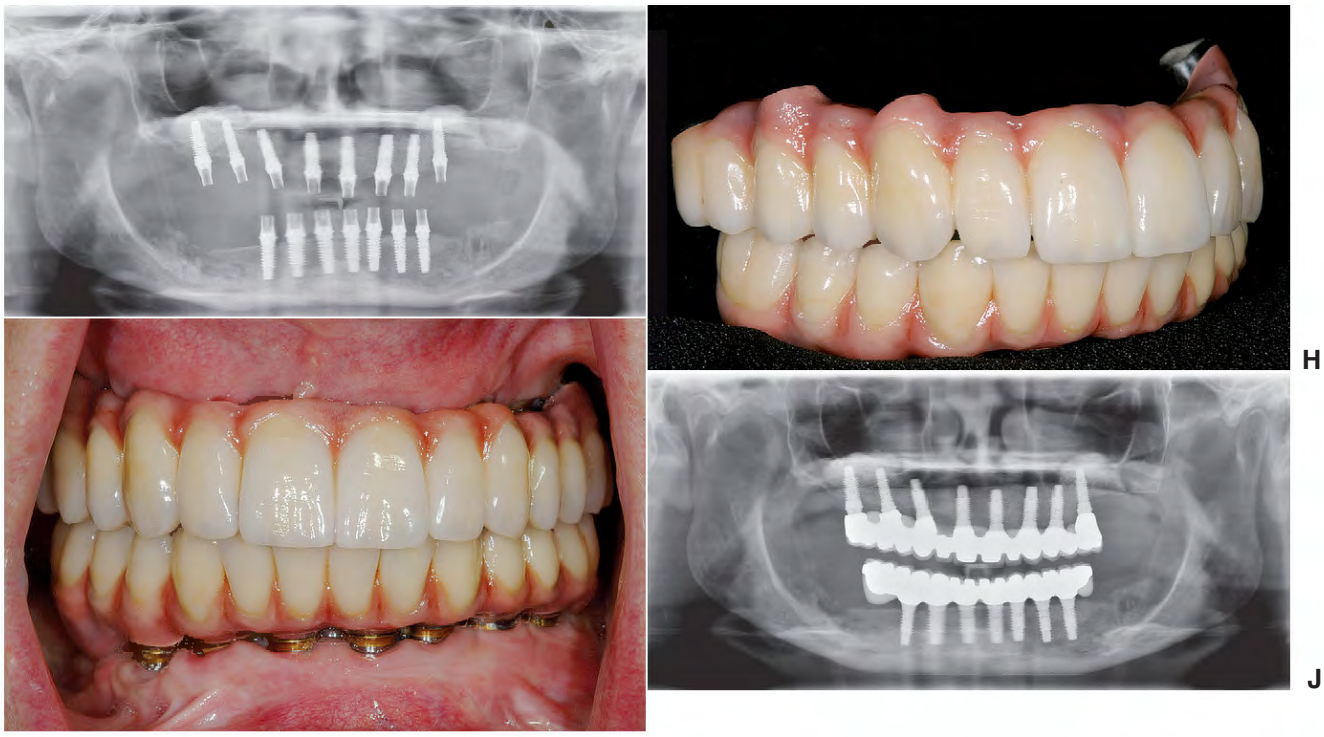


Figure 40-22, cont'd **G,** A panoramic radiograph of the eight maxillary and seven mandibular implants. **H,** The maxillary and mandibular fixed restorations. **I,** The maxillary and mandibular porcelain-metal fixed restorations in centric relation occlusion. **J,** A panoramic radiograph of the iliac crest bone grafts to the maxilla and mandible, implants inserted in both arches, and the final restoration.

abuse are the main causative factors. The prognosis depends greatly on the size of the tumor, location, extent, and differentiation. Treatment is accomplished by surgical removal of the lesion, radiation therapy, and/or combined chemotherapy. The surgical excision of the lesion often requires extensive resection of the floor of the mouth, variable portions of the mandible, and regional lymph nodes. Preoperative radiation therapy increases postoperative complications, with infection being the most common. Nerve damage with eventual shoulder drop, hemorrhage, and necrosis of the flaps are also possible complications. The patient is left with severe physiologic, cosmetic, and psychological deficits. The remaining mandibular segment is retruded, then deviated medially and upward. The deviation is often increased during opening by as much as 1 or 2 cm. The loss of mandibular continuity impairs mastication, deglutition, phonation, and respiration. The patient presents a concave facial asymmetry because of the loss of tissues and the adherence of the skin to the deep tissues of the neck. The deviation and partial loss of innervation around the commissures result in drooling of saliva.[37] Optimum treatment of mandibulectomy patients is achieved with coordinated surgical, prosthodontic, and speech therapies. The decision to reconstruct the mandible immediately or in a second phase depends on the amount and character of the remaining tissues (more than 5000 cGy of irradiation reduces the healing capacity of hard and soft tissue), the prognosis of the treatment, the age and general health of the patient, and whether any residual tumor remains.[111-115]

The risk of osteoradionecrosis (ORN) is higher in the presence of a soft tissue bed of poor quality (i.e., hypoxic, hypovascular, hypocellular).[113-115] Surgical reconstruction of the mandible with large defects may also require replacement of soft and hard tissue. Additional soft tissue to cover the defect may use myocutaneous flaps combined with hyperbaric oxygen and may be indicated in patients to induce angiogenesis and fibroblastic activity.[115]

Vascular Bone Grafts

Facial deformities caused by ablative surgical techniques have long been a challenge for the reconstructive surgeon and prosthodontist. Advances in microvascular surgical techniques using composite vascularized grafts have dramatically increased the predictability of jaw discontinuity reconstruction. Vascularized bone grafts are more often indicated when blood supply is severely compromised to the graft site or when the recipient bed is scarred. The most common conditions are the patient with cancer who has undergone radiation therapy or the Division E bone anatomy. Vascularized iliac crest grafts have been used with increasing frequency for mandibular reconstruction, because this area provides a large quantity of corticocancellous bone with favorable contours.[116-120] An ilium microvascular graft consists of

WWW.HIGHDENT.IR

همیار دندانسازان

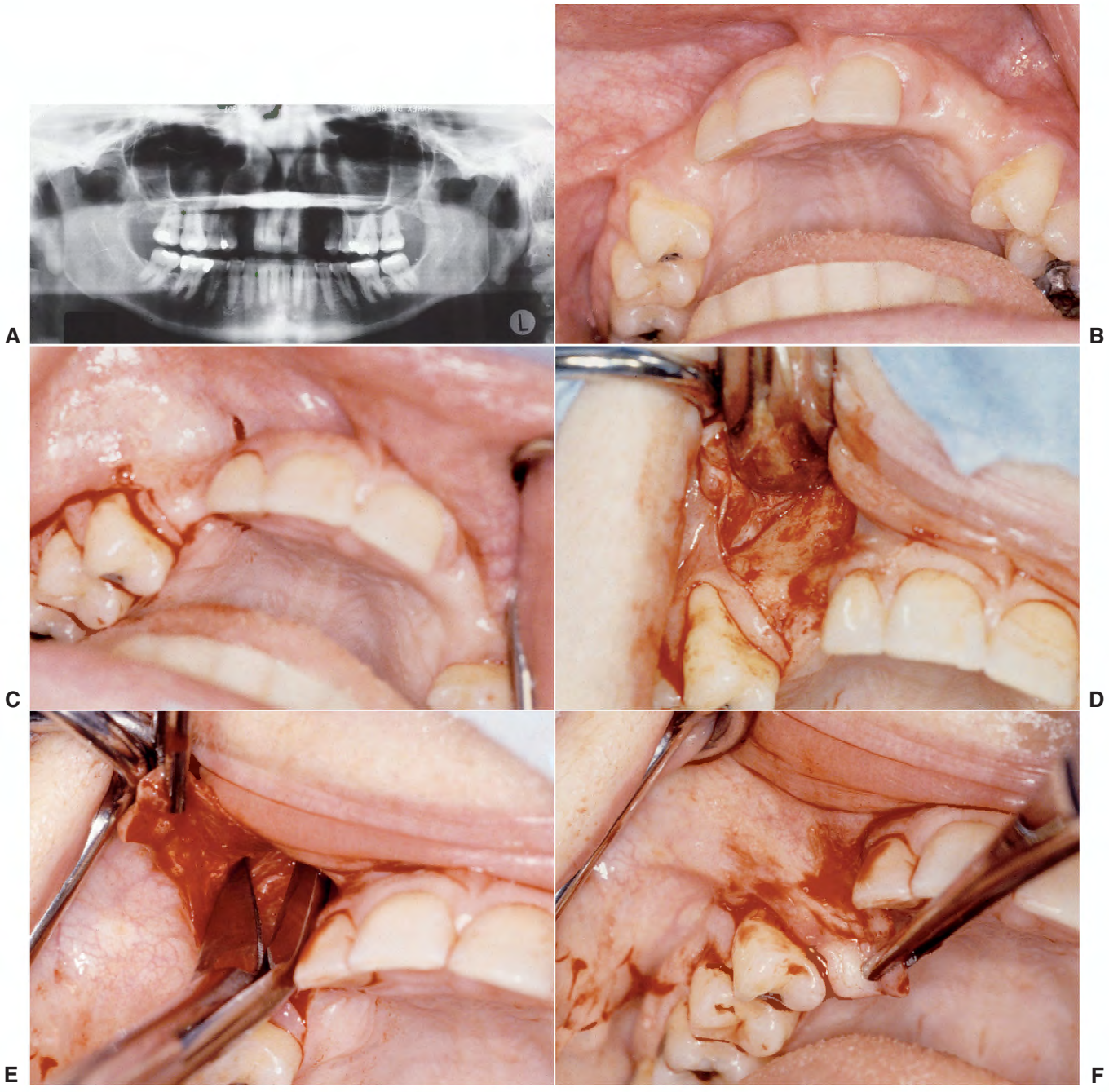


Figure 40-23 **A,** A panoramic radiograph of a partially edentulous patient in the maxilla. **B,** An intraoral view of the patient missing a maxillary right canine and left lateral and canine. **C,** Incision is made with papilla-saving vertical release incisions. **D,** A full-thickness flap is reflected, exposing the lateral bone in the canine site. **E,** Soft tissue scissors are pushed into the facial flap, 3 mm below the tissue. The tissue scissors are then opened, and a blunt dissection creates a submucosal space. **F,** The facial flap is released to obtain primary closure. *Continued*

a portion of the iliacus and gluteus medius muscles, the anterior and medial aspect of the iliac crest, and the deep circumflex iliac artery and variable veins. More recently, the fibula has emerged as the free-tissue transfer of choice in many reconstructive centers for restoring composite mandibular defects. Its proponents report a greater total thickness of cortical bone and high implant survival rates.[121,122] The application of osseointegrated implants in these grafts has significantly improved the outcome of prosthetic rehabilitation.[112,120,122-128]

In contrast to nonvascularized free bone grafts, bone successfully transferred by microvascular surgery remains viable without cortical or trabecular bone necrosis.[129,130] Therefore the advantage of the vascularized bone graft

WWW.HIGHDENT.IR

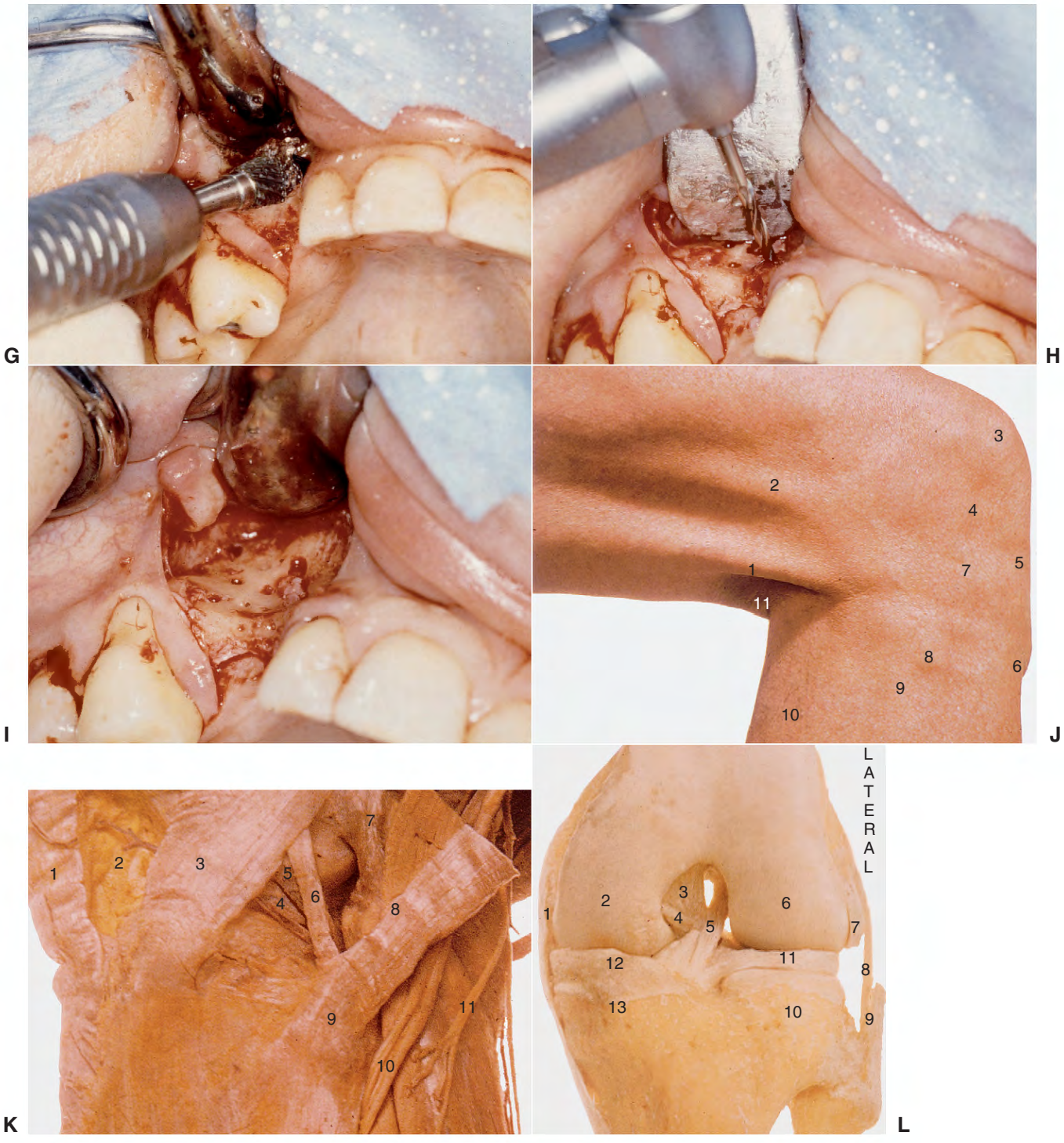


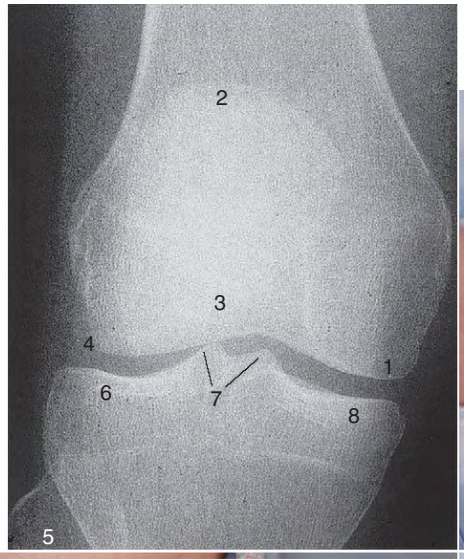
Figure 40-23, cont'd **G,** A carbide bur prepares the host bone surface. **H,** A small-diameter drill is used to decorticate the facial plate of bone. **I,** The canine site has been prepared for a barrier membrane bone graft. **J,** The right lateral knee site: *6* is the tibial tuberosity site, and *8* is the lateral head of the tibia. **K,** Under the fascia is the iliotibial tract, which connects to Gerdy's tubercle of the tibia. **L,** The tibial plateau is most superior, and Gerdy's tubercle is below the plateau and lateral to the tuberosity.

Continued

is that it maintains normal physiologic function. The improved potential for incorporation of a vascularized bone graft indicates its use in compromised regions, such as postirradiation sites. The success of implants placed in healed vascularized grafts for mandibular reconstruction appears similar to their success in non-grafted bone sites.[131-136] The simultaneous placement of implants with microvascular bone flap reconstruction has shown an approximately 80% success rate using Ti implants with a short follow-up. Often difficulty exists in attaining primary implant stability because the quality of iliac free vascular grafts is often very

WWW.HIGHDENT.IR

همیار دندانسازان



M

N

O

P

Q

R

Figure 40-23, cont'd **M,** A radiograph of the head of the tibia may assess the thickness of the cortical plate. **N,** The right leg is prepared for surgery. A surgical marker outlines the patella, the tuberosity, and the lateral aspect of the tibia. An oblique line is drawn over Gerdy's tubercle, in the direction of the iliotibial tract. An anesthetic needle is used to anesthetize and palpate the underlying bone. **O,** A No. 15 scalpel incises the skin along the iliotibial tract. A layer of fat is not unusual below the skin. **P,** The iliotibial tract is identified, and an incision is made parallel to its fibers. **Q,** The periosteum is reflected, and a self-retaining retractor is positioned. **R,** The osteotomy is parallel to the head of the tibia and over Gerdy's tubercle.

Continued

spongeous with a thin cortical layer.[136] It is more advantageous to allow the graft to mature before implant placement, because for these patients, it is more difficult to determine the prosthetic support needs at the time of bone grafting.

Although free microvascular grafts from the fibula and ilium are popular among many reconstructive surgeons, Marx and Morales[137] pointed out that these grafts have several limitations from a dental perspective. The fibula is straight and limited in height, which

WWW.HIGHDENT.IR

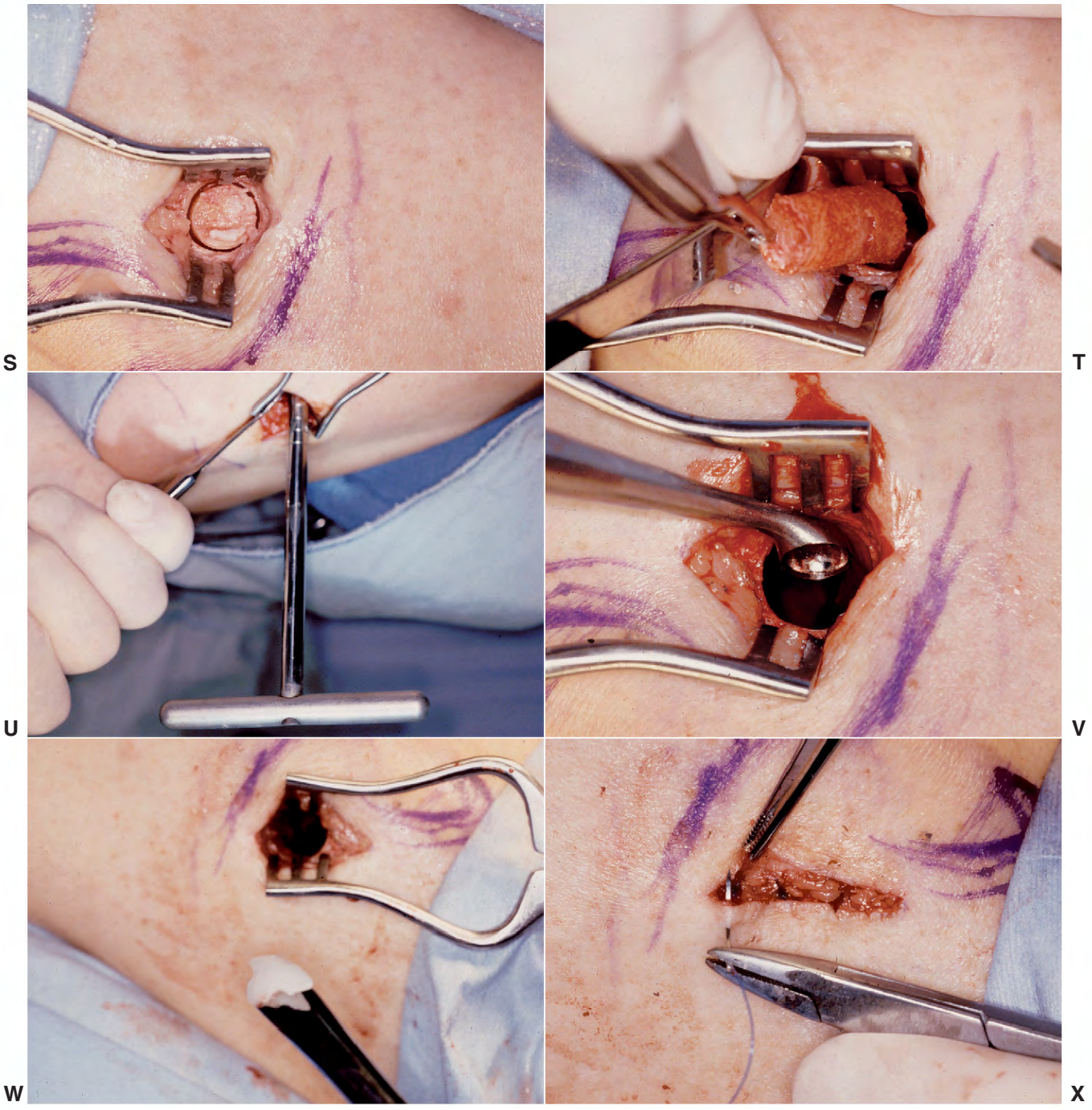


Figure 40-23, cont'd **S,** The bone core is visualized. **T,** The bone core is removed from the tibia. **U,** The hand trocher, 8 mm in diameter, harvests trabecular bone through the initial osteotomy site. **V,** A bone curette is used to scoop additional trabecular bone. **W,** A collagen sponge and antibiotic may be placed into the donor site. **X,** Sutures are used to close the wound in layers. Resorbable sutures should be used for the first layer. The skin is approximated with subcuticular sutures.

Continued

compromises arch form. Marx has shown impressive results in the treatment of discontinuity defects, with the use of cancellous marrow grafts from the ilium supported with growth factor additions.[138]

The scapula is also a source of bone in microvascular grafts for jaw reconstruction. Teot et al.[139] and Swartz et al.[140] reported on the potential of the lateral scapular border with blood supply by branches of the circumflex scapular artery.[124,141] The scapula allows the design of multiple cutaneous panels on separate vascular pedicles to facilitate three-dimensional (3D) reconstruction. Complications are those common to the microvascular grafts and restricted elevation for the arm if scarring occurs. Once the vascularized grafts are healed, the soft tissue and bone may be receptive for endosteal implants after the usual protocol based on prosthesis type, amount of available bone, and density of bone.

WWW.HIGHDENT.IR

همیار دندانسازان

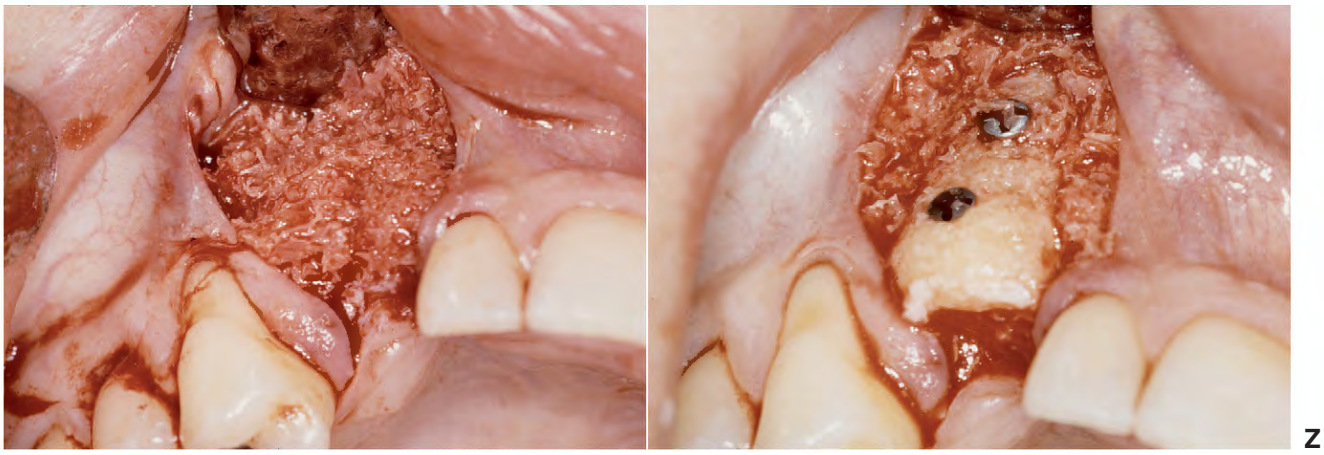


Figure 40-23, cont'd **Y,** The trabecular bone is placed over the host site. **Z,** The cortical bone of the tibia may be secured with fixation screws.

Figure 40-24 **A,** Platelet-rich plasma (PRP) is placed over the graft site. **B,** The three-tooth edentulous site is restored with trabecular donor bone from a tibia. **C,** PRP is placed over the trabecular bone. **D,** A barrier membrane is placed over the graft site. *Continued*

WWW.HIGHDENT.IR

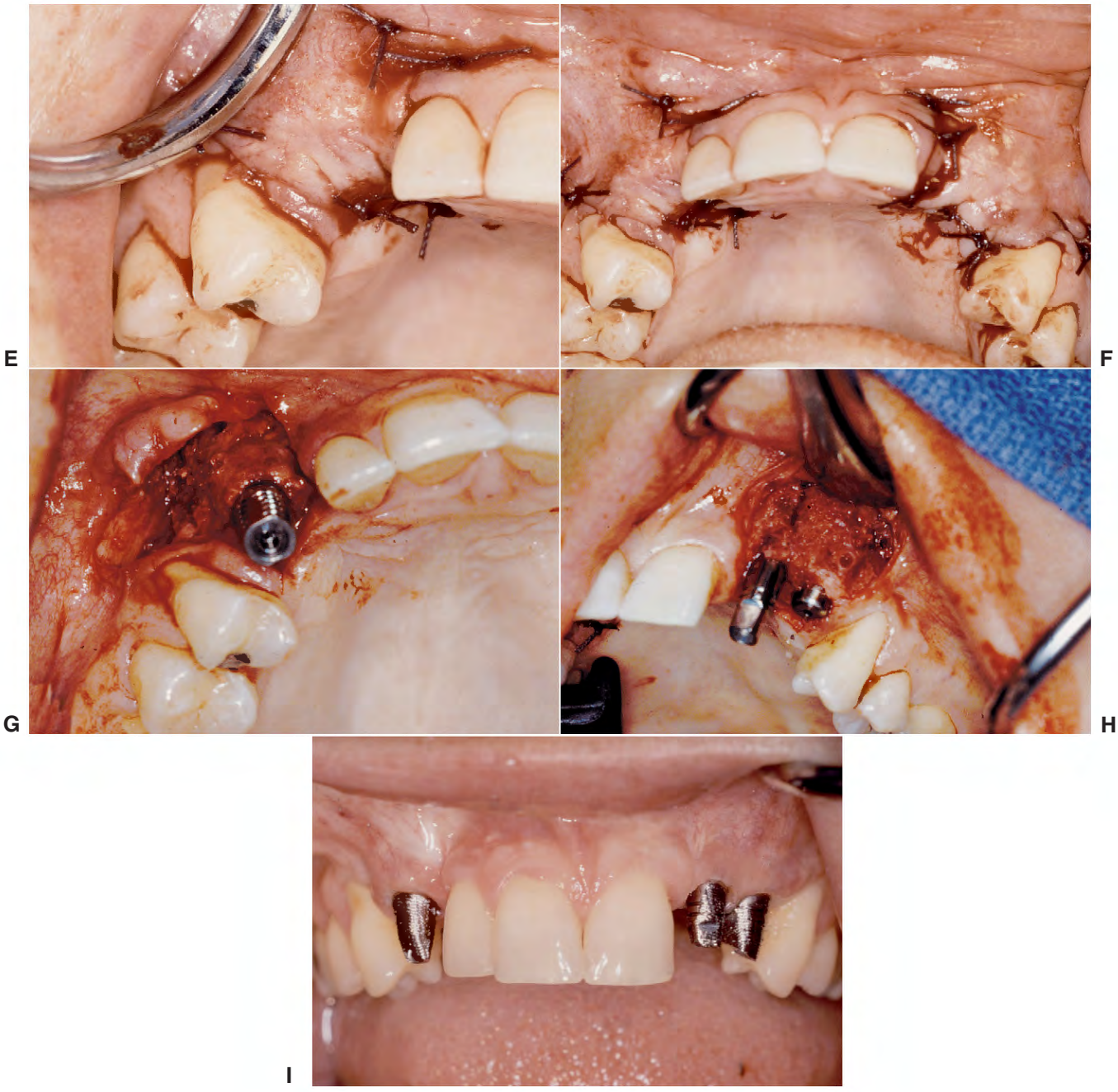


Figure 40-24, cont'd **E,** The soft tissues are approximated for primary closure without tension. **F,** The right and left sites were grafted with tibial donor bone. **G,** After 7 months, an implant is placed into the maxillary right canine region. **H,** Two implants are placed into the left maxillary edentulous site. **I,** After 4 months the implants are uncovered; abutments are inserted and prepared for restoration.

Distraction Osteogenesis

Distraction osteogenesis refers to the formation of new bone between vascular bone surfaces created by an osteotomy and separated by gradual distraction.[142] Ilizarov[143] developed the technique for the management of musculoskeletal conditions, such as posttraumatic defects and deformed limbs that demonstrated induction of bone formation between osseous segments as they were pulled apart. Distraction osteogenesis has also been applied to the craniofacial skeleton. It has been used in the repair of continuity defects, mandibular lengthening, and maxillary advancement. Recently research has focused on the use of distraction osteogenesis for alveolar ridge augmentation and implant placement.[144-150]

The concurrent soft tissue neogenesis and stretching of mucosa that accompany the osseous distraction are unique characteristics that may allow augmentation in more challenging clinical situations.[151] For instance, soft tissue integrity is often a limiting factor in bone grafting for implant placement, especially in areas such as the posterior mandible.

WWW.HIGHDENT.IR

همیار دندانسازان

Complications: Receptor Site

The general causes of failure for autogenous grafts or of the donor site are variable and well documented. The primary cause of failure of bone grafts in conjunction with endosteal implants include improper diagnosis, treatment planning, and/or sequencing; poor soft tissue management; too many implants placed at surgery in grafted bone only; improper implant position at surgery; and esthetic requirements ignored during augmentation. Improper diagnoses include sinus pathologic condition before sinus grafting, periapical pathologic condition, or advanced periodontal disease, which may compromise the graft as a result of infection, as well as systemic or local disease, which compromises hard and soft tissue healing.

Improper treatment planning includes an attempt to restore a patient to an FP-1 prosthesis when the intraoral condition is Division D. These patients may receive a fixed restoration after the autogenous graft, if adequate amounts of bone, soft tissue coverage at surgery, and bone modeling occur, but a natural-appearing prosthesis cannot be promised. Patients requesting this specific result are prone to unrealistic expectations. Poor treatment plans also include too few and too short implants to support the final prosthesis. For example, a maxillary fixed prosthesis almost always opposes a fixed restoration or natural teeth. The moment forces are greater, the esthetic demands are greater, the bone density is poorer, and the SA augmentations are more critical to success. To obtain more than 95% implant and prosthetic survival in the grafted maxilla, eight to 10 implants may be required, with limited posterior cantilever of the prosthesis.

Poor treatment sequencing may include attempting to place all the implants at the time of the autogenous graft. Division C–h and most Division D mandibles are best treated and have fewer complications with only screws for fixation of the block graft. Division C–w atrophic conditions usually have fixation of a block of bone with a lateral screw or a particulate graft and barrier membrane with a tunnel approach and no implant placement. Only implants that can be positioned without compromise to the position, angulation, contour, hygiene considerations, and direction of load for ideal conditions should be placed at the graft surgery. The more implants placed at the time of the graft, the greater the risk for incorrect placement of implants, mandibular fracture, and splitting of the graft block. The loss of one implant may spread to two or three, and the risk of prosthetic compromise dramatically increases. In addition, the progressive bone-loading concepts used during prosthetic reconstruction are very important in patients with autogenous augmentation.

The primary postoperative complication is wound dehiscence that is directly related to implant failure, thus the decision by the authors to delay implant placement. The treatment is similar to procedures in the intraoral donor graft chapter (see Chapter 39).

Careful closure without tension is paramount to avoid incision line opening. It usually necessitates periosteal release to ensure that the soft tissue passively drapes over the graft. In addition, the fabrication of a transitional prosthesis without pressure is a mandatory step in the prevention of wound dehiscence. The patient should be thoroughly educated to wear the transitional prosthesis as little as necessary. A maxillary denture with no lingual flange and/or the lingual acrylic cut out completely (leaving only a row of teeth) over the grafted area and with contact only on the hard palate and tuberosities may be worn only when necessary. A modified night guard appliance adjusted to lower anterior teeth and with bite blocks in the tuberosities may also be used when lower anterior teeth are present.

Other postoperative complications similar to other implant procedures include postoperative infection, loss of a portion of the graft, dehiscence, pain, and sinusitis. Poor soft tissue management has the most devastating effect during the immediate postoperative phase of the autogenous graft and is reported most often. Improper flap design may result in compromised blood supply to the underlying graft. Trauma from the opposing teeth, or excessive tension on the incision line, results in the incision line opening and graft exposure with loss of significant height and/or width of bone. Soft tissue management also includes stage II uncovery of the implants. Adequate width of attached gingiva of minimum thickness is the soft tissue interface of choice around the implants. Autogenous tissue grafts and gingivoplasty techniques are commonly required for graft patients.

A Division D mandible is often less than 7 mm in width. An implant placed through the block graft and into the underlying host bone may not go through the center of this atrophic ridge. Instead, the osteotomy may be off center and have removed all the lateral cortical plate and one half the inferior plate at the implant site. When the implant is threaded into position, or shortly thereafter, the mandible may fracture.[73,74] This compromises the rigidity of the implant graft and the mandibular bony healing. This complication may be reduced by using 2-mm fixation screws, rather than implants, to stabilize the graft.

The primary reason to place screws in conjunction with the graft is to ensure the stability of the grafted bone. Wires may stretch and allow graft movement during healing. In addition, they may erode the graft on the surface regions where they are tied with pressure. Most wires are stainless steel, and fibrous tissue often forms at the interface. As a result, a fibrous tissue trail may form through the bone and result in soft tissue next to the implant placed in the site. Fixation screws rigidly position and maintain the block. In addition,

WWW.HIGHDENT.IR

they may be inserted at any angle or location, which allows more flexibility at the time of surgery to secure the bone block.

When implants are placed to stabilize the graft, they should be thoroughly evaluated when the additional implants are placed 6 months later. If an implant-bone defect exists, then the area may be repaired with autogenous bone. Another option is to remove the implant and reinsert another one deeper into position, or the implant can be removed and another one placed in a different site.

The patient requiring autogenous grafts is usually compromised from the lack of soft tissue support. The maxilla resorbs toward the medial aspect in the posterior and anterior regions. The mandible resorbs toward the lateral in the Division C–h and D conditions. As a consequence, the autogenous grafts should be placed in position for the required esthetic result, not the current position of the atrophic bone. This requires maxillary grafts to be cantilevered to the facial plane, or the surgeon performs a Le Fort osteotomy in conjunction with the interpositional graft placement. Mandibular grafts should be placed with a lingual inclination but with implants at a more vertical trajectory.

SUMMARY

The use of autogenous grafts for or in conjunction with implants has been reported for more than 20 years. During this period, predictable techniques and refinements have occurred. The grafting procedures may be indicated for the moderately resorbed jaw with demanding prosthetic requirements. The autogenous grafts are usually indicated for severe resorption, to improve anatomical conditions for long-term survival of implants and the related prosthesis. The Misch-Judy bone classification may be used as a diagnostic guide for surgical approach and prosthetic concerns.[33a] An additional Division E is provided for discontinuity defects of the jaws. This chapter addresses these indications, survival rates, and failures of autogenous grafts in conjunction with endosteal implants.

References

1. Misch CE, Zaki H, Dietsh F: Osteointegrated implants in a microvascular graft to restore a subtotal mandibulectomy case report, *Int J Oral Implantol* 7:25-29, 1990.
2. Brånemark PI, Breine U, Hallen O et al: Repair of defects in the mandible, *Scand J Plast Reconstr Surg* 4:100-108, 1970.
3. Breine U, Brånemark PI: Reconstruction of alveolar jaw bone, *Scand J Plast Reconstr Surg* 14:23-48, 1980.
4. Misch CE, Dietsh F: Autogenous bone grafts for endosteal implants, indications and failures, *Int J Oral Implantol* 8:13-20, 1991.
5. Misch CE, Dietsh F: Endosteal implants and iliac crest grafts to restore severely resorbed, totally edentulous maxillae—a retrospective study, *J Oral Implantol* 20:100-110, 1994.
6. Tolman DE: Reconstructive procedures with endosseous implants in grafted bone—a review of the literature, *Int J Oral Maxillofac Implants* 10:275-294, 1995.
7. Chiapasco M, Abati S, Ramundo G et al: Behavior of implants in bone grafts or free flaps after tumor resection, *Clin Oral Implants Res* 11:66-75, 2000.
8. Verhoeven JW, Cune MS, Ruijter J: Permucosal implants combined with iliac crest onlay grafts used in extreme atrophy of the mandible: long term results of a prospective study, *Clin Oral Implants Res* 17:58-66, 2006.
9. Clayman L: Implant reconstruction of the bone-grafted maxilla: review of the literature and presentation of 8 cases, *J Oral Maxillofac Surg* 64:674-682, 2006.
10. Sjostrom M, Sennerby I, Milson H et al: Reconstruction of the atrophic edentulous maxilla with free iliac crest grafts and implants: a 3-year report of a prospective clinical study, *Clin Implant Dent Relat Res* 9:46-59, 2007.
11. Schultes G, Gaggl A, Karcher H: Stability of dental implants in microvascular osseous transplants, *Plast Reconstr Surg* 109:916-921, 2002.
12. Raghoebar GM, Schoen P, Meijer IIJ et al: Early loading of endosseous implants in the augmented maxilla: a 1-year prospective study, *Clin Oral Implants Res* 14:697-702, 2003.
13. Van der Meij EH, Blankestijn J, Berns RM et al: The combined use of two endosteal implants and iliac crest onlay grafts in the severely atrophic mandible by a modified surgical approach, *Int J Oral Maxillofac Surg* 34:152-157, 2005.
14. Umstadt HH, Vollinger J, Muller HH et al: Implants in avascular iliac crest bone transplants. Prospective study of 176 implants, *Mund Kiefer Gesichtschir* 3:S93-S98, 1999.
15. Stellingsma C, Raghoebar GM, Meijer HJ et al: Reconstruction of the extremely resorbed mandible with interposed bone grafts and placement of endosseous implants: a preliminary report on outcome of treatment and patients' satisfaction, *Br J Oral Maxillofac Surg* 36:290-295, 1998.
16. Verhoeven JW, Cune MS, Teriou M et al: The combined use of endosteal implants and iliac crest onlay grafts in the severely atrophic mandible: a longitudinal study, *Int J Oral Maxillofac Surg* 26:351-357, 1997.
17. Dahl GSA: Om Molijhoten fur Implantation: karen au meitalskett som bas eller fur fasta eller autagbara proteser, *Odontol Tidskr* 51:440, 1943.
18. Judy KWM, Misch CE: Evolution of the mandibular subperiosteal implant, *N Y J Dent* 53:9-11, 1983.
19. Cram DL, Roberts HD, Baum R: Ramus endosseous frame implant for use with patient's dentures, *J Am Dent Assoc* 84:156-162, 1972.
20. Linkow LI: The pterygoid extension implant for the totally and partially edentulous maxillae, *Dent Concepts* 12:17-28, 1973.
21. Tulasne JF: Implant treatment of missing posterior dentition. In Zarb G, Albrektsson T, editors: *The Brånemark implant*, Chicago, 1989, Quintessence.
22. Blackstone CH, Parker ML: Rebuilding the residual alveolar ridge, *J Oral Surg* 14:45, 1965.
23. Converse JM: *Reconstructive plastic surgery, principles and procedures in correction, reconstruction and transplantation*, ed 2, Philadelphia, 1977, WB Saunders.

WWW.HIGHDENT.IR

همیار دندانسازان

24. Jackson IT, Helden G, Marx R: Skull bone graft in maxillofacial and craniofacial surgery, *J Oral Maxillofac Surg* 44:949-955, 1986.
25. Tessier P: Autogenous bone grafts taken from the calvarium for facial and cranial applications, *Clin Plast Surg* 9:531, 1982.
26. Wang J, Waite D, Steinhauser E: Ridge augmentation: an evaluation and follow-up report, *J Oral Surg* 34:66-602, 1976.
27. Masella R, Mercier P: Surgical and prosthodontic reconstruction of the severely handicapped edentulous patient, *J Prosthet Dent* 50:377-383, 1983.
28. Davis WH, Martinoff JT, Kaminski RM: Long term follow up of transoral rib grafts for mandibular atrophy, *J Oral Maxillofac Surg* 42:606-609, 1984.
29. Curtis T, Ware W, Beirne OR et al: Autogenous bone grafts for atrophic edentulous mandibles, a final report, *J Prosthet Dent* 57:73-78, 1987.
30. Kratochvil FJ, Boyne PJ: The combined use of a subperiosteal implant and bone marrow grafts in deficient mandibles, *J Prosthet Dent* 27:645-653, 1972.
31. Malchiodi L, Quaranta A, D'Addona A et al: Jaw reconstruction with grafted autologous bone: early insertion of osseointegrated implants and early prosthetic loading, *J Oral Maxillofac Surg* 64:1190-1198, 2006.
32. Misch CE: Maxillary sinus augmentation for endosteal implants: organized alternative treatment plans, *Int J Oral Implantol* 4:49-58, 1987.
33. Consensus statement, Academy of Osseointegration Sinus Graft Consensus Conference, The Center for Executive Education, Wellesley, Mass, November 1996, Babson College.

33a. Misch CE: Divisions of available bone in implant dentistry, *Int J Oral Implantol* 7:9-17, 1990.

33b. Misch CE: Prosthetic options in implant dentistry, *Int J Oral Implantol* 7: 17-21, 1991.

34. Evans CE, Galasko CSB, Ward C: Effect of donor age on the growth of in vitro cells obtained from human trabecular bone, *J Orthop Res* 8:234-237, 1990.
35. Marx RE: Biology of bone grafts, *Oral Maxillofac Surg Knowled Update* 1:RCN, 1-17, 1994.
36. Boyne PJ: Autogenous cancellous bone marrow transplants, *Clin Orthop Relat Res* 73:119-209, 1970.
37. Fonseca RJ, Davis WH: *Reconstructive preprosthetic oral and maxillofacial surgery,* Philadelphia, 1986, WB Saunders.
38. Shirota T, Ohno K, Motohashi M et al: Histologic and microradiologic comparison of block and particulate cancellous bone and marrow grafts in reconstructed mandibles being considered for dental implant placement, *J Oral Maxillofac Surg* 54:15-20, 1996.
39. Howes R, Bowness JM, Grotendorst GR et al: Platelet derived growth factors enhances demineralized bone matrix induced cartilage and bone formation, *Calcif Tissue Int* 42:34-38, 1988.
40. Tayapongsak P, O'Brien DA, Montero CB: Autologous fibrin adhesive in mandibular reconstruction with particulate cancellous bone and marrow, *J Oral Maxillofac Surg* 52:161-165, 1994.
41. Petersen GW: *Oral surgery,* Philadelphia, 1988, WB Saunders.
42. Misch CE, Dietsh F: Bone grafting materials in implant dentistry, *Implant Dent* 2:158-167, 1993.
43. Beumer J 3rd, Curtis TA, Firtell DN: *Maxillofacial rehabilitation, prosthetic and surgical considerations,* St Louis, 1979, Mosby–Year Book.
44. Listrom RD, Symington JM: Osseointegrated dental implants in conjunction with bone grafts, *J Oral Maxillofac Surg* 17:116-118, 1988.
45. Keller EE, Triplett WW: Iliac bone grafting, review of 160 consecutive cases, *J Oral Maxillofac Surg* 45:11-14, 1987.
46. Levy RN, Siffert RS: Inner table iliac bone graft, *Surg Gynecol Obstet* 128:705, 1969.
47. Hall MB, Smith RG: The medial approach for obtaining iliac bone, *J Oral Surg* 39:462-465, 1981.
48. Laurie SW, Kaban LB, Mulliken JB et al: Donor site morbidity after harvesting rib and iliac bone, *Plast Reconstr Surg* 73:933, 1984.
49. Leake PL: A new alloplastic tray for osseous contour defects, *J Oral Maxillofac Surg* 2:164, 1974.
50. Converse JM, Campbell RM: Bone grafts in surgery of the face, *Surg Clin North Am* 39:365, 1974.
51. Keathlay CJ: *Postoperative morbidity of iliac crest donor site in preprosthetic surgery.* Paper presented at the sixty-fourth annual meeting of the American Association of Oral and Maxillofacial Surgery, Boston, Oct 1988.
52. Swart JN, Allard RHB: Subperiosteal onlay augmentation of the mandible, a clinical and radiographic survey, *J Oral Maxillofac Surg* 43:183-187, 1985.
53. Hall MB: Marginal bone loss around Brånemark fixtures in bone grafts used for augmentation [abstract], *J Oral Maxillofac Surg* 48:117, 1990.
54. Nystrom E, Legrell PE, Forssell A et al: Combined use of bone grafts and implants in the severely resorbed maxilla. Postoperative evaluation by computed tomography, *Int J Oral Maxillofac Surg* 24:20-25, 1995.
55. Misch CE: Extraoral autogenous donor bone grafts for endosteal implants. In Misch CE, editor: *Contemporary implant dentistry*, ed 2, St Louis, 1999, Mosby.
56. Jensen J, Simonsen EK, Pedersen SS: Reconstruction of the severely resorbed maxilla with bone grafting and osseointegrated implants, a preliminary report, *J Oral Maxillofac Surg* 48:27-32, 1990.
57. Adell R, Lekholm U, Grondahl K et al: Reconstruction of severely resorbed edentulous maxillae using osseointegrated fixtures in immediate autogenous bone grafts, *Int J Oral Maxillofac Implants* 5:233-246, 1990.
58. Keller EE, Van Roeckel NB, Desjardins RP et al: Prosthetic surgical reconstruction of the severely resorbed maxilla with iliac bone grafting and tissue integrated prostheses, *Int J Oral Maxillofac Implants* 2:155-165, 1987.
59. Keller EE, Tolman DE: Mandibular ridge augmentation with simultaneous onlay iliac bone graft and endosseous implants—a preliminary report, *Int J Oral Maxillofac Implants* 7:176-184, 1992.
60. Kahnberg KE, Nystrom E, Bartholdsson L: Combined use of bone grafts and Brånemark fixtures in the treatment of severely resorbed maxillae, *Int J Oral Maxillofac Implants* 4:297-304, 1989.
61. Nystrom E, Ahlquist J, Legrell PE et al: Onlay bone grafts and implants in the reconstruction of severely resorbed maxillae. In *Proceedings of the Seventh International Congress on Reconstructive Preprosthetic Surgery*, Copenhagen, Denmark, 1997.
62. Nystrom E, Ahlquist J, Kahnberg KE et al: Bone grafts and implants in the treatment of severely resorbed maxillae. Radiographic evaluation of preoperative bone dimensions, post-operative bone loss and changes in soft tissue profile, *Int J Oral Maxillofac Surg* 25:35, 1996.
63. Tidwell JK, Blijdorp PA, Stoelinga PTW et al: Composite grafting of the maxillary sinus for placement of endosteal

WWW.HIGHDENT.IR

implants: a preliminary report of 48 patients, *Int J Oral Maxillofac Surg* 21:204, 1992.
64. Triplett RG, Schow SR: Autologous bone grafts and endosseous implants. Complementary techniques, *J Oral Maxillofac Surg* 54:486-494, 1996.
65. Neyt L, DeClercq CAS, Abeloos JVS: Reconstruction of the severely resorbed maxilla with a combination of sinus augmentation, inlay bone grafting and implants, *J Oral Maxillofac Surg* 55:1397-1401, 1997.
66. Schliephake H, Neukam FW, Wichmann M: Survival analysis of endosseous implants in bone grafts used for the treatment of severe alveolar ridge atrophy, *J Oral Maxillofac Surg* 55:1227-1233, 1997.
67. Nystrom E, Kahnberg KE, Albrektsson T: Treatment of a severely resorbed maxilla with bone graft and titanium implants—histologic review of autopsy specimens, *Int J Oral Maxillofac Implants* 8:167-172, 1993.
68. Collins TA, Brown GK, Johnson N et al: Team management of atrophic edentulism with autogenous inlay, veneer and split grafts with endosseous implants. In Block MS, Kent JN, editors: *Endosseous implants for maxillofacial reconstruction*, Philadelphia, 1995, WB Saunders.
69. Keller EE, Tolman DE, Brånemark PI: Surgical reconstruction of advanced maxillary resorption with composite grafts (autogenous iliac bone and endosseous titanium implants). In Worthington P, Brånemark PI, editors: *Advanced osseointegration surgery: applications in the maxillofacial region*, Chicago, 1992, Quintessence.
70. Aparicio C, Brånemark PI, Keller EE et al: Reconstruction of the premaxilla with autogenous iliac bone in combination with osseointegrated implants, *Int J Oral Maxillofac Implants* 8:61-67, 1993.
71. lsaksson S, Alberius P: Maxillary alveolar ridge augmentation with onlay bone grafts and immediate endosseous implants, *J Craniomaxillofac Surg* 20:2-7, 1992.
72. Datillo D, Misch CM, Arena S: Interface analysis of coated implants in a human vascularized iliac bone graft, *Int J Oral Maxillofac Implants* 10:405-409, 1995.
73. Misch CE: *Extraoral bone grafts.* Lecture presented at the American College of Oral Implantology, Tarpon Springs, Fla, Jan 1998.
74. Schliephake H, Newkam FW: Bone replacement with porous hydroxyapatite blocks and titanium screw implants: an experimental study, *J Oral Maxillofac Surg* 49:151-156, 1991.
75. Wiltinger K: Orale Rehabilitation durch implantatfixierte autologe Beckenspantransplantate—eine Resorptionanalyse mit der Panoramaschichtaufname [dissertation], Freiburg, Germany, 1993. University of Freiburg.
76. Behairy YM, Al-Sebai W: A modified technique for harvesting full-thickness iliac crest bone graft, *Spine* 26:695-697, 2001.
77. Cocklin J: Autologous bone grafting—complications at the donor site, *J Bone Joint Surg* 53B:153, 1971.
78. Bosworth DM: Repair of herniae through iliac-crest defects, *J Bone Joint Surg Am* 37:1069-1073, 1955.
79. Challis JH, Lyttle JA, Stuart AE: Strangulated lumbar hernia and volvulus following removal of iliac crest bone graft, *Acta Orthop Scand* 46:230-233, 1975.
80. Lotem M, Maor P, Haimoff H et al: Lumbar hernia at an iliac bone graft donor site. A case report, *Clin Orthop Relat Res* 80:130-132, 1971.
81. Joshi A, Kostakis GC: An investigation of post-operative morbidity following iliac crest graft harvesting, *Br Dent J* 196:167-171, discussion 155, 2004.
82. Seiler JG 3rd, Johnson J: Illiac crest autogenous bone grafting: donor site complications, *J South Orthop Assoc* 9:91-97, 2000.
83. Resnick DK: Reconstruction of anterior iliac crest after bone graft harvest decreases pain: a randomized, controlled clinical trial, *Neurosurgery* 57:526-529, 2005.
84. Simmons EH, Bhalla SK: Anterior cervical discectomy and fusion. A clinical and biomechanical study with eight-year follow-up, *J Bone Joint Surg Br* 51:225-237, 1969.
85. Fowler BL, Call BE, Rowe DE: Complications associated with harvesting autogenous iliac bone graft, *Am J Orthop* 24:895-903, 1995.
86. Jones AA, Dougherty PJ, Sharkey NA et al: Iliac crest bone graft: osteotome versus saw, *Spine* 18:2048-2052, 1993.
87. Ghent WR: Further studies on meralgia paresthetica, *Can Med Assoc J* 14:85, 871-875, 1961.
88. Welkel AM, Habal MB: Meralgia paresthetica: a complication of iliac bone procurement, *Plast Reconstr Surg* 60:572-574, 1977.
89. Bents RT: Ilioinguinal neuralgia following iliac bone-grafting: report of two cases and review of the literature, *J Bone Joint Surg Am* 66:1306-1308, 1984.
90. Marx RE, Morales MJ: Morbidity from bone harvest in major jaw reconstruction: a randomized trial comparing the lateral anterior and the posterior approaches to the ilium, *J Oral Maxillofac Surg* 48:196, 1988.
91. Frost HM: The regional acceleratory phenomenon, a review, *Henry Ford Hosp Med J* 31:3-9, 1983.
92. Marx RE, Wong ME: A technique for the compression and carriage of autogenous bone during grafting procedures, *J Oral Maxillofac Surg* 45:988, 1987.
93. Marx RE, Snyder R, Kline SN: Cellular survival of human marrow during placement of marrow cancellous graft, *J Oral Surg* 37:712, 1979.
94. Tolman DE, Keller EE: Management of mandibular fractures in patients with endosseous implants, *Int J Oral Maxillofac Implants* 6:427-436, 1991.
95. Mason M, Triplett RG, Van Sickels JE et al: Mandibular fractures through endosseous cylinder implants: report of cases and reviwe, *J Oral Maxillofac Surg* 48:311-317, 1990.
96. Kennedy BD, Hiranaka DK: Use of a modified epidural catheter for analgesia after iliac crest bone procurement, *J Oral Maxillofac Surg* 53:342-343, 1995.
97. Al-Sayyad MJ, Abdulmajeed TM: Fracture of the anterior iliac crest following autogenous bone grafting, *Saudi Med J* 27:254-258, 2006.
98. Dawson EG, Lotysch M 3rd, Urist MR: Intertransverse process lumbar arthrodesis with autogenous bone graft, *Clin Orthop Relat Res* Jan-Feb:90-96, 1981.
99. DePalma AF, Rothman RH, Lewinnek GE et al: Anterior interbody fusion for severe cervical disc degeneration, *Surg Gynecol Obstet* 134:755-758, 1972.
100. Fernyhough JC, Schimandle JJ, Weigel MC et al: Chronic donor site pain complicating bone graft harvesting from the posterior iliac crest for spinal fusion, *Spine* 17:1474-1480, 1992.
101. DePalma AF, Rothman RH, Lewinnek GE et al: Anterior-interbody fusion for severe cervical disc degeneration, *Surg Gynecol Obstet* 134:755-758, 1972.
102. Cowley SP, Anderson LD: Hernias through donor sites for iliac-bone grafts, *J Bone Joint Surg Am* 65:1023-1025, 1983.

WWW.HIGHDENT.IR

همیار دندانسازان

103. Cricchio G, Lundgren S: Donor site morbidity in two different approaches to anterior iliac crest bone harvesting, *Clin Implant Dent Relat Res* 5:161-169, 2003.
104. Catone GA, Reimer LB, McNeir D et al: Tibial autogenous cancellous bone as an alternative donor site in maxillofacial surgery: a preliminary report, *J Oral Maxillofac Surg* 50:1258-1263, 1992.
105. O'Keeffe RM, Reimer BL, Butterfield LS: Harvesting of autogenous cancellous bone graft from the proximal tibial metaphysis: a review of 230 cases, *J Orthop Trauma* 5:469-474, 1991.
106. Ling GYC: Quantitative analysis of proximal tibial metaphyseal cancellous bone available for augmentation of maxillofacial bony defects, *J Oral Maxillofac Surg* 53(suppl):93, 1995.
107. Van Damme PA, Merkx MAW: A modification of the tibial bone-graft-harvesting technique, *Int J Oral Maxillofac Surg* 25:346-348, 1996.
108. Hoffman HT, Harrison N, Sullivan MJ et al: Mandible reconstruction with vascularized bone grafts, *Arch Otolaryngol Head Neck Surg* 117:917-925, 1991.
109. Ding M, Dalstra M, Danielsen CC et al: Age variations in the properties of human tibial trabecular bone, *J Bone Joint Surg Br* 79:995-1002, 1997.
110. Perel SM, Drane JB, Williams EO: Mandibular replacements, a review of the literature, *J Am Dent Assoc* 94:120, 1977.
111. Murphy J, Weisman R, Kent K: The use of stabilization plates in the immediate repair of defects following mandibular resection, *Oral Surg Oral Med Oral Pathol* 68:380-384, 1989.
112. Buchbinder D, Urken ML, Vickery C et al: Functional mandibular reconstruction of patients with oral cancer, *Oral Surg Oral Med Oral Pathol* 68:499-502, 1989.
113. King M, Casarett G, Weber D: A study of irradiated bone: histopathology and physiological changes, *J Nucl Med* 20:1142-1149, 1979.
114. Marx RE, Johnson R: Studies in the radiobiology of osteoradionecrosis and their clinical significance, *Oral Surg Oral Med Oral Pathol* 64:379-390, 1987.
115. Marx RE, Ames JR: The use of hyperbaric oxygen therapy in bony reconstruction of the irradiated and tissue deficient patient, *J Oral Maxillofac Surg* 40:412-420, 1982.
116. Urken ML, Buchbinder D, Weinberg H et al: Primary placement of osseointegrated implants in microvascular mandibular reconstruction, *Otolaryngol Head Neck Surg* 101:56-73, 1989.
117. Buchbinder D, Urken ML, Vickery C et al: Bone contouring and fixation in functional, primary microvascular mandibular reconstruction, *Head Neck* 13:191-199, 1991.
118. Lukash FM, Tenebaum NS, Moskowitz G: Long-term fate of the vascularized iliac crest bone graft for mandibular reconstruction, *Am J Surg* 160:399-401, 1990.
119. Ostrup LT, Fredrickson JM: Distant transfer of a free living bone graft by microvascular anastomoses, *Plast Reconstr Surg* 54:274, 1974.
120. Riediger D: Restoration of masticatory function by microsurgically revascularized iliac crest bone graft using endosseous implants, *Plast Reconstr Surg* 81:861-876, 1988.
121. Barber HD, Seckinger RJ, Hayden RE et al: Evaluation of osseointegration of endosseous implants in irradiated, vascularized fibula flaps to the mandible: a pilot study, *J Oral Maxillofac Surg* 53:640, 1995.
122. Roumanas ED, Markowitz BL, Lorant JA et al: Reconstructed mandibular defects: fibula free flaps and osseointegrated implants, *Plast Reconstr Surg* 99:356-365, 1997.
123. Lukash FM, Sachs SA, Fischman B et al: Osseointegrated denture in a vascularized bone transfer: functional jaw reconstruction, *Ann Plast Surg* 19:538-544, 1987.
124. Schliephake H, Schmelzelsen R, Husstedt H et al: Comparison of the late results of mandibular reconstruction using nonvascularized or vascularized grafts and dental implants, *J Oral Maxillofac Surg* 57:944-950, 1999.
125. Kovacs AF: The fate of osseointegrated implants in patients following oral cancer surgery and mandibular reconstruction, *Head Neck* 22:111-119, 2000.
126. Chana JS, Chang YM, Wei FC et al: Segmental mandibulectomy and immediate free fibula osteoseptocutaneous flap reconstruction with endosteal implants: an ideal treatment method for mandibular ameloblastoma, *Plast Reconstr Surg* 113:80-87, 2004.
127. Jaquiery C, Rohner D, Kunz C et al: Reconstruction of maxillary and mandibular defects using prefabricated microvascular fibular grafts and osseointegrated dental implants—a prospective study, *Clin Oral Implants Res* 15:598-606, 2004.
128. Ortakoglu K, Suer BT, Ozyigit A et al: Vertical distraction osteogenesis of fibula transplant for mandibular reconstruction: a case report, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e8-e11, 2006.
129. Kobayashi E, Ishihara Q, Mataga I: Effects of the placement of endosseous implants in vascularized bone grafts on bone union in beagle dogs, *Int J Oral Maxillofac Surg* 34:659-667, 2005.
130. Kuriloff DB, Sullivan MJ: Mandibular reconstruction using vascularized bone grafts, *Otolaryngol Clin North Am* 24:1391-1418, 1991.
131. Sanger JR, Head MD, Matloub HS et al: Enhancement of rehabilitation by use of implantable adjuncts with vascularized bone grafts for mandible reconstruction, *Am J Surg* 156:243-247, 1988.
132. Tuminelli FJ: The use of vascularized bone grafts in osseointegration. A clinical report, *J Prosthet Dent* 62:371-375, 1989.
133. Fenton AH, Mock D, Baker G et al: Reconstruction of the resected mandible using titanium implants in a free vascularized graft, *J Can Dent Assoc* 55:465-468, 1989.
134. Schmelzeisen R, Hausamen JE, Neukam FW et al: Combination of microsurgical tissue reconstruction with osteointegrated dental implants: presentation of a technique, *Int J Oral Maxillofac Surg* 19:209-211, 1990.
135. Zlotolow IM, Huryn JM, Piro JD et al: Osseointegrated implants and functional prosthetic rehabilitation in microvascular fibula free flap reconstructed mandibles, *Am J Surg* 165:677-681, 1992.
136. Hoffman HT, Harrison N, Sullivan MJ et al: Mandible reconstruction with vascularized bone grafts, *Arch Otolaryngol Head Neck Surg* 117:917-925, 1991.
137. Marx RE, Morales MJ: The use of implants in the reconstruction of oral cancer patients, *Dent Clin North Am* 42:177-201, 1998.
138. Marx RE, Ehler WJ, Peleg M: Mandibular and facial reconstruction rehabilitation of the head and neck cancer patient, *Bone* 19:595-625, 1996.
139. Teot L, Bossé JP, Moufarrage R et al: The scapular crest pedicled bone graft, *Int J Microsurg* 3:257-262, 1981.

WWW.HIGHDENT.IR

140. Swartz WM, Banis JC, Newton ED et al: The osteocutaneous scapular flap for mandibular and maxillary reconstruction, *Plast Reconstr Surg* 77:530-545, 1986.
141. Disa JJ, Cordeiro PG: Mandible reconstruction with microvascular surgery, *Semin Surg Oncol* 19:226-234, 2000.
142. Aronson J: The biology of distraction osteogenesis. In Maiocchi AB, Aronson J, editors: *Operative principles of Ilizarov: fracture treatment, nonunion, osteomyelitis, lengthening, deformity correction*, Baltimore, 1991, Williams & Wilkins.
143. Ilizarov GA: The tension-stress effect on the genesis and growth of tissues. I. The influence of stability of fixation and soft-tissue preservation, *Clin Orthop* 238:249-281, 1989.
144. Block MS, Chang A, Crawford C: Mandibular alveolar ridge augmentation in the dog using distraction osteogenesis, *J Oral Maxillofac Surg* 54:309-314, 1996.
145. Block MS, Baughman DG: Reconstruction of severe anterior maxillary defects using distraction osteogenesis, bone grafts, and implants, *J Oral Maxillofac Surg* 63:291-297, 2005.
146. Jensen OT, Cockrell R, Kuhlke L: Anterior maxillary alveolar distraction osteogenesis: a 5 year study, *Int J Oral Maxillofac Surg* 17:52, 2002.
147. Chiapasco M, Lang NP, Bosshardt DD: Quality and quantity of bone following alveolar distraction osteogenesis in the human mandible, *Clin Oral Implants Res* 17:394-402, 2006.
148. Degidi M, Pieri F, Marchetti C et al: Immediate loading of dental implants placed in distracted bone a case report, *Int J Oral Maxillofac Implants* 19:488-540, 2004.
149. Marchetti C, Corinaldesi G, Pieri F et al: Alveolar distraction osteogenesis for bone augmentation of everely atrophic ridges in 10 consecutive cases: a histologic and histomorphometric study, *J Periodontol* 78:260-266, 2007.
150. Chiapasco M, Consolo U, Bianchi A et al: Alveolar distraction osteogenesis for the correction of vertically deficient edentulous ridges: a multicenter prospective study on humans, *Int J Oral Maxillofac Implants* 19:399-407, 2004.
151. Aronson J: Limb-lengthening, skeletal reconstruction and bone transport with the Ilizarov method, *J Bone Joint Surg* 79A:1243-1258, 1997.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

This page intentionally left blank

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 41

An Implant Is Not a Tooth: A Comparison of Periodontal Indices

Carl E. Misch

The primary function of a dental implant is to act as an abutment for a prosthetic device, similar to a natural tooth root and crown. The restoring dentist designs and fabricates a prosthesis similar to one supported by a tooth and, as such, also evaluates and treats the dental implant similarly to a natural tooth. Yet fundamental differences in the support system have to be recognized. The purpose of this chapter is to compare the periodontal indices for a natural tooth and an osteointegrated implant.

LITERATURE REVIEW

Several dental health criteria have been adapted for implants.[1-12] The clinical criterion most commonly reported is the survival rate, or whether the implant is still physically in the mouth or has been removed.[2] Proponents of this method say it provides the clearest presentation of the data; critics argue implants that should be removed because of pain, disease, or the inability to be restored still may be maintained yet wrongfully reported as successful. Reports of natural teeth used to support a prosthesis follow a similar criterion: whether the restoration is still in the mouth. Therefore survival rates rather than success rates are the most common method to report the "success" of the prosthesis, whether the prosthesis is supported by implants or natural teeth. A majority of reports that include clinical criteria include mobility, radiographic assessment, and gingival and plaque indices. Subjective criteria of discomfort and patient satisfaction also are mentioned.

The American Dental Association Council on dental materials, instruments, and equipment states that consideration for an endosteal implant should be given to the evaluation of (1) durability; (2) bone loss; (3) gingival health; (4) pocket depth; (5) effect on adjacent teeth; (6) function; (7) esthetics; (8) presence of infection, discomfort, paresthesia, or anesthesia; (9) intrusion on the mandibular canal; and (10) the patient's emotional and psychological attitude and satisfaction.[3,4] Smith and Zarb[5] suggested that patient comfort, sulcus depth, gingival status, damage to adjacent teeth, and violation of the maxillary sinus, mandibular canal, or floor of the nasal cavity are not attributable to the material or design of an implant. As a result, they suggest these should be considered separately and should not be computed in the percentage of implant successes. One may reasonably state that factors controlled primarily by the dentist and the psychological attitude of the patient are not conditions influenced by the implant. However, the sulcus depth and gingival status next to the implant indeed may be related to implant design or surface condition. For example, a smooth, polished collar contributes to crestal bone loss, which affects sulcus depth. The implant surface condition may allow bacteria to form readily on the surface after crestal bone loss and may affect the gingival status of the implant.

This chapter compares the following criteria for implants and natural teeth: (1) longevity, (2) pain, (3) mobility versus rigid fixation, (4) percussion, (5) crestal bone loss, (6) radiographic evaluation, (7) keratinized tissue, (8) probing depths, (9) bleeding index, (10) peri-implant disease, and (11) implant failure.

Periodontal indices are often used for evaluation of dental implants. However, implants are fundamentally different than natural teeth in that they do not decay, have no dental pulps to function as early indicators of disease, and have no periodontal membrane. A comparison of natural teeth and implants for each criterion provides insight into their differences in the health-disease continuum. Once one understands the basis for evaluation, these criteria may then be used to establish a health-disease implant quality scale related to patient treatment.

WWW.HIGHDENT.IR

LONGEVITY

Success criteria for endosteal implants have been proposed previously by several authors, including Schnitman and Shulman,[6] Cranin et al.,[7] McKinney et al.,[8] Albrektsson et al.,[9,11] and Albrektsson and Zarb.[10] As Box 41-1 shows, the report by Albrektsson et al.[9] was specific for implants with rigid fixation and is used widely today. However, the amount of crestal bone lost during the first year may affect the sulcus depth and environment for the longevity of the implant. Yet this criterion is not mentioned in Albrektsson and Zarb's classification of health. In addition, survival rates suggested in this guideline are low compared with present-day reports and do not consider the prosthesis survival rather than implant longevity.

The consideration of a minimum implant survival rate should be in the context of the final prosthesis' survival. For example, many early reports indicated that a fixed prosthesis in a completely edentulous arch may be supported by four implants. More recently, the suggestion has been made to fabricate a fixed prosthesis with immediate loading on as few as three or four implants. In a study of 25 patients with 25 prostheses supported by only four implants, there would be 100 implants. A 75% implant success rate would result in 0% prosthesis success, if each patient lost only one implant. An 85% implant 5-year survival rate still would affect almost half the implant restorations. Of course, this survival rate is not acceptable. Implant survival by itself is not an acceptable criterion to evaluate an implant system, and studies must include the restoration. Albrektsson et al.[9] have stated that the required implant success rate is a minimum of 85% for 5 years and 80% for 10 years. These success rates are similar to prosthesis success on natural teeth.[13] However, the initial proposed criteria do not evaluate the prosthesis and may be unacceptable to the patients undergoing restoration. Implant survival and the associated prosthesis survival rates need to be evaluated together, because the restoration is the most important aspect to the patient.

Dental implant success rates reported in the literature typically do not address prosthesis failure. Instead, as long as the prosthesis may be refabricated, the reported implant success is not affected. More relevant information is gained when longevity criteria include information on the prosthesis. The clinical criteria established by Misch for optimum to satisfactory health for implants also evaluates prosthesis survival, not only implant survival, and suggests a minimum of 90% prosthesis survival for 10 years[1] (Box 41-2).

Of course, these rates require even greater implant survival, and overengineering the support system often is required to obtain this goal. If eight rather than four implants support a fixed prosthesis, possibly one or two implants may be lost and the same prosthesis still may be used without additional implants and only slight modification of the restoration.

As a result, for 25 patients each restored with eight implants to support a full-arch restoration, and if each patient lost one implant, the survival of the prosthesis still may be 100% and the implant survival 87%.

For the patient and doctors involved in treatment, to lose 10% of the implants before fabrication of the prosthesis is far better than to have 5% implant failure after delivery of the restoration.[14] The average implant restoration in a partially edentulous patient has three implants as support. A 5% difference in implant survival may affect 15% of the prostheses. In addition, computed data of dental implant survival/success should include all implants inserted, not just the implants restored or those successfully loaded after 1 year.

Box 41-1 Criteria for Implant Success

- An individual unattached implant is immobile when tested clinically.
- The radiograph does not demonstrate any evidence of periimplant radiolucency.
- Vertical bone loss is less than 0.2 mm annually after the first year of service of the implant.
- Individual implant performance is characterized by an absence of persistent or irreversible signs and symptoms such as pain, infections, neuropathies, paresthesia, or violation of the mandibular canal.
- In the context of the foregoing, success rates of 85% at the end of a 5-year observation period and 80% at the end of a 10-year period are minimum criteria for success.

From Albrektsson T, Zarb GA, Worthington P et al: The long-term efficacy of currently used dental implants: a review and proposed criteria of success, *Int J Oral Maxillofac Implants* 1:1, 1986.

PAIN

Subjective findings of pain, tenderness, and sensitivity are common dental conditions that the dentist treats as part of a general practice. Pain and tenderness are

Box 41-2 Suggested Criteria for Implant Success

- Implant quality scale* of 1, 2, or 3 with a survival rate better than 90% at 10 years
- Prosthesis survival rate better than 90% at 10 years
- Implants that are supporting a prosthesis

*For the Misch implant quality scale, see Chapter 42.

WWW.HIGHDENT.IR

subjective criteria and depend on the patient's interpretation of the degree of discomfort. *Pain* is defined as an unpleasant sensation ranging from mild discomfort to excruciating agony. Tenderness is more an unpleasant awareness of the region. A natural tooth often becomes hyperemic and sensitive to cold as the first indicator of a problem. A tooth with a more serious condition becomes sensitive to heat and painful to percussion, indicating pulpitis. Dental emergencies usually are associated with pain, and the dentist is adept at its diagnosis and treatment planning. An implant rarely is troubled by the subjective criteria of pain or sensitivity. The implant does not become hyperemic and is not temperature sensitive, and the early warning signs and symptoms of a problem may not be present. In addition, pain rarely is associated with the implant after healing. This criterion is less contributory to implant health determination.

Once the implant has achieved primary healing, absence of pain under vertical or horizontal forces is a primary subjective criterion. Percussion and forces up to 500 g (1.2 psi) are used clinically to evaluate tooth or implant pain or discomfort. Usually (but not always), pain does not occur unless the implant is mobile and surrounded by inflamed tissue or has rigid fixation but impinges on a nerve. The most common condition that causes discomfort is when a loose implant abutment is entrapping some of the soft tissue in the abutment-implant connection. Once the soft tissue in the region is eliminated and the abutment is secured, the discomfort subsides. When the abutment-implant connection is secure and pain is present, consideration is given to an implant body fracture.

On rare occasions an implant may cause discomfort during function, although a clinical examination is unable to identify a cause. The persistent presence of pain during percussion or function on properly inserted implants and components often requires removal of the implant, even in the absence of mobility. Because pain is a subjective criterion, the dentist asks the patient to relate the pain from the implant site on a scale of 1 to 10, with 1 being a slight aggravation and 10 being the most intense pain the patient can perceive. When the patient reports a pain level greater than 5, the dentist should strongly consider removal of the implant. Pain from rigidly fixated implants is rare and is observed as an early problem, whereas pain from a mobile implant may occur early or late in treatment. In either case, the condition rarely improves. Pain on loading has been observed more often on immediately loaded implants compared with those healing unloaded for an extended period.

Implant sensitivity or mild tenderness rather than pain in a rigid implant is most unusual and signals a more significant complication for an implant than for a tooth. Tenderness during function or percussion usually implies healing in the proximity of a nerve or, on rare occasions, bone stress beyond physiologic limits.

If the implant tenderness immediately after surgery occurs in the proximity of the mandibular canal, the implant may be unthreaded 1 mm and reevaluated for a decrease in symptoms after 3 or more weeks. If the tenderness is after stage I healing and is not due to surgical encroachment on an anatomical landmark, stress may be the causative element. Attention is first brought to the soft tissue and prosthetic components. Treatment then consists of the elimination of as much stress on the implant or prosthesis as is possible for 3 or more weeks. The dentist especially should address occlusion and parafunctional habits in the presence of implant sensitivity. Most often the prosthesis should be modified, or additional implants may be placed to dissipate the forces. The tenderness may be decreased with these procedures but rarely is eliminated. Instead, the dentist notifies the patient of the poor prognosis and asks whether the tenderness is significant enough to warrant the removal of the implant. It should be emphasized this condition is rare and has been observed only a few times by the author in more than 30 years.

MOBILITY

Tooth Movement

The tooth exhibits normal physiologic movements in vertical, horizontal, and rotational directions. The amount of movement of a natural tooth is related to its surface area and root design. Therefore the number and length of the roots; their diameter, shape, and position; and the health of the periodontal ligament primarily influence a tooth's mobility. A healthy tooth exhibits zero clinical mobility in a vertical direction. Actual initial vertical tooth movement is about 28 µm and is the same for anterior and posterior teeth.[15] The vertical movement of a rigid implant has been measured as 2 to 3 µm under a 10-lb force and is due mostly to the viscoelastic properties of the underlying bone.[16]

Horizontal tooth mobility is greater than vertical movement. A very light force (500 g) moves the tooth horizontally 56 to 108 µm. The initial horizontal mobility of a healthy, "nonmobile" posterior tooth is less than that of an anterior tooth and ranges from 56 to 75 µm, which is two to nine times the vertical movement of the tooth. Initial horizontal mobility is even greater in anterior teeth and ranges from 90 to 108 µm in health.[15,17]

Muhlemann found that tooth movement may be divided into initial mobility and secondary movement.[18] The initial mobility is observed with a light force, occurs immediately, and is a consequence of the periodontal ligament. If an additional force is applied to the tooth, a secondary movement is observed, which is related directly to the amount of force. The secondary tooth movement is related to the viscoelasticity of the bone and measures as much as 40 µm under considerably greater force.

WWW.HIGHDENT.IR

Implant Movement

Rigid fixation is a clinical term that means the absence of observed clinical mobility. *Osseointegration* is a histologic term defined as bone in direct contact with an implant surface at the magnification of a light microscope.[19] Over the years, these two terms have been used interchangeably, and implant abutment support is most predictable with rigid fixation. Rigid fixation indicates the absence of clinical mobility of an implant tested with vertical or horizontal forces less than 500 g, similar to evaluating teeth. Lack of clinically observable movement does not mean the true absence of any movement. A "nonmobile" posterior natural tooth actually moves horizontally 56 to 73 µm. The human eye does not perceive this movement.

The anterior teeth, which often have slight clinically observable movement, actually move approximately 0.1 mm. A healthy implant moves less than 73 µm; thus it appears as zero clinical mobility.[16] Lack of implant mobility (IM) does not always coincide with a direct bone-implant interface.[8] However, when observed clinically, rigid fixation usually means that at least a portion of the implant is in direct contact with bone, although the percentage of bone contact cannot be specified.[20] A mobile implant indicates the presence of connective tissue between the implant and bone.

The implant-bone interface also exhibits lateral movement. Sekine et al.[16] evaluated the movement of endosteal implants with rigid fixation and found a range of 12 to 66 µm of movement in the labiolingual direction. Komiyama[21] reported 40 to 115 µm of implant movement in the mesiodistal direction under a force of 2000 g (about 4.5 psi) and a labiolingual range of 11 to 66 µm.

Rangert et al.[22] suggested that part of this movement may be due to component flexure. However, the greater implant movement in the mesiodistal dimension corresponds to the lack of cortical bone between the implants in this direction compared with the thicker lateral cortical plates present in the labiolingual dimension. The mobility of implants varies in direct proportion to the load applied and the bone density and reflects the elastic deformation of bone tissue. These mobility characteristics corroborate the findings of Fenton et al.,[23] who applied a 500-g load for 4 seconds to maxillary anterior teeth and osseointegrated implants. The implants were displaced a mean of 10 µm with a rapid elastic return (less than 1 millisecond), whereas the teeth showed a mean displacement of 57 µm with a prolonged viscoelastic return.

Increased tooth mobility may be caused by occlusal trauma or bone loss. However, increased tooth mobility alone is not a criterion of periodontal health or pathology. Unlike a tooth, for which mobility is not a primary factor for longevity, mobility is a primary determining factor for implant health.[24,25] Rigid fixation is also an excellent indicator of the health status because it is an easy, objective test. As such, rigid fixation is usually the first clinical criterion evaluated for a dental implant. The techniques to assess rigid fixation are similar to those used for natural tooth mobility. Two rigid instruments apply a labiolingual force of approximately 500 g.[26] The amplitude of tooth mobility may be rated from 0 to 4, where 0 is normal mobility from physiologic movement, 1 is detectable increased mobility, 2 is visible mobility up to 0.5 mm, 3 is severe mobility up to 1 mm, and 4 is extreme mobility including vertical movement. This same gradient may be used for oral implants with slight modification. As Box 41-3 depicts, IM-0 corresponds to the absence of clinical mobility, IM-1 demonstrates detectable increased movement, IM-2 is visible mobility movement up to 0.5 mm, IM-3 is severe horizontal mobility greater than 0.5 mm, and IM-4 is visible horizontal and vertical movement. The implant mobility scale was used frequently for plate form implants because a clinical goal was for slight mobility when joining the device to natural teeth. However, the goal for root form implants always should be rigid fixation and IM-0 status.

A natural tooth with primary occlusal trauma exhibits an increase in clinical mobility and radiographic periodontal ligament space. Once the cause of trauma is eliminated, the tooth may return to zero clinical mobility and a normal radiographic appearance. This scenario is not predictable around an implant. Implants with slight detectable mobility of approximately 0.1 mm of horizontal movement (IM-1), similar to the mobility of a healthy central incisor, on occasion may return to rigid fixation and zero mobility. However, to reachieve rigid fixation, the implant should be taken completely out of occlusion for several months.

Chances improve to return rigid fixation to an implant if no mobility is noted before the implant is placed into function. An implant with horizontal movement greater than 0.5 mm (IM-3) is at much greater risk than a tooth. The dentist should not restore an implant with any clinical mobility, because the risk of failure is great. However, once the prosthesis is completed and IM-1 develops, the risk is small to decrease almost all stress and evaluate implant mobility after several months. An osseointegrated implant with greater than 0.5 mm

Box 41-3 Clinical Implant Mobility Scale

Scale	Description
0	Absence of clinical mobility with 500 g in any direction
1	Slight detectable horizontal movement
2	Moderate visible horizontal mobility up to 0.5 mm
3	Severe horizontal movement greater than 0.5 mm
4	Visible moderate to severe horizontal and any visible vertical movement

WWW.HIGHDENT.IR

horizontal mobility (IM-3) or any vertical mobility (IM-4) should be removed to avoid continued bone loss and future compromise of the implant site.

On occasion, an implant that was rigid may spin in the bone at stage II uncovery, when the implant abutment is threaded into position.[27,28] The weak bone-implant interface is broken when the shear forces of adding an abutment are placed on the implant body. If this occurs, the implant cover screw should be reinserted and the implant allowed to "reintegrate" with the bone. The chances are good that 3 additional months of healing will allow the implant to reestablish a bone-implant interface. At the reinsertion of the abutment, a lesser torque is used initially, so the interface does not strip again. After an additional time of progressive loading, the abutment screw may be tightened as usual, although a countertorque method on the abutment is suggested.

The Periotest (Gulden-Medizinteknik, Bensheim an der Bergstrasse, Germany) is a computer-mechanical device, developed by Schulte, that measures the dampening effect or attenuation degree against objects by developing a force of 12 to 18 N against a pistonlike device, which then measures the distance the piston recoils into the chamber after striking an object.[29] A soft surface or mobile object gives higher recordings than a hard or rigid object. The recordings range from negative (8) to positive (50) numbers. Teeth with zero clinical mobility have typical ranges from 5 to 9. The degree or absence of clinical movement around an implant corresponds to values ranging from −8 to +9, or 17 degrees. This greatly aids the dentist's tactile senses. The bone density around the implant may be correlated with these numbers. This device has been used as a clinical tool to evaluate slight changes in implant rigid fixation or to identify prostheses that become partially unretained[30-32] (Figure 41-1). More recently, a nondestructive resonance frequency analysis technique to measure implant stability and osteointegration has been introduced and provides similar valuable information as to the clinical movement and bone density around implants.[33,34]

PERCUSSION

Percussion often is used on teeth to determine which tooth is sensitive to function or is beginning to abscess. In the past, percussion was used to evaluate the presence of rigid fixation. However, percussion is an indicator neither of clinical health nor of rigid fixation. The ringing sound that occurs on percussion only corresponds to the presence of some bone at the interface because 2 mm of bone and 16 mm of bone-implant interface sound almost identical. Percussion may be used to diagnose pain or tenderness with an implant but is misleading if used to determine the status of rigid fixation.

CRESTAL BONE LOSS

Periodontal probing is used to assess attachment levels to the tooth and is a prime indicator of health. Radiographic bone loss around a tooth does not indicate the presence of a disease state but is a reflection of past or present periodontal disease. Occlusal trauma may cause an increase in tooth mobility but does not cause marginal bone loss in the absence of periodontal disease.

The marginal bone around the implant crestal region is usually a significant indicator of implant health. Most often the surgical trauma causes little bone loss, but on occasion bone loss may reach several millimeters. The dentist may assess the presence of surgical bone loss before fabrication of the prosthesis. Crestal bone loss after prosthesis delivery is a primary indicator of the need for initial preventive therapy. Early loss of crestal bone beyond 1 mm from the microgap of the abutment after prosthesis delivery usually results from excess stress at the permucosal site or implant crest module design.[24,25,35]

The level of the crestal bone is measured from the crestal position of the implant at the stage II uncovery surgery. When the abutment is attached to the implant body, approximately 0.5 to 1 mm of connective tissue forms apical to this connection.[36] An implant originally placed 2 mm above the bone and another countersunk 2 mm below the bone have a different initial bone loss history after the abutment is attached to the implant. Whenever possible, the implant should be inserted at or above the bone crest to avoid an increase in the sulcus depth around the implant related to the crestal bone loss after abutment placement. Initial bone loss during

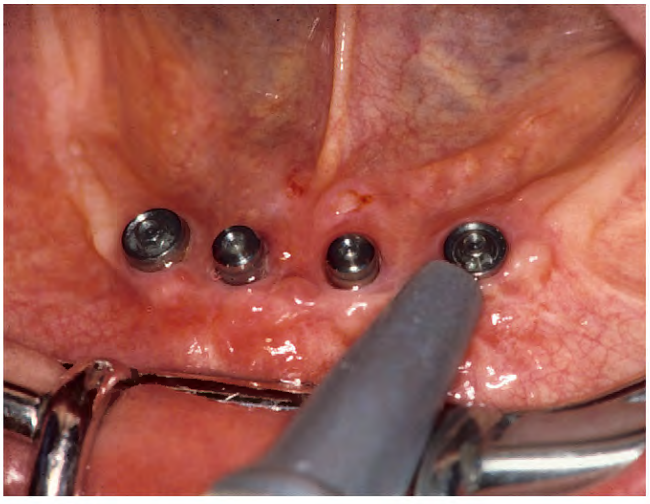


Figure 41-1 The Periotest (Gulden-Medizinteknik) may be used to evaluate implant rigid fixation or prostheses that become partially unretained. A clinical evaluation of zero mobility may correspond to a −8 to +9 Periotest value. These numbers may indicate changes in bone density around the implant or failure of a retention mechanism for the prosthesis.

WWW.HIGHDENT.IR

the surgical healing phase also may vary for submerged and unsubmerged healing protocols.[37-43]

Once the implant is connected to a permucosal element, the marginal bone may be lost during the first month from (1) the position of the abutment-implant connection or (2) the crest module design of the implant. The abutment-implant connection will cause 0.5 to 1.0 mm of bone loss when it is at or below the bone. In addition, when smooth metal is below the abutment-implant connection and extends onto the neck of the implant, additional bone loss will occur in direct relation to the smooth metal region. The bone levels will most often reside at the first thread or at a roughened surface after the first month a permucosal element or abutment extends through the soft tissue.

The initial bone loss beyond the abutment connection and smooth neck region of the implant after function is often a result of excessive stress at the crestal implant-bone interface.[24,25,35] The dentist should evaluate and reduce stress factors, such as occlusal forces, cantilever length, and especially parafunction, on observation of initial bone loss after loading. Secondary bone loss around an implant is usually a compound condition created by bacteria and increased stress (a result of parafunction or increasing crown height from crestal bone loss and anaerobic bacteria forming once the sulcus is greater than 5 to 6 mm).[44]

Several studies report marginal bone loss after the first year of function in the range of 0 to 0.2 mm.[19,43,45-48] Adell et al.[19] determined that successful implants after the first year of loading had an average 0.1 mm of bone loss for each following year. Cox and Zarb[45] observed a similar amount of mean bone loss of 0.1 to 0.13 mm per year after the first year of prosthesis function. Kline et al. reported these numbers represented an average of bone loss measurements. The majority of implants do not lose bone each year.[43] However, if one implant in 10 loses 1 mm of bone, the average bone loss is 0.1 mm. One should be careful when an implant is losing bone as shown radiographically. One should suspect occlusal overload, including parafunctional habits. Occlusal overload is especially notable when only one or two implants have lost bone and the other implants in the restoration are not affected.

Clinical observations obtained by probing or radiographic measurements of 0.1 mm per year for bone loss are more operator sensitive and not reliable. Probing changes of 0.5 mm or more are also more realistic to monitor. Therefore a yearly clinical assessment of bone loss in increments of 0.5 mm or more is suggested to monitor incremental marginal bone loss.

Slight changes in interproximal bone loss can be determined by radiographs. The threaded implant pitch (distance between the threads) is a known distance for each system (e.g., 0.6 mm for a classic Brånemark design) and can be used as a radiographic marker.

Under ideal conditions a tooth or implant should lose minimum bone. However, it is not possible to quantify how much bone loss indicates success or failure. In general, if more than half the implant height has lost crestal bony contact, the implant is at significant risk and is considered a failure, regardless of the original amount of implant-bone contact. In addition, the probing depth of the soft tissue should be considered related to the bone loss. If an implant has lost 5 mm of bone and has a probing depth of 10 mm, the situation is much worse than an implant with 6 mm of bone loss and a 3-mm probing depth.

RADIOGRAPHIC EVALUATION

The radiographic assessment of natural teeth assists in determining the presence of decay, lesions of endodontic origin, and periodontal bone loss. Radiographs may be used to evaluate the result of periodontal diseases on the supporting bone but cannot indicate the presence or absence of the disease process. Assessments of bone loss for natural teeth may include: (1) the presence or absence of intact lamina dura; (2) the width of the periodontal ligament space; (3) the bone crest morphology (even or angular); and (4) the distance from the cement-enamel junction (CEJ) and the coronal level to periodontal ligament (PDL) (normal or abnormal width). Normal radiographic bone levels next to natural teeth are typically between 1 to 3 mm from the CEJ.

Implants do not decay and do not develop endodontic-related conditions. However, the crestal bone region is often the most diagnostic for the ranges of optimum, satisfactory, and compromised health conditions. Radiographic interpretation is one of the easiest clinical tools to use to assess implant crestal bone loss but has many limitations. A radiograph only illustrates clearly the mesial and distal crestal levels of bone. However, early bone loss often occurs on the facial aspect of the implant. An absence of radiolucency around an implant does not mean bone is present at the interface, especially in the anterior mandible. As much as 40% decrease in density is necessary to produce a traditional radiographic difference in this region because of the dense cortical bone.[49] When the bone is wide, the V-shaped crestal defect may be surrounded by cortical bone and, as a result, the radiograph is less diagnostic.

Parallel periapical radiographs are more difficult to obtain for implants than for teeth. The implant is often apical to the apex of the preexisting natural tooth. As a result, the apex of the implant often is located beyond muscle attachments or in regions almost impossible to capture with a parallel radiographic method. A foreshortened image to accommodate the apical portion of the implant defeats the purpose of radiographic interpretation of the crestal bone. Crestal bone loss is evaluated

WWW.HIGHDENT.IR

best with vertical bitewing films or periapical radiographs that do not include the apical portion of the implant.

The clear depiction of the threads on the radiograph indicates use of a proper angulation. If the threads are clear on one side but fuzzy on the other, the angulation was incorrect by approximately 10%[50] (Figure 41-2). Ideally, the abutment-implant connection should appear as a clear line between the two components. When the top of the implant is placed at the crest of the regional bone, the amount of crestal bone loss is most easy to evaluate. If both sides of a threaded implant are unclear, the radiograph is not diagnostic for crestal bone loss assessment.

A peri-implant radiolucency indicates the presence of surrounding soft tissue and is a sign of implant failure. The cause may be from infection (bacterial), iatrogenic (heat-induced bone loss), nonrigid fixation (iatrogenic or patient-induced), or local bone-healing disorders.

On rare occasions an apical radiolucency has been observed on a nonmobile implant. This is most likely a perforation of one of the lateral cortical plates of bone but also may be from contamination of the drill, overheating, or infection.[51,52]

If the apical radiolucent region expands or is accompanied by a fistula, reentry surgery and correction are warranted. If the implant is mobile, it must be removed. If the implant has rigid fixation and the crestal half is in good order, the apical cause of the radiolucency must be removed and aggressively curetted, which may include sectioning and removal of the apical portion of the implant.

The implant quality of health evaluation protocol depends on clinical observations. A baseline radiograph is obtained at the initial delivery of the prosthesis. By this time the biological width and influence of the implant crest module design have already contributed to its influence on crestal bone loss. Because crestal bone changes often occur during the first year of loading, preventive maintenance appointments are scheduled every 3 to 4 months and a periapical/vertical bitewing radiograph at 6 to 8 months is compared with the baseline. Another vertical bitewing radiograph is taken at 1 year and compared with the previous two images. If no changes are apparent, subsequent radiographic examinations may be scheduled for every 3 years, unless other clinical signs warrant more frequent examinations.

If crestal changes are evident by probing or radiographs, stress reduction and hygiene are modified accordingly. Radiographs are taken and reviewed every 6 to 8 months until the bone is stable for two consecutive periods. If bone loss greater than 2 mm is observed from the bone levels noted at the prosthesis delivery, the dentist should strongly suspect parafunction. Night guards and stress reduction on the affected implants are indicated.

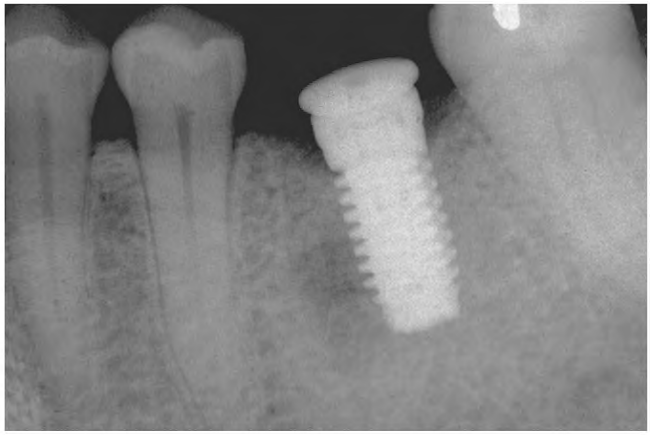


Figure 41-2 On this periapical radiograph the threads are clear on one side only. The central ray was not directed completely perpendicular to the implant body, but was within 10 degrees. This film is not ideal but is clinically acceptable in most situations.

KERATINIZED TISSUE CONCERNS

The absence or presence of a zone of keratinized gingiva around teeth and oral implants remains a controversial issue.[53-71] No direct evidence confirms or denies the need for nonmobile keratinized tissue next to natural teeth. Lang and Loe[53] advocate a minimum 2 mm of keratinized gingiva and 1 mm of attached gingiva to maintain gingival health. In longitudinal studies, however, Wennstrom[54] and Kennedy et al.[55] demonstrated that the lack of adequate keratinized and attached tissue does not compromise the long-term health of soft and hard tissue as long as patients maintain good oral hygiene. Moreover, Stetler and Bissada[57] addressed mucogingival considerations in restorative dentistry in 1987. They concluded that if subgingival restorations were to be placed in areas of minimally keratinized gingiva and less than optimal plaque control, augmentation to widen the zone of keratinized tissue may be warranted. They also noted that in unrestored teeth, the difference in the inflammatory status of sites with or without a wide zone of keratinized tissue was not significant. Valderhaug and Birkeland demonstrated that subgingival placement of a restoration was associated with a significantly higher rate of gingival inflammation, attachment loss, and gingival recession over 10 years.[58] The tooth with the least amount of keratinized tissue is often the mandibular first premolar. Yet this tooth is rarely the first tooth lost from periodontal disease. If all other periodontal indices are normal, the amount or absence of keratinized gingiva has little to do with the expected longevity of the tooth.

The need for keratinized tissue around dental implants seems more controversial than that around teeth.[59-71] In theory, structural differences in implants compared with teeth make them more susceptible to the development of inflammation and bone loss when exposed to plaque accumulation or microbial invasion

WWW.HIGHDENT.IR

(e.g., less vascular supply, less fibroblasts, lack of connection tissue attachment to cementation).[36] Several reports demonstrate the long-term implant survival in the absence of keratinized tissue[19,60,61] (Figure 41-3). Although reports are more cautious with mobile mucosa next to an implant, nonmobile tissue appears to be the primary criterion relative to tissue type.

Although keratinized tissue around a tooth may not be mandatory for long-term health, a number of benefits are present with keratinized mucosa. The color, contour, and texture of the soft tissue drape should be similar around implants and teeth. The interdental papillae should ideally fill the interproximal spaces. A high smile line often exposes the free gingival margin and interdental papillae zones. The keratinized tissue is more resistant to abrasion. Hygiene aids are more comfortable to use. The degree of gingival recession appears related to the absence of keratinized gingiva. Root sensitivity and esthetic concerns may be associated with gingival recession. From a restorative dental aspect, keratinized mucosa is more manageable during the retraction and impression-making process. Subgingival margin placement is more precise, as is long-term stability, in the presence of keratinized tissue.

The presence of keratinized tissue next to an oral implant presents greater benefits than with natural teeth (Figure 41-4). Some reports indicate the lack of keratinized tissue may contribute to implant failure. Kirsch and Ackermann[68] reported that the most important criterion for implant health in the posterior mandible was related to the absence or presence of keratinized gingiva. Mobile, nonkeratinized mucosa exhibits greater probing depths, which has been confirmed histologically. A study by Warrer et al.[67] in monkeys found that an absence of keratinized mucosa increases the susceptibility of periimplant regions to plaque-induced destruction (Figure 41-5).

Keratinized gingiva has more hemidesmosomes; thus the junctional epithelial attachment zone may be of benefit when in keratinized tissue. The orientation of collagen fibers in the connective tissue zone of an implant often appears perpendicular to the implant surface, whereas these fibers in mobile, nonkeratinized tissue run parallel to the surface of the implant. Schroeder et al.,[59] James and Schultz,[69] McKinney et al.,[8] and Listgarten et al.[70] have suggested that mobile mucosa may disrupt the implant-epithelial attachment zone and contribute to an increased risk of inflammation from plaque.

In addition to the general advantages, keratinized tissue around implants may be beneficial in several

Figure 41-3 Attached keratinized tissue in the mandible is often less in the first premolar region. Likewise, a first premolar implant site often has little keratinized tissue. Yet no current study indicates that the first premolar tooth or implant is the least predictable location for health or survival. This suggests that although ideal for long-term maintenance and health, the presence of keratinized tissue may not be mandatory.

Figure 41-4 The ideal soft tissue situation around teeth or implants is keratinized, nonmobile tissue.

Figure 41-5 Mobile, nonkeratinized tissue around a threaded or rough implant often is associated with plaque retention and increased risk of complications.

WWW.HIGHDENT.IR

other ways. In a two-stage protocol, the implant is less likely to become exposed during the healing process. The formation of an interdental/implant papillae is completely unpredictable with mobile unkeratinized tissues. However, no clinical or histologic benefits are reported with unkeratinized nonmobile mucosa. When the unkeratinized tissue is mobile, several reports state that this is unsatisfactory. Ono et al.[72] have proposed a classification of attached gingiva and surgical alternatives to improve soft tissue types in edentulous sites for implant placement. Meffert et al.[73] prefer to obtain keratinized tissue before implant placement, especially in the posterior regions. Interestingly, the studies that have advocated the need for keratinized mucosa around dental implants have investigated implants with rough surfaces.[67,68,74,75] On the other hand, the studies that have questioned the need of keratinized mucosa around dental implants have also examined implants with smooth surfaces.[61,64,76]

Failure of rough surface implants (e.g., hydroxyapatite [HA]-coated and plasma-sprayed implants) have been related to a lack of keratinized mucosa.[74,77] A study by Chung et al. evaluated the significance of keratinized mucosa in the maintenance of dental implants with different surface conditions.[78] All 69 patients and 339 implants in the study had implant restorations for at least 3 years and as long as 24 years with an average of 8.1 years. Bleeding index, modified plaque index, gingival index, probing depth, width of attached keratinized mucosa, and amount of attached mucosa were recorded. In addition, average annual bone loss was calculated using past and present radiographs. Gingival inflammation and plaque accumulation was significantly higher in patients with less than 2 mm of keratinized mucosa or 1 mm of attached mucosa. The surface condition of the implant was not statistically significant in this study, although the smooth implants with less than 2 mm of keratinized mucosa were less stable than other groups relative to the soft tissue profile.

This study found the average annual bone loss was not influenced by the amount of keratinized or attached mucosa or the type of implant surface configuration (smooth versus rough). The greatest amount of bone loss was observed with rough implants in keratinized mucosa of less than 1 mm, but the difference was not statically relevant. This compares to a metaanalysis by Esposito et al. who reported 20% less peri-implantitis in smooth-surface implants as compared with rough-surface implants.[79] The presence of keratinized mucosa was most significantly advantageous in the soft tissue health of posterior implants, as indicated by the gingival index. Posterior implants, even in the presence of keratinized tissue, had a 3.5-fold higher annual bone loss than anterior implants (0.14 versus 0.04 mm).[78] Therefore implant location appears more important than the presence of keratinized mucosa.

The question relative to the need for keratinized tissue around implants should be modified to "Which would you prefer?" No one has stated that the unkeratinized tissue is better than keratinized tissue for any reason; therefore the controversy has abated. Some authors prefer keratinized mucosa more intensely than others. If one side of controversy demonstrates benefits while the other side states that keratinized tissue is not mandatory, both sides may be correct.

In specific clinical instances, attached, keratinized gingiva is more often desirable. For example, a fixed prosthesis type 1 (FP-1) restoration in the esthetic zone requires keratinized mucosa to develop the soft tissue drape around the implant crowns. A second prime example is a mandibular overdenture, which benefits from a vestibule and zone of nonmobile tissue around the implant abutments.

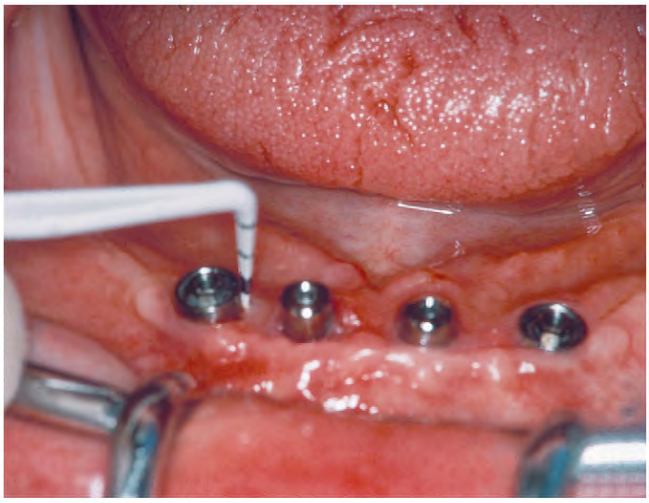


Figure 41-6 Peri-implant probing may be used to evaluate soft tissue depth, bleeding index, the presence of exudate, tissue mobility thickness, and the amount of crestal bone loss.

PROBING DEPTHS

Probing depths around teeth are an excellent proven means to assess the past and present health of natural teeth. The increasing sulcus depth around natural teeth is related to disease and bone loss. Likewise, probing depth indices often are used to evaluate dental implants (Figure 41-6). However, relating implant sulcus depth to health is controversial.

When probing next to an implant or a tooth, the probe not only measures the sulcus depth, but also penetrates the junctional epithelial attachment. A primary reason is found in the different makeup of the biological width[36,38-42,80,81] (Tables 41-1, 41-2). Therefore the probe next to a natural tooth measures the sulcus depth and the depth of the junctional epithelial attachment. The connective tissue attachment zone in a natural tooth has 13 different fiber groups of which six physically

WWW.HIGHDENT.IR

Table 41-1 Comparison of Tooth and Implant Support Structures

STRUCTURE	TOOTH	IMPLANT
Connection	Cementum, bone, periodontium	Osseointegration, bone functional ankylosis ligament
Junctional epithelium	Hemidesmosomes and basal lamina (lamina lucida and lucida, lamina densa zones)	Hemidesmosomes and basal lamina (lamina, lamina densa, and sublamina lucida zones)
Connective tissue	Thirteen groups: perpendicular to tooth surfaces ↓ collagen, ↑ fibroblasts	Only two groups: parallel and circular fibers No attachment to the implant surface and bone ↑ collagen, ↓ fibroblasts
Biological width	2.04 to 2.91 mm	3.08 mm
Vascularity	Greater; supraperiosteal and periodontal ligament	Less, periostal
Probing depth	3 mm in health	2.5 to 5.0 mm (depending on soft tissue depth)
Bleeding on probing	More reliable	Less reliable

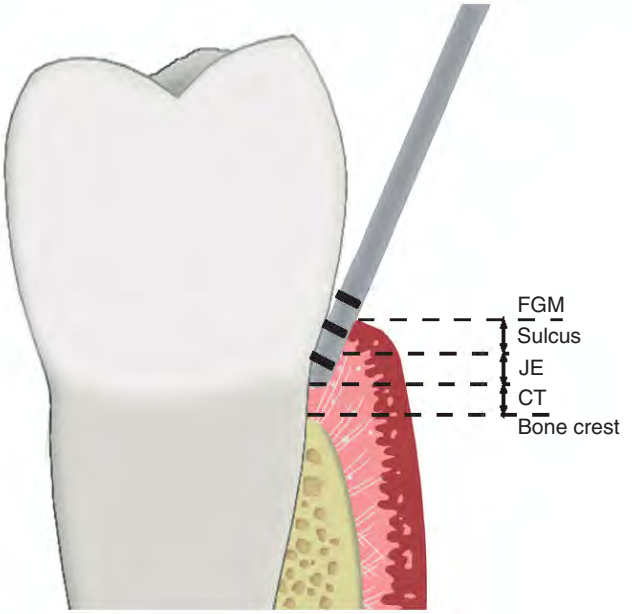


Figure 41-7 The biological width of a natural tooth has a connective tissue zone that inserts into the cementum of the tooth. A periodontal probe will penetrate the sulcus and the junctional epithelial attachment. *FGM,* Free gingival margin; *JE,* junctional epithelium; *CT,* connective tissue.

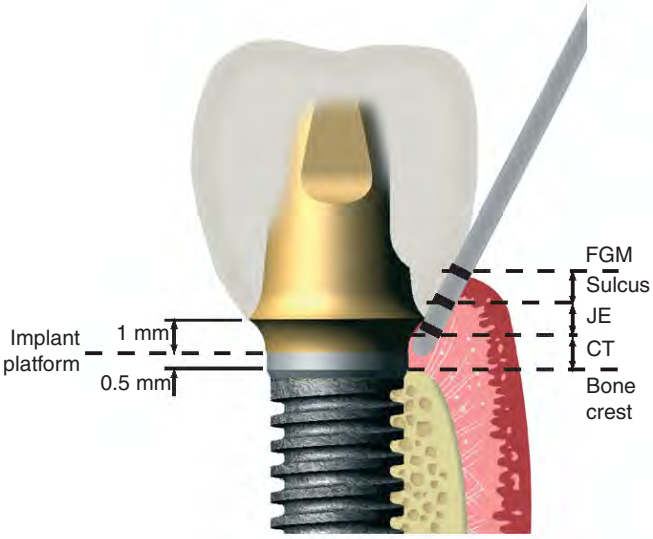


Figure 41-8 An implant has no connective fibers in the connective tissue zone that insert into the implant. The peri-implant probe penetrates the sulcus, junctional epithelial attachment, and most of the connective tissue zone. *FGM,* Free gingival margin; *JE,* junctional epithelium; *CT,* connective tissue.

insert into the cementation of a tooth. This zone stops the further probe penetration and acts as a barrier for the ingress of bacteria. The connective tissue attachment zone with ideal health is approximately 1 mm in height above the alveolar bone crest. An implant has a sulcus, a junctional epithelial attachment, and a zone of connective tissue (Figure 41-7). However, the structures are not similar to a tooth. The junctional epithelial attachment zone has a hemidesmosome attachment with a lucida and a lamina densa layer, as well as a sublaminal zone and glycoaminoglucine layer. Thus the zone has less attachment strength to the implant and probe, and bacteria can go through this region easier than with a tooth. In addition, the connective tissue zone has only two fiber groups and neither of those insert into the implant. As a result, with an implant, the probe goes beyond the sulcus, through the junctional epithelial attachment, through the type III collagen connective tissues, and reaches closer to the bone.[82] The tissue above the bone before implant insertion may also be 4 mm thick or more. As a result of greater tissue thickness before surgery and a 1-mm greater probing depth, the probing depth next to a healthy implant is typically greater than that of a healthy natural tooth (Figure 41-8).

Because the probe penetrates deeper next to an implant compared with a tooth, one should take care not to contaminate the implant sulcus with bacteria from a

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Table 41-2 Biological Width

	TEETH		IMPLANT								
			SUBMERGED PROTOCOL						NONSUBMERGED PROTOCOL		
	Gargiulo et al. 1960[80]	Berglundh et al. 1991[36]	Berglundh et al. 1991[36]	Abrahamsson et al. 1997[40]		Hermann et al.[38]			Cochran et al. 1997[42]		
			No abutment Disconnection/reconnection			Abutment Disconnection/reconnection			N/A		
			Crestal placement IAJ at the bone crest				Supracrestal placement IAJ 1 mm above the bone crest	Supracrestal placement IAJ 1 mm below the bone crest	Unloaded	Loaded 2 months	Loaded 12 months
SD	0.41-1.09 average 0.69								0.49 ± 0.32	0.50 ± 0.30	0.16 ± 0.14
JE	0.44-1.56 average 0.97	2.05 ± 0.06*	2.14 ± 0.47†	2.04 ± 0.22†	1.65 ± 0.24‡				1.16 ± 0.47	1.44 ± 0.41	0.88 ± 0.81
CT§	0.69-1.49 average 1.07	1.12 ± 0.03	1.66 ± 0.23	1.28 ± 0.11	0.85 ± 0.26				1.36 ± 0.64	1.01 ± 0.32	1.05 ± 0.38
IAJ-B	N/A	N/A		0.78 ± 0.17	1.49 ± 0.19	1.57 ± 0.22	2.64 ± 0.24	1.25 ± 0.40	2.90 ± 0.52	2.91 ± 0.49	2.95 ± 0.68

From Jalbout Z, Tabourian G, editors: *Glossary of implant dentistry,* Upper Montclair, NJ, 2004, International Congress of Oral Implantologists and New York University.
SD, Sulcus depth: *JE,* junctional epithelium; *CT,* connective tissue attachment; *B,* the marginal level of bone-implant contact (also known as first bone-to-implant contact); *IAJ,* implant-abutment junction (also known as interface, microgap); *N/A,* Not applicable; *IAJ-B,* Distance for IAJ to B.
*JE is the distance from GM (the gingival marginal) to aJE (the apical termination of the junctional epithelium).
†JE is the distance from GM (the margin of soft tissue adjacent to the titanium abutment) to aJE (the apical termination of the junctional epithelium).
‡JE is the distance from PM (the marginal portion of the peri-implant mucosa) to aJE (the level of apical termination of the junctional epithelium).
§CT for implants is the distance from aJE (the level of apical termination of the junctional epithelium) to B (the marginal level of bone-implant contact, also known as first bone-implant contact).
All measurements are in millimeters.

WWW.HIGHDENT.IR

diseased periodontal site. The location of the probe tip subgingivally for a tooth depends on the pressure used, the presence of inflammation, and the angle at which the probe is introduced in the sulcus depth between the junctional epithelium and the root surface. In addition, reports in the literature suggest that the reproducibility of attachment level measurements may be questionable independently from the instrument used to perform the measurements.[83,84] These variables are similar for a dental implant. In addition, fixed prostheses with subgingival margins of crowns with wider emergence profiles often make probe positioning difficult around the 4 mm diameter of most implant bodies.

The benefit of probing the implant sulcus has been challenged in the literature because sound scientific criteria are lacking. The potential for damage to the fragile attachment or marring of the implant surface exists during probing. The correct pressure recommended for probing is 20 g, yet conventional probing often exerts a force more than five times this level and varies greatly. Pressure-sensitive probes have been made available to address these issues but are rarely used in a clinical practice.[85-87] Lekholm et al.[65] found that the presence of deep pockets was not accompanied by accelerated marginal bone loss. Stable, rigid, fixed implants were reported with pocket depths ranging from 2 to 6 mm. Healthy, partially edentulous implant patients consistently exhibit greater probing depths around implants than around teeth.

The implant sulcus depth may be a reflection of the original soft tissue thickness of the area before implant placement. The posterior maxillary tissue can be thicker than 4 mm after tooth extraction and subsequent bone volume loss before implant placement. Gingivoplasty to reduce the flap thickness and pocket depth can be performed at the initial surgery. The advantage of the reduction in tissue thickness at this time is the tissue heals and matures as the bone-implant interface develops. However, thinning the flap at the initial surgery may cause greater loading of the implant body during healing from an overlying soft tissue-borne temporary prosthesis. After initial bone healing, the stage II uncovery surgery also may correct tissue thickness; however, when an overlying soft tissue prosthesis is worn during healing it may prematurely load the developing implant interface and cause bone loss or failure.

An increasing probing depth is a more significant sign than a probing depth unrelated to a time interval because it usually signifies bone loss, except in cases of gingival hyperplasia or hypertrophy. Probing using fixed reference points on the abutment or crown margin allows evaluation of crestal bone loss versus tissue hypertrophy (Figures 41-9 and 41-10).

Despite the limitations, charting the attachment level in implant permucosal areas does aid the dentist in monitoring these regions. As the sulcus depth increases, the oxygen tension decreases. The bacteria in an implant sulcus are similar to those of a natural tooth.[88,89] A toothbrush and daily hygiene procedures cannot clean a sulcus greater than 2 mm.[90] Sulcus depths greater than 5 to 6 mm have a greater incidence of anaerobic bacteria[60,87] and often require gingivectomy or bone revision surgery. Therefore as a general rule, to enable the patient to perform effective daily hygiene, the ideal implant sulcus should be maintained at less than 5 mm.

The monitoring of early crestal bone loss is most important during the first critical year of stress accommodation of the bone. Minor bone changes are clinically easier to observe with a periodontal probe than with radiographs. Early bone loss may occur on the facial aspect of the implant; radiographs only demonstrate clearly the mesial and distal regions.

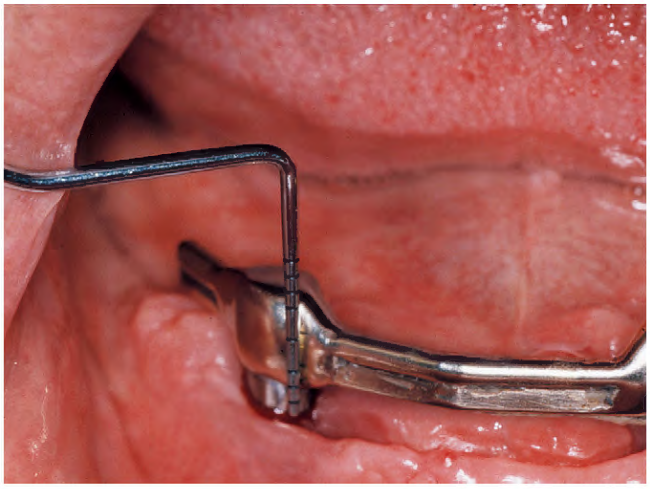


Figure 41-9 Because the soft tissue may shrink or hypertrophy, a long-term record of bone changes can be monitored from the restoration margin. This implant has a bleeding index of 2.

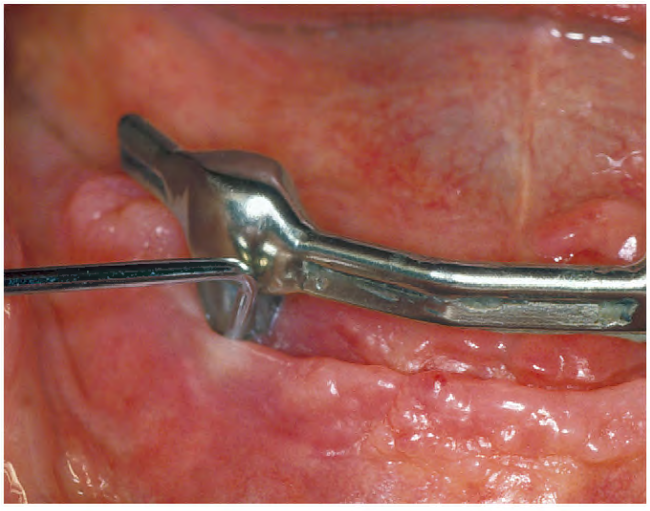


Figure 41-10 On the facial aspect of the implant, the probing depth is greater than 12 mm. A radiograph cannot assess the bone levels on the facial or lingual aspects of an implant.

WWW.HIGHDENT.IR

Changes in crestal bone levels warrant close monitoring and early intervention. Patient education to reduce parafunctional stress on the implant system, the use of parafunctional appliances, and other stress-reducing methods are required when early crestal bone loss beyond the first thread is detected. Despite the uncertain meaning of pocket depth increase, probing is an appropriate method to assess potential deleterious changes in the peri-implant environment and should be performed every 3 to 4 months for 1 year after prosthesis delivery. After this time, if crestal bone levels are stable, probing is still relevant. Probing also reveals tissue consistency, bleeding, and exudate. Therefore probing is important not only to measure increasing sulcus depths but also to allow the dentist to evaluate several peri-implant parameters at the same time and at the same sites.

Controversy exists as to the material from which the probe should be fabricated. In theory, different metal types (e.g., stainless steel, titanium) should not come into contact because of a risk of contamination of the two metals and the resulting galvanic corrosion that may develop and cause crestal bone loss. As a result of this fear, the suggestion has been made that only titanium surgical instruments be used to contact the implant and that only titanium or plastic instruments be used to probe or scale the implant.

Touching the surface of the abutment subgingivally with a stainless steel instrument is not of clinical concern. However, scratching the surface may contribute to plaque migration following the direction of the scratch. Plaque follows the direction of scratches on a titanium plate, even though right angles and a maze pattern may be scratched onto the surface. Therefore, when probing almost to the bone level around the implant, one should take care not to scratch the surface because plaque that forms at the surface may follow the scratch subgingival to the bone level. This is particularly important during scaling procedures or during the removal of cement below a crown margin. One should use semicircular strokes, parallel to the sulcus or crown margin, to scale the implant above the bone. If a scratch occurs, plaque will not have a direct "highway" below the tissue.

BLEEDING INDEX

Gingival bleeding when probing correlates with inflammation and the plaque index. A bleeding index is an indicator of sulcus health. Easily ulcerated sulcular epithelium representing inflammation from plaque is the primary cause of bleeding when probing. Bleeding also can be provoked by undue pressure on the probe.

Controversy surrounds the issue of using bleeding and gingival health as an implant health indicator.[70] Unlike a natural tooth, implant success in the first few years is related more often to biomechanical equilibrium than to gingival health. Compared with a natural tooth, the soft tissue inflammation from bacteria may be more restricted to above the crestal bone because of the lack of a periodontal membrane or fibrous tissue between the implant and the bone interface. As a result, the bleeding index may not be as important a factor when evaluating the early implant quality of health. Adell et al.[64] found no evidence that gingivitis was a precursor of progressive bone loss. Lekholm et al.[65] also found that gingivitis and deep sulcular pockets were not accompanied by accelerated bone loss.

Both of these reports apply to a machined-surface titanium screw design (e.g., Nobel Biocare). In contrast Kirsch and Mentag[77] found a correlation between the gingival sulcus depth and implant failure. The implant design studied in this report had an intramobile element with a larger implant body abutment crevice and a titanium plasma spray body (e.g., IMZ).

A similar correlation between implant failure and gingival health status was observed when a porous titanium alloy microball surface was exposed above the bone (e.g., Endopore).[92] Jepsen et al. identified the elevated levels of proteolytic enzymes in the implant sulcus with inflammation and bleeding on probing as predictors of implant disease.[93] The correlation between gingival health and implant success appears in part to be related to the cervical surface condition of the implant.

Lekholm et al.[64] and Quirynem et al.[91] found that plaque and gingivitis around implants were correlated. Steflik et al.[20] found that the gingival bleeding index correlated highly with the plaque index and the crevicular fluid index. The dentist already is encouraged to probe the sulcular region to evaluate bone loss. Periodontal probing is less demanding than the determination of a gingival sulcular fluid volume index. One may observe the bleeding index while probing for sulcus depth and therefore may record it easily to help establish gingival health.

Regardless of whether gingival health is relative to success, all dentists agree that the ideal soft tissue condition around an implant is an absence of inflammation. Radiographic bone loss and increased pocket depth have been correlated with sulcular bleeding.[20] Therefore the gingival status around an implant should be recorded and used to monitor the patient's daily oral hygiene. However, surrounding soft tissues around implants have fewer blood vessels than teeth; therefore inflammation is typically less around implants than around teeth.[36,94,95]

The most common bleeding gingival index used for implants is the Loe and Silness gingival index.[26] When used on teeth, this index scores gingival inflammation from 0 to 3 on the facial, lingual, and mesial surfaces of all teeth. The symptom of bleeding comprises a score of at least 2.[96]

WWW.HIGHDENT.IR

The gingival index scores also may be used to record the gingival inflammation on the facial, lingual, and mesial surfaces of all implants. A simplified gingival index has been suggested for use with teeth in a clinical practice setting because the differences in scoring levels of gingival indexes are too gross.[26] Because the bleeding index evaluates this inflammation, the Loe and Silness index is adequate for implants, and because fewer implants typically are used to restore a region compared with the presence of natural teeth, one also may evaluate the distal surface because the implants are more than 2 mm apart and access often is unobstructed. When the sulcus depth is less than 5 mm and the bleeding index increases, use of chlorhexidine often is indicated, along with other professional and home care methods. Sulcus depths in excess of 5 to 6 mm have a greater incidence of bleeding and usually require gingivectomy or revision surgery.

During the first year of clinical examinations for the peri-implant tissues, the dentist should record color, form, and consistency along with bleeding on probing and should probe depths for all sites. After 1 year of stable probing depths, the examination may be restricted to facial and lingual checks at maintenance appointments and may be correlated with radiographic observation for the mesial and distal surfaces. Removal of the prosthesis for evaluation is not indicated unless warrented by changing conditions. Repeated removal of a screw-retained fixed prosthesis causes wear of the screw attachment system and causes more frequent partially unretained restorations over the long term.

PERI-IMPLANT DISEASE

Gingivitis is a bacteria-induced inflammation involving the region of the marginal gingiva above the crest of bone and next to a natural tooth. It is always associated with plaque and may be classified as (1) acute necrotizing, (2) ulcerative, (3) hormonal, (4) drug induced, or (5) spontaneously occurring.[27] These categories also can relate to the gingival tissues around an implant because the mode of attachment of the gingiva to a tooth and implant has been reported to be similar.[69]

The bacteria in gingivitis around a tooth may affect the epithelial attachment but without loss of connective tissue attachment. Because the connective tissue attachment of a tooth extends an average of 1.07 mm above the crestal bone, at least 1 mm of protective barrier above the bone is left. In contrast, no connective tissue attachment zone exists around an implant because no connective fibers extend into the implant. Therefore no connective tissue barrier exists to protect the crestal bone around an implant.[69,97]

Periodontitis around teeth is characterized by apical proliferation and ulceration of the junctional epithelium, progressive loss of the connective tissue attachment, and loss of alveolar bone. Bacteria also cause periodontitis. The disease has been classified as adult, rapidly progressive, localized juvenile, and prepubertal periodontitis.

After prosthesis delivery, early crestal bone loss around an implant usually is not caused by bacteria. Most often the bone loss results from stress factors too great for the immature, incompletely mineralized bone-implant interface or an extension of the biological width into a smooth metal crest module.[24] Therefore an implant may exhibit early crestal bone loss with a different mechanism or cause compared with natural teeth. However, on occasion, bacteria may be the primary factor. Anaerobic bacteria have been observed growing in the microgap between the implant and the abutment or in the sulcus of implants, especially when sulcus depths are greater than 5 mm (Table 41-3; Box 41-4).

The term *peri-implantitis* describes the bone loss around an implant. The loss may be induced by stress, bacteria, or a combination of both. Stress-induced bone loss occurs without bacteria as the primary causative agent. However, once the bone loss from stress or bacteria deepens the sulcular crevice and decreases the oxygen tension, anaerobic bacteria may become the primary promoters of the continued bone loss.

An exudate or abscess indicates exacerbation of the peri-implant disease and possible accelerated bone loss (Figures 41-11 and 41-12). Short-term antibiotic treatment and aggressive topical application of chlorhexi-

Table 41-3 Subgingival Microflora Associated with Human Dental Implants

	Pocket Depth (mm)	
MICROFLORA	<5	>6
Spirochetes (%)	2	32
Motile rods (%)	16	18
Coccoids (%)	64	30

Box 41-4 Stable Integrated Implants

As Subgingival Pocket Depth Increases

Shallow
Gram-positive facultative cocci, rods
Gram-negative anaerobic cocci, rods
Motile rods
Spirochetes
Black pigmented bacteroides
Fusobacterium

Deep
Vibrios organisms

WWW.HIGHDENT.IR

Figure 41-11 Peri-implant probing measures pocket depths and may indicate the presence of exudate. Symptoms of exudate should be addressed with aggressive therapy.

Figure 41-12 Surgical reentry into the implant site from Figure 41-13 demonstrates advanced bone loss around the facial aspect of the implant.

dine or local antibiotics, with thorough, extensive professional and patient care of the soft tissue, are indicated. An exudate persisting for more than 1 to 2 weeks usually warrants surgical revision of the peri-implant area to eliminate causative elements.

Once the exudate is eliminated, the bacterial smear layer remaining on the implant surface must be eliminated before bone can grow in close contact with the implant. The reduced bone height, after the exudate episode, makes the implant more prone to secondary occlusal trauma. Therefore the dentist must reevaluate stress factors for the new bony condition and often must reduce them to improve long-term performance.

SUMMARY

An unhealthy implant most often is treated similarly to a natural tooth presenting the same conditions. Implementation of aggressive implant maintenance treatment is warranted with any detectable horizontal mobility, exudate, a pocket depth of 5 mm and increasing, a bleeding index of 2 or above, and slight tenderness to percussion and function. An implant may need less treatment than a natural tooth when sulcus depths are compared. However, based on mobility, an implant needs more treatment than a tooth.

The ultimate decision of implant success lies with the practicing dentist rendering continued dental therapy for the patient. A single-tooth implant with 0.5 mm of mobility is less at risk than a 12-unit fixed prosthesis implant abutment with 0.2 mm of mobility. A gray scale of decision making exists, and absolute rules make for easy decisions, but rules are not necessarily the correct ones for all patients.

References

1. Misch CE: Implant quality scale: a clinical assessment of the health disease continuum, *Oral Health* 88:15-25, 1998.
2. Ten Bruggenkate C, Van der Kwast WAM, Oosterbeek HS: Success criteria in oral implantology: a review of the literature, *Int J Oral Implantol* 7:45-53, 1990.
3. Council on Scientific Affairs: Dental endosseous implants: an update, *J Am Dent Assoc* 129:1238-1239, 1996.
4. American Dental Association acceptance program for endosseous implants: *Council on Scientific Affairs* [revised July 1993], Chicago, 1993, American Dental Association.
5. Smith DC, Zarb GA: Criteria for success of osseointegrated endosseous implants, *J Prosthet Dent* 62:567-572, 1989.
6. Schnitman PA, Shulman LB: Recommendations of the consensus development conference on dental implants, *J Am Dent Assoc* 98:373-377, 1979.
7. Cranin AN, Silverbrand H, Sher J et al: The requirements and clinical performance of dental implants. In Smith DC, Williams DF, editors: *Biocompatibility of dental materials*, vol 4, Boca Raton, Fla, 1982, CRC Press.
8. McKinney RV, Koth DC, Steflik DE: Clinical standards for dental implants. In Clark JW, editor: *Clinical dentistry*, Harperstown, Pa, 1984, Harper & Row.
9. Albrektsson T, Zarb GA, Worthington P et al: The long-term efficacy of currently used dental implants: a review and proposed criteria of success, *Int J Oral Maxillofac Implants* 1:1-25, 1986.
10. Albrektsson T, Zarb GA: Determinants of correct clinical reporting, *Int J Prosthodont* 11:517-521, 1998.
11. Albrektsson D, Isidor F: Consensus report session IV. In Lang NP, Karring T, editors: *First European workshop on periodontology*, London, 1994, Quintessence.
12. MDBR dental implants: benefit and risks—a National Institutes of Health–Harvard consensus development conference, Pub 81:1531, Washington, DC, 1980, US Department of Health and Human Services.
13. Schwartz-Arad D, Herzberg R, Levin L: Evaluation of long-term implant success, *J Periodontol* 76:1623-1628, 2005.

WWW.HIGHDENT.IR

14. Goodacre CJ, Bernal G, Rungcharassaeng K et al: Clinical complications in fixed prosthodontics, *J Prosthet Dent* 90:31-41, 2003.
15. Parfitt GS: Measurement of the physiologic mobility of individual teeth in an axial direction, *J Dent Res* 39:608-612, 1960.
16. Sekine H, Komiyama Y, Hotta H et al: Mobility characteristics and tactile sensitivity of osseointegrated fixture-supporting systems. In Van Steenberghe D, editor: *Tissue integration in oral maxillofacial reconstruction,* Amsterdam, 1986, Excerpta Medica.
17. Rudd KD, O'Leary TJ, Stumpf AJ: Horizontal tooth mobility in carefully screened subjects, *Periodontics* 2:65-68, 1964.
18. Muhlemann HR, Tooth mobility: a review of clinical aspects and research findings, *J Periodontol* 38:686-708, 1967.
19. Adell R, Lekholm U, Rockler B et al: A 15-year study of osseointegrated implants in the treatment of the edentulous jaw, *Int J Oral Surg* 10:387-416, 1981.
20. Steflik DE, Koth DC, McKinney RV Jr: Human clinical trials with the single crystal sapphire endosteal dental implant: three year results, statistical analysis, and validation of an evaluation protocol, *J Oral Implantol* 13:39-53, 1987.
21. Komiyama Y: Clinical and research experience with osseointegrated implants in Japan. In Albrektsson T, Zarb G, editors: *The Brånemark osseointegrated implant,* Chicago, 1989, Quintessence.
22. Rangert B, Gunne J, Sullivan DY: Mechanical aspects of Brånemark implant connected to a natural tooth: an in vitro study, *Int J Oral Maxillofac Implants* 6:177-186, 1991.
23. Fenton AH, Jamshaid A, David D: Osseointegrated fixture mobility, *J Dent Res* 66:114, 1987.
24. Oh TJ, Yoon J, Misch CE et al: The causes of early implant bone loss: myth or science, *J Periodontol* 73:322-333, 2002.
25. Misch CE: Early crestal bone loss etiology and its effect on treatment planning for implants, *Postgrad Dent* 2:3-17, 1995.
26. Rateitschak KH: Periodontology. In Rateitschak KH, Rateitschak EM, Wolf HF et al, editors: *Color atlas of dental medicine,* ed 2, New York, 1989, Thieme.
27. Jividen G, Misch CE: Reverse torque testing and early loading failures: help or hindrance, *J Oral Implantol* 26: 82-90, 2000.
28. Ivanoff CJ, Sennerby L, Lekholm U: Reintegration of mobilized implants: an experimental study in rabbit tibia, *Int J Oral Maxillofac Surg* 26:310-315, 1997.
29. Teerlinck J, Quirynen M, Darius MS et al: Periotest, an objective clinical diagnosis of bone apposition towards implants, *Int J Oral Maxillofac Implants* 6:55-61, 1991.
30. Chai JY, Yamada J, Pang IC: In vitro consistency of the Periotest instrument, *J Prosthodont* 2:9-12, 1993.
31. Manz MC, Morris HF, Ochi S: An evaluation of the Periotest system. I. Examiner reliability and repeatability of readings, *Implant Dent* 1:142-147, 1992.
32. May KB, Edge MJ, Lang BR et al: The Periotest method: implant supported framework precision of fit evaluation, *J Prosthodont* 5:206-213, 1996.
33. Meredith N, Alleyne D, Cauley P: Quantitative determination of the stability of the implant tissue interface using resonance frequency analysis, *Clin Oral Implants Res* 7:261-267, 1996.
34. Lachmann S, Yves Laval J, Jager B et al: Resonance frequency analysis and damping capacity assessment, *Clin Oral Implants Res* 17:80-84, 2006.
35. Misch CE, Suzuki JB, Misch-Dietsh FD et al: A positive correlation between occlusal trauma and peri-implant bone loss—literature support, *Implant Dent* 14:108-116, 2005.
36. Berglundh T, Lindhe J, Ericsson I et al: The soft tissue barrier at implants and teeth, *Clin Oral Implants Res* 2:81-90, 1991.
37. Barboza EP, Caula AL, Carvalho WR: Crestal bone loss around submerged and exposed unloaded dental implants: a radiographic and microbiological discipline study, *Implant Dent* 11:162-169, 2002.
38. Herrmann JS, Cochran DL, Nummikoski PV et al: Crestal bone changes around titanium implants: a radiographic evaluation of unloaded non-submerged and submerged implants in the canine mandible, *J Periodontol* 68:1117-1130, 1997.
39. Tal H: Spontaneous early exposure of submerged implants. I. Classification and clinical observations, *J Periodontol* 70:213-219, 1999.
40. Abrahamsson I, Berglundh T, Lindhe J: The mucosal barrier following abutment dis/reconnection: an experimental study in dogs, *J Clin Periodontol* 24:568-572, 1997.
41. Cochran DL, Herman JS, Schenk RK et al: Biologic width around titanium implants: a histometric analysis of the implanto-gingival junction around unloaded and loaded submerged implants in the canine mandible, *J Periodontol* 68:186-198, 1997.
42. Herrmann JS, Buser D, Schenk RK et al: Crestal bone changes around titanium implants. A histometric evaluation of unloaded non-submerged and submerged implants in the canine mandible, *J Periodontol* 71:1412-1424, 2000.
43. Kline R, Hoar JE, Beck GH et al: A prospective multicenter clinical investigation of a bone quality-based dental implant system, *Implant Dent* 11:224-34, 2002.
44. Steflik DE, McKinney RV, Koth DL: Ultrastructural (TEM) observations of the gingival response to the single crystal sapphire endosteal implant, *J Dent Res* 61:231, 1982.
45. Cox JF, Zarb GA: The longitudinal clinical efficacy of osseointegrated implants: a 3-year report, *Int J Oral Maxillofac Implants* 2:91-100, 1987.
46. Van Steenberghe D, Lekholm U, Bolender C et al: Applicability of osseointegrated oral implants in the rehabilitation of partial edentulism: a prospective multicenter study on 558 fixtures, *Int J Oral Maxillofac Implants* 5:272-281, 1990.
47. Quirynen M, Naert I, van Steenberghe D: Fixture design and overload influence marginal bone loss and fixture success in the Brånemark system, *Clin Oral Implants Res* 3:104-111, 1992.
48. DeLuca S, Zarb G: The effect of smoking on osseointegrated dental implants. Part II: Periimplant bone loss, *Int J Prosthodont* 19:560-566, 2006.
49. White SC, Pharoah M: *Oral radiology: principles and interpretation,* ed 5, St Louis, 2004, Mosby.
50. Gröndahl K, Ekestubbe A, Gröndahl HG: *Radiography in oral endosseous prosthetics,* Goteborg, Sweden, 1996, Nobel Biocare AB.
51. McAllister BS, Masters D, Meffert RM: Treatment of implants demonstrating periapical radiolucencies, *Pract Periodontics Aesthet Dent* 4:37-41, 1992.
52. Piattelli A, Scarano A, Piattelli M: Abscess formation around the apex of a maxillary root form implant: clinical and microscopical aspects—a case report, *J Periodontol* 66:899-903, 1995.

WWW.HIGHDENT.IR

53. Lang NP, Loe H: The relationship between the width of keratinized gingiva and gingival health, *J Periodontol* 43:623-627, 1972.
54. Wennstrom JL: Lack of association between width of attached gingiva and development of soft tissue recession: a 5-year longitudinal study, *J Clin Periodontol* 14:181-184, 1987.
55. Kennedy J, Bird W, Palcanis K et al: A longitudinal evaluation of varying widths of attached gingiva, *J Clin Periodontol* 12:667, 1985.
56. Miyasato M, Crigger M, Egelberg J: Gingival condition in areas of minimal and appreciable width of keratinized gingiva, *J Clin Periodontol* 4:200-209, 1977.
57. Stetler K, Bissada NF: Significance of the width of keratinized gingiva on the periodontal status of teeth with submarginal restoration, *J Periodontol* 58:696-700, 1987.
58. Valderhaug J, Birkeland JM: Periodontal conditions in patients 5 years following insertion of fixed prostheses. Pocket depth and loss of attachment. *J Oral Rehab* 3: 237-243, 1976.
59. Schroeder A, Pohler O, Sutter F: Tissue reaction to a titanium hollow cylinder implant with titanium plasma sprayed surface, *Schweiz Monatsschr Zahnmed* 86:713-727, 1976.
60. Becker W, Becker BE, Newman MG et al: Clinical microbiologic findings that may contribute to dental implant failure, *Int J Oral Maxillofac Implants* 5:31-38, 1990.
61. Strub JR, Gaberthuel TW, Grunder U: The role of attached gingiva in the health of peri-implant tissue in dogs: clinical findings, *Int J Periodontics Restorative Dent* 11:317-333, 1991.
62. Wennstrom JL, Bengazi F, Lekholm U: The influence of the masticatory mucosa on the peri-implant soft tissue condition, *Clin Oral Implants Res* 5:1-8, 1994.
63. Krekeler G, Schilli W, Diemer J: Should the exit of the artificial abutment tooth be positioned in the region of the attached gingiva? *Int J Oral Surg* 14:504-508, 1985.
64. Adell R, Lekholm U, Rockler G et al: Marginal tissue reactions at osseointegrated titanium fixtures. I. A 3-year longitudinal prospective study, *Int J Oral Maxillofac Implants* 15:39-52, 1986.
65. Lekholm U, Adell R, Lindhe J et al: Marginal tissue reactions at osseointegrated titanium fixtures. II. A cross-section retrospective study, *Int J Oral Maxillofac Surg* 15:53-61, 1986.
66. Nevins M, Kenney E, van Steenberghe D et al, editors: Consensus report: implant therapy II. Proceedings of the 1996 World Workshop in Periodontics, *Ann Periodontol* 1:816-820, 1996.
67. Warrer K, Buser D, Lang NP et al: Plaque-induced peri-implantitis in the presence or absence of keratinized mucosa: an experimental study in monkeys, *Clin Oral Implants Res* 6:131-138, 1995.
68. Kirsch A, Ackermann KL: The IMZ osteointegrated implant system, *Dent Clin North Am* 33:733-791, 1989.
69. James RA, Schultz RL: Hemidesmosomes and the adhesion of junctional epithelial cells to metal implants: a preliminary report, *J Oral Implantol* 4:294, 1974.
70. Listgarten M, Lang NP, Schroeder HE et al: Periodontal tissues and their counterparts around endosseous implants, *Clin Oral Implants Res* 2:81-90, 1991.
71. Rapley JW, Mills MP, Wylam J: Soft tissue management during implant maintenance, *Int J Periodontics Restorative Dent* 12:373, 1992.
72. Ono Y, Nevins M, Cappetta M: The need for keratinized tissue for implants. In Nevins M, Mellonig JT, editors: *Implant therapy,* Chicago, 1998, Quintessence.
73. Meffert RM, Langer B, Fritz ME: Dental implants: a review, *J Periodontol* 63:859-870, 1992.
74. Block MS, Kent JN: Factors associated with soft- and hard-tissue compromise of endosseous implants, *J Oral Maxillofac Surg* 48:1153-1160, 1990.
75. Deporter DA, Watson PA, Pilliar RM et al: A histological evaluation of a functional endosseous, porous-surfaced, titanium alloy dental implant system in the dog, *J Dent Res* 67:1190-1195, 1988.
76. Zablotsky M. The surgical management of osseous defects associated with endosteal hydroxyapatite-coated and titanium dental implants, *Dent Clin North Am* 36:117-149; discussion 49-50, 1992.
77. Kirsch A, Mentag P: The IMZ endosseous two phase implant system: a complete oral rehabilitation treatment concept, *J Oral Implantol* 12:576-589, 1986.
78. Chung DM, OH TJ, Shotwell JL et al: Significance of keratinized mucosa in maintenance of dental implants with different surfaces, *J Periodontol* 77:1410-1420, 2006.
79. Esposito M, Coulthard P, Thomsen P et al: The role of implant surface modification, shape and material on the success of osseointegrated dental implants: a Cochrane systematic review, *Eur J Prosthodontics Restorative Dent* 13: 15-31, 2005.
80. Gargiulo A, Wentz F, Orban B: Dimensions and relations of the dentogingival junction in humans, *J Periodontol* 32:261-268, 1961.
81. Vacek JS, Gher ME, Assad DA et al: The dimensions of the human dentogingival junction, *Int J Periodontics Restorative Dent* 14:154-165, 1994.
82. Ericsson I, Lindhe J: Probing at implants and teeth: an experimental study in the dog, *J Clin Periodontol* 20:623-627, 1993.
83. Villata L, Baelum V: Reproducibility of attachment level recordings using an electronic and a conventional probe, *J Periodontol* 67:1292-1300, 1996.
84. Best AM, Burmeister JA, Gunsolley JC et al: Reliability of attachment loss measurements in a longitudinal clinical trial, *J Clin Periodontol* 17:564-569, 1990.
85. Page RC: Summary of outcomes and recommendations of the workshop on CPITN, *Int Dent J* 44:589-594, 1994.
86. Hunter FM: Development of a disposable pressure sensitive periodontal probe and comparison of probing forces [abstract], *J Dent Res* 69:277, 1990.
87. Rams TE, Slots J: Comparison of two pressure sensitive periodontal probes and a manual periodontal probe in shallow and deep pockets, *Int J Periodontics Restorative Dent* 13:521-529, 1993.
88. Mombelli A, Van Oosten MAC, Schurch E et al: The microbiota associated with successful or failing osseointegrated titanium implants, *Oral Microbiol Immunol* 2: 145-151, 1987.
89. Rams TE, Roberts TW, Tatum H Jr et al: The subgingival microflora associated with human dental implants, *J Prosthet Dent* 5:529-534, 1984.
90. Stefani LA: The care and maintenance of the dental implant patient, *J Dent Hygiene* 62:447-466, 1988.
91. Quirynen M, Naert I, Teerlinck J et al: Periodontal indices around osseointegrated oral implants supporting overdentures. In Schepers E, Naert J, Theunier G, editors: *Overdentures on oral implants,* Leuwen, Belgium, 1991, Leuwen University Press.

WWW.HIGHDENT.IR

92. Deporter HS, Friedland B, Watson P et al: A clinical and radiographic assessment of a porous surface titanium alloy dental implant in dogs, *Int J Oral Implantol* 4:31-37, 1987.
93. Jepsen S, Ruhling A, Jepsen K et al: Progressive peri-implantitis. Incidence and prediction of peri-implant attachment loss, *Clin Oral Implants Res* 7:133-42, 1996.
94. Berglundh T, Lindhe T, Jonsson K et al: The topography of the vascular systems in the periodontal and peri-implant tissues in the dog, *J Clin Periodontol* 21:189-193, 1994.
95. Lindhe J: *Textbook of clinical periodontology*, Copenhagen, 1983, Munksgaard.
96. Loe J, Silness J: Periodontal disease in pregnancy: prevalence and severity, *Acta Odontol Scand* 21:532-551, 1963.
97. Bauman GR, Rapley JW, Hallmon WW et al: The peri-implant, *Int J Oral Maxillofac Implants* 8:273-280, 1993.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Chapter 42

Maintenance of Dental Implants: Implant Quality of Health Scale

Jon B. Suzuki, Carl E. Misch, Diana Bronstein, Lynn D. Terracciano-Mortilla

Implant dentistry has evolved into a well-understood clinical science with expansive documented research to validate what, years ago, many believed to be experimental. There has been an incredible amount of focus on the biology and biomechanics of implant dentistry, and results have helped to develop and refine techniques based on proven data rather than unsubstantiated trial-and-error procedures. The evolution of research and understanding of biological concepts in implant dentistry has caused many areas of conflict and controversy. Theories and techniques have developed and changed, and the types of materials used in implant dentistry have changed dramatically.

The tremendous growth in this field created new ideas and terminology that are either undefined or being redefined based on new knowledge.[1] What one study proves in certain cases, another may completely disprove. It can become confusing for a clinician to know the correct protocols, procedures, tools, and techniques to use. As materials and techniques are researched and developed, previous theories may undergo criticism and controversy. It does not necessarily negate the findings of the past; however, we must modify techniques and tools to keep current with the changes in technology and research. One area of expansion of knowledge and conflict of views relates to the maintenance of dental implants. Early research explored techniques and instruments that were current for the methods and materials of that time. Although many of those implants still exist and are functional in patients, research and advances in technology have given us newer materials and advances in implant design and structure that do not necessarily possess the same challenges from a maintenance perspective.

A thorough review of mucoepithelial attachment is essential before commencing any maintenance procedures. Controversies and parameters for probing and radiograph exposure essential to clinicians are also discussed here. Thorough reviews of the tooth versus implant differences, as well as identification of the bacterial similarities and differences, also are discussed. The literature review explores the success or failure of implants and discusses in length the parameters that need to be adhered to when evaluating implants and the peri-implant tissue.

Once the clinician understands the parameters of implants and teeth, a specific plan can be created for the patient and the clinician can advise what is expected during each phase of treatment and demonstrate various oral hygiene options to suit the patient's needs during each stage of therapy. Patients must be thoroughly educated and retrained before deciding to commence implant therapy.[2] The ability of the patient to understand financial, time, and maintenance obligations is crucial and must be made clear to the patient initially and during subsequent appointments. In addition to educating patients, clinicians will need to assess compliance with a home care routine[3] and must possess the clinical competencies to perform maintenance procedures. As the number of dental implants placed in patients continues to increase, there is a need to understand the importance of maintenance as it relates to long-term implant success.[4] The role of the dental hygienist in implant maintenance and care is increasing and becoming more defined[5] (Box 42-1).

Implants and their prosthetic devices are different from natural teeth and may require different procedures and instruments for both professional and patient care.[6] Instruments must be effective at removing biofilms and accretions, and procedures performed by patients and/or clinicians should avoid damage to all portions of the implant, abutment, restoration, and tissues.[7] Establishment and maintenance of a soft tissue seal around the transmucosal portion of the implant can enhance the success of an implant. This barrier is fundamentally a result of wound healing. The maintenance of healthy peri-implant tissues may contribute to implant success factors. Tissues and an implant sulcus

WWW.HIGHDENT.IR

Box 42-1 Role of the Dental Hygienist in Implant Maintenance

- Identification of potential implant patients
- Education and motivation throughout treatment
- Development, continual assessment, and modification of patient-specific oral hygiene procedures
- Evaluation of prosthesis (components, attachments, mobility, and retention)
- Evaluation of peri-implant tissue
- Probing
- Exposure of clinically acceptable radiographs
- Removal of biofilms, soft and hard accretions
- Recommendation of oral hygiene implements
- Determination of a patient specific recall interval
- Cotherapist to identify potential problems or complications
- Documentation of implant status

Box 42-2 Plaque Biofilm Development and Colonization

Identify potential implant patients
↓
Bacterial adhesion
↓↓
Supragingival plaque
Gram+, streptococci, Actinomyces
↓↓↓
Plaque maturation (gram-, rods, and filaments)
↓↓↓↓
Well-differentiated subgingival plaque
(Gram-, anaerobes)

free of inflammation and infection, as well as an absence of virulent bacteria, can enhance a patient's general and oral health.

PLAQUE BIOFILM AND DENTAL IMPLANTS

The differences between tooth and implant biologies make dental implants more susceptible to inflammation and bone loss in the presence of bacterial plaque accumulation.[8] Biofilms are the primary causative factor of periodontal disease processes. Sticky masses of bacteria with a polysaccharide matrix, water, and bacteria accumulate on hard and soft surfaces in the oral cavity and can be disturbed and removed with mechanical or chemical obliteration. If undisturbed, mature plaque will form. Current chemotherapeutics cannot penetrate thick biofilm, and rough surfaces have been found to hold more biofilm[9] than smooth surfaces. Bacteria will migrate from teeth to implants and from implant to implant.[10] Similar to teeth, clinical findings of failing implants include inflammation, pockets, and progressive bone loss.[11] Another similarity lies in the bacteria responsible for periodontitis and peri-implantitis.

When evaluating the peri-implant microbiota, Lee et al.[12] compared microbial changes between patients with a history of periodontal or peri-implant infections and implants that have been in function for a length of time. This study found a history of periodontitis had a greater impact on the peri-implant microbiota than implant-loading time. The major influence on the peri-implant microbiota was, however, the microbiota on remaining teeth. *Porphyromonas gingivalis* and *Bacteroides forsythus*, red complex periodontal pathogens, colonized several implants, although all implants were successfully osteointegrated. Thus it is important to educate patients about their responsibility to decrease plaque effectively, especially if they have a history of periodontal disease.

Plaque biofilm development and maturation have similarities for natural teeth and dental implants. The gingival sulcus in periodontal health and the perimucosal attachment of a successful dental implant are essentially similar.[13] In a study by Mombelli and Mericske-Stern of the plaque from 18 edentulous patients with successful dental implants, facultative anaerobic cocci (52.8%) and facultative anaerobic rods (17.4%) were reported. However, the pathogens *P. gingivalis* and spirochetes were absent, and minimal (7.3%) gram-negative rods were present.[13]

Generally, pellicle—a naturally occurring glycoprotein in the saliva—first adheres to the intraoral structure, whether it be a tooth or an implant. Gram-positive cocci bacteria are the first "early colonizers," beginning with single cocci and progressing to streptococci forms (Box 42-2).

Without appropriate oral hygiene measures (e.g., brushing, flossing, interdental cleaning), additional bacteria colonies including gram-negative rod-shaped bacteria synergistically grow with the established gram-positive bacteria. The gram-negative bacteria are frequently facultative or strict anaerobic bacteria and are considered "late colonizers." Many, if not the majority, of these gram-negative bacteria are black pigmented and are classified under a number of genera (e.g., *Bacteroides, Prevotella, Porphyromonas, Fusobacterium*).

Plaque biofilm reported to be associated with failing dental implants also consists largely of gram-negative rods.[14] Clinically, failing dental implants are characterized by soft tissue inflammation, increased probing depths, increased mobility, and peri-implant radiolucency. Specific pathogens in implant pockets greater than 6 mm include *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia*, and *P. gingivalis*, in more than one third of the sites, as confirmed by DNA analysis.[15]

More specific studies on plaque biofilm around dental implants suggest similarities[16] between periodontal

WWW.HIGHDENT.IR

diseases and failing implants, but differences have also been reported.[17,18] Mombelli[18] did not detect spirochetes in plaque samples from well-maintained and clinically healthy implants. Rams et al. noted higher proportions of staphylococci (15.1%)[19] than usually found in gingivitis (0.06%) and periodontitis (1.2%) sites.[19] This finding suggests that staphylococci may be more significant in developing peri-implantitis lesions than previously recognized.

Changes involve both the hard and soft tissues surrounding an implant. The implant may exhibit all the signs of peri-implant mucositis, as well as exudate, increase of pocket depth, and bone loss. If left untreated, significant bone loss, infection, and mobility could result, leading to the failure of an initially integrated implant.

Comparisons of plaque biofilms have been reported in a limited study of Brånemark and ITI (Straumann Institute) implants and are remarkably similar in controlled studies. Mombelli et al.[20] compared 10 patients with Brånemark implants and 10 patients with ITI implants and sampled the deepest pockets around the implants. After 3 and 6 months, several periodontal pathogens were cultured and isolated, including *P. gingivalis*, *P. intermedia*, *Fusobacterium nucleatum*, and various spirochetes. None of the implants was colonized by *A. actinomycetemcomitans*. Longer investigations by Leonhardt et al.[21] extended these microflora reports on dental implants in 19 patients. At 3 years, the osteointegrated implants were colonized predominantly by *P. gingivalis*, *P. intermedia*, and *A. actinomycetemcomitans*.

Natural dentitions with dental implants appear to increase the risk for implant infections, compared with completely edentulous patients. This suggests that natural teeth may serve as a reservoir for periodontal pathogens that may extend their growth to contiguous implants in the same oral cavity.[22] Quirynen and Listgarten[23] reported that proportions of coccoid forms (65.8%), motile rods (2.3%), and spirochetes (2.1%) in implant pocket areas were similar to the microorganisms in natural teeth (55.6%, 4.9%, and 3.6%, respectively).

Fully edentulous patients exhibited more coccoid forms (71.3%), fewer motile rods (0.4%), and no spirochetes. Quirynen and Listgarten[23] concluded that microflora in partially edentulous implant patients were potentially more pathogenic than fully edentulous patients. Implants with longevity of more than 3 or 4 years appear to have greater numbers of bacteria than implants in place for 1 or 2 years.[24]

Peri-implant mucositis is an inflammatory change of the soft tissue surrounding an implant. Peri-implant mucositis around an implant is similar to gingivitis around a tooth. There is no loss of attachment apparatus for teeth with gingivitis nor loss of bone for implants with peri-implant mucositis. The primary etiology is plaque biofilm. Like gingivitis, peri-implant mucositis is reversible once the etiologic agent, plaque biofilm, is removed. If allowed to progress, peri-implantitis may result, which includes loss of osteointegration, similar to loss of attachment and bone with periodontitis.

SOFT TISSUE INTERFACE

Patient Oral Hygiene

Removal of supragingival plaque with use of toothbrushes can significantly reduce the amount and composition of the subgingival microbiota. This reduction should translate to decreased risk of periodontal disease initiation or recurrence. Furthermore, the decreased prevalence of periodontal pathogens in supragingival plaque lowers potential reservoirs of these species.[25]

The absence of adequate keratinized mucosa in endosseous dental implants, especially in posterior implants, was associated with higher plaque accumulation and gingival inflammation, but not with more annual bone loss, regardless of their surface configurations.[26] The implant type, with the presence or absence of keratinized tissue, may be a challenge for oral hygiene procedures for many patients. The clinician should stress the importance of adequately performing plaque control and select products and procedures well suited to the needs and ability of the patient.

Patients rely on clinicians to suggest or recommend products for oral hygiene procedures. As with most patients, the "tell-show-do" method of home care instruction is important. Documentation in the patient record regarding recommendations and instructions, as well as the patient's compliance and effectiveness, will be important to evaluate long-term success for each patient.[27,28] When choosing and recommending implements for oral hygiene, the clinician should take into consideration the location, length, and angulations of abutments, superstructure design, anatomical limitations, patient habits, motivation, and manual dexterity of each patient.[29]

Contributing factors that may influence product selection are plaque and calculus accumulation, as well as the general health of the patient (including diseases and medications). To avoid patients becoming discouraged and poorly motivated, it is wise to keep oral hygiene instructions simple.[30,31] Partially edentulous patients exhibit higher pathogenic bacterial counts than edentulous patients, which may cause seeding of pathogenic bacteria from one site to another.[32]

The final prosthesis should allow for access by the patient and clinician to keep the areas plaque free.[33] The clinician should instruct the patient in the use of toothbrushes (manual or automatic); floss (with threading devices, if necessary); tufted brushes; interdental brushes (with coated wires); toothpicks; and oral irrigators. Patient instructions may include the use of antimicrobials, such as cetylpyridium chloride (Crest Pro-Health, Proctor and Gamble, Cincinnati,

WWW.HIGHDENT.IR

Ohio) or chlorhexidine gluconate (Peridex, OMNII-3M, West Palm Beach, Fla.; Perioguard, Colgate, New York, N.Y.) due to substantivity and ability to inactivate oral bacteria.[34] Chlorhexidine gluconate can be used as a rinse or applied specifically to the site with brushes or cotton swabs.

If oral irrigation is used, the patient should be instructed to use the lowest setting and direct the irrigation flow through the contacts to avoid excessive pressure to the implant tissue cuff. Incorrect use could alter tissue adaptation and induce bacteremia around the implant.[35] Additionally, clinicians should recommend that the patient be gentle, yet thorough, postsurgically to avoid complications of healing from aggressive hygiene procedures.

INSTRUMENT SELECTION

Maintaining a smooth surface of titanium without pits and scratches can prevent plaque accumulation.[36] Instrument selection should depend on tip designs that are not bulky (to avoid unnecessary tissue manipulation), and the design should facilitate manipulation by the clinician. A clinician may also evaluate the prosthesis design, location of deposits, and tenacity of calculus to help select appropriate instruments.

Metallic ultrasonic and sonic scalers have been reported to gouge titanium.[37] A plastic or rubber sleeve over an ultrasonic scaler appears not to alter titanium.[38] Conventional ultrasonic scalers with a nonmetal tip also are suitable for implant maintenance.[39] Air polishers are effective and safe for maintenance procedures around implants.[40,41]

Stainless steel–tipped instruments have been found to be detrimental to a smooth titanium surface.[42] This is important to consider when sharpening implant hygiene instruments to ensure a sharpening stone free of steel scrapings. A variety of nonmetallic, plastic, graphite, nylon, or Teflon-coated instruments are available and have been proven to be safe to use on titanium implant surfaces.[37-49] Improved gingival and soft tissue architecture result from scaling with these instruments. A titanium curette and a rubber cup with flour of pumice are suitable for cleaning implant surfaces.[43,44]

After the removal of calculus, polishing with a rubber cap and toothpaste, fine prophy paste, commercial implant polishing pastes, and tin oxide have been shown to be safe for titanium surfaces.[45,46] A rubber point or soft untufted rotary brush can also be used.[47]

The primary purpose of instruments used is to thoroughly remove plaque and calculus. Ramaglia et al. demonstrated that a plastic curette and air-powder-water spray did not alter the implant surface, but these instruments may leave deposits. These deposits should be removed by irrigation to avoid any adverse tissue healing.[50]

IMPLANT MAINTENANCE PROCEDURES

As dental implants become more widely used as replacements for missing teeth, clinicians will certainly see increases in clinical problems in complex cases.[14] Frequent recall visits after implant placement and restorations are necessary for evaluation and establishment of good oral hygiene after treatment. Healthy tissue should have no inflammation with a primary etiology of plaque and calculus formation. The recall visit also is a time to detect potential problems to encourage early intervention should a problem arise. An appropriate periodontal probe should be used for pocket measurements.

Unlike the attachment to the porosities of teeth, the adherence and tenacity of calculus around implants are usually less binding. This can decrease the chance of traumatizing the tissue and perimucosal seal during procedures to remove deposits. With adequate oral hygiene, subgingival calculus should be minimal, if present at all. Because titanium usually does not have calculus embedded into it, adequate oral hygiene should prevent the accumulation of heavy accretions. If crestal bone loss has occurred, the type of implant surface or coating exposed may encourage plaque or calculus adherence.

Healthy implant tissues may have a tight seal and can create challenges while scaling. Because the perimucosal seal is more fragile than a normal tooth sulcus, it is important to use short, exploratory working strokes with light pressure. Depending on the location of the calculus, a horizontal, vertical, or oblique stroke may need to be used while avoiding tissue trauma.[51] When an instrument must be used subgingivally to remove calculus or excess cement, insertion and instrumentation should be gentle and light strokes should be in a semicircular pattern. Attention to placing the blade carefully under the deposit and drying calculus or cement with compressed air may make detection and removal easier and more comfortable for the patient.

Poor tissue tone (i.e. flaccid, friable tissue) around an implant abutment can harbor food, plaque, and calculus and increase the occurrence of inflammation and infection. If maintenance and hygiene procedures cannot adequately improve tissue tone, surgical correction may be necessary to reduce chronic inflammation and infection.

Hygiene procedures performed by the clinician and patient can be limited by prosthetic designs that have bulky restorations and inadequate embrasures, preventing ready access to the implant–gingival margin interface area.[52] Because loss of osseous support is a factor in implant failure, problems that may arise from a prosthesis may prove difficult to maintain in the treatment planning phase.[53] Periodic occlusion monitoring may detect discrepancies to indicate occlusal changes needed.[54]

WWW.HIGHDENT.IR

CHEMOTHERAPEUTIC AGENTS

Chlorhexidine gluconate has been shown to reduce plaque in the oral cavity and around dental implants.[55] Long-term use of antimicrobials such as chlorhexidine gluconate, cetylpyridium chloride, or phenolic compounds may be used along with brushes and floss to minimize staining.[20] It is significant to note that the alcohol in newer generations of chlorhexidines serves to preserve and stabilize the solution.[56] Studies have shown a decrease of antimicrobial benefits in preparations that are alcohol free.[56] If the clinician uses subgingival irrigation, the cannula should be inserted carefully in the peri-implant tissue to avoid gouging the surface. Care should be taken to avoid inserting the cannula to the base of the implant sulcus to prevent fluid distention into surrounding tissues.[57] Chlorhexidine gluconate has proved to be a useful irrigant.[58] It is also wise to use a neutral sodium fluoride in a patient with dental implants because certain acidic fluorides can alter titanium.[59,60]

A study on nonsurgical mechanical treatment on sites with peri-implantitis lesions with microencapsulated minocycline (Arestin, Orapharma, Johnson and Johnson, Warminster, Pa.) and 0.12% chlorhexidine gel found reductions of pocket depths and bleeding on probing for as long as 12 months.[61] Local antibiotic therapies also have proved successful in the treatment of peri-implantitis.[62]

The effect of mechanical treatment alone or combined with antiseptic therapy on peri-implant tissues translated into less inflammation, as evaluated histomorphometrically, when compared with nontreated controls. The proportion of inflammation found in the mucosal tissues of the control implants was greater than the proportion found for both treatment groups. More importantly, both treatment groups showed similar low proportions of inflammation after 2 months of treatment. Thus it can be concluded that mechanical treatment alone or combined with chlorhexidine, sustained by plaque control, results in inflammation compatible with health.[63]

Systemic antibiotics also may be useful to treat infection. Assessing the problem, complication, or condition to be treated is as important as knowing the cause.[64]

IMPLANT QUALITY OF HEALTH SCALE: A CLINICAL ASSESSMENT OF THE HEALTH-DISEASE CONTINUUM

The criteria for success in implant dentistry remain complex. The vast majority of clinical studies reporting success and failure do not qualify the type of success achieved. Instead, the term *success* primarily has been used interchangeably with survival of the implant. The term *failure* has been used to indicate the implant is no longer present in the mouth. Nearly all reports in the prosthetic literature also report success as survival. What is success for a natural tooth? In the periodontal literature, a quality of health is presented, and well-established guidelines based on clinical criteria describe the ideal health of natural teeth. The general term *success in implant dentistry* should be replaced with the concept of *quality of health,* with a health-disease continuum describing the status of implants.

Periodontal indices often are used to evaluate dental implants; however, the latter are fundamentally different because they do not decay, have no dental pulps to function as early indicators of disease, and have no periodontal membrane. In addition, the terms used to describe periodontal disease conditions are often inappropriate when applied to implants. As the mechanisms, causative factors, pathogenesis, and host factors are better understood, the descriptions of implant-related diseases have evolved. Success criteria for endosteal implants have been proposed previously by other authors, including Schnitman and Shulman,[65] Cranin et al.,[66] McKinney et al.,[67] Albrektsson et al.,[68] and Smith and Zarb.[69] As Box 42-3 shows, the report by Albrektsson et al.[68] was specific for implants with rigid fixation and is used widely today. An implant quality of health scale with five levels has been established by James and modified by Misch.[70,71] The James-Misch scale also proposes management modalities corresponding to these five levels. In 2007, a consensus conference in Pisa, Italy (sponsored by the International Congress of Oral Implantologists) modified the James-Misch scale to four conditions that describe success, survival and failure.[71a]

Ideal clinical conditions for natural teeth include absence of pain, less than 0.1 mm of initial horizontal mobility under lateral forces of less than 100 g, less than 0.15 mm secondary mobility with lateral forces

Box 42-3 Criteria for Implant Success

- An individual, unattached implant is immobile when tested clinically.
- A radiograph does not demonstrate any evidence of peri-implant radiolucency.
- Vertical bone loss is less that 0.2 mm annually after the first year of service of the implant.
- Individual implant performance is characterized by an absence of persistent or irreversible signs and symptoms such as pain, infections, neuropathies, paresthesia, or violation of the mandibular canal.
- In the context of the foregoing, a success rate of 85% at the end of a 5-year observation period and 80% at the end of a 10-year period are minimum criteria for success.

From Albrektsson T, Zarb GA, Worthington P et al: The long-term efficacy of currently used dental implants: a review and proposed criteria of success, *Int J Oral Maxillofac Implants* 1:1, 1986.

WWW.HIGHDENT.IR

of 500 g, absence of observed vertical mobility, periodontal probing depths of less than 2.5 mm, radiographic crestal bone height 1.5 to 2.0 mm below the cement-enamel junction, intact lamina dura, no bleeding on probing, no exudate, and absence of recession or furcation involvement on multirooted teeth (Box 42-4).[72] Many of these same criteria are listed as ideal conditions for dental implants.[65-71]

The American Academy of Periodontology has defined five periodontal types for diagnosis and treatment of natural teeth, shown in Table 42-1.[73] The American Academy of Periodontology categories of disease do not indicate strict success or failure, but rather a range from health to disease. This classification allows a clinical approach to treatment in each category. A similar scale for implants has been established as an aid to diagnosis and treatment that also proposes management approaches according to the signs and symptoms.[70]

Box 42-4 Ideal Clinical Conditions of Teeth

- Absence of pain
- Less than 0.1 mm initial horizontal mobility under lateral forces less than 100 g
- Less than 0.15 mm secondary mobility with lateral forces of 500 g
- Absence of observed vertical mobility
- Periodontal probing depths less than 2.5 mm
- Radiographic crestal bone height 1.5 to 2.0 mm below cement-enamel junction
- Intact lamina dura
- No bleeding on probing
- No exudate
- Absence of recession
- Absence of furcation involvement on multirooted teeth

The scale presented for implant quality of health based on clinical evaluation was agreed upon by the International Congress of Oral Implantologists in 2007[71a] (Table 42-2). This quality of health scale allows the dentist to evaluate an implant using the listed criteria, place it in the appropriate category, and then treat the implant accordingly. The prognosis also is related to the quality scale.

Group I represents implant success with optimum health conditions. No pain is observed with palpation, percussion, or function. No mobility is noted in any direction with loads less than 500 g of implant movement (IM). Less than 2.0 mm of crestal bone has been lost since the placement of the implant. The implant has no history of exudate, and no radiolucency is present around the implant body (Figure 42-1). The probing depth is equal to or less than 5 mm and is stable after the first year. Ideally, the bleeding index is 0 to 1. Group I implants follow a normal maintenance program. Prognosis is very good to excellent.

Table 42-1 Classification of Periodontitis

TYPE	TERMINOLOGY	DESCRIPTION
I	Gingivitis	Inflammation of the gingiva characterized by changes in color, gingival form, position, surface appearance, and appearance and presence of bleeding or exudate
II	Slight periodontitis	Progression of gingival inflammation into deeper periodontal structures and alveolar bone crest with slight bone loss; usual periodontal probing depth of 3 to 4 mm with slight loss of connective tissue attachment and slight loss of alveolar bone
III	Moderate periodontitis	More advanced stage of type II; increased destruction of the periodontal structures, noticeable loss of bone support, possibly accompanied by an increase in tooth mobility; furcation involvement possible in multirooted teeth
IV	Advanced periodontitis	Further progression of periodontitis, with major loss of alveolar bone support, usually accompanied by increased tooth mobility; furcation involvement in multirooted teeth likely
V	Refractory progressive periodontitis	Several unclassified types of periodontitis included that are characterized by rapid bone and attachment loss or by slow but continuous bone and attachment loss; condition resistant to normal therapy and usually associated with gingival inflammation and continued pocket formation

Modified from Council on Dental Care Programs: Reporting periodontal treatment under dental benefit plans, *J Am Dent Assoc* 17:371-373, 1988.

WWW.HIGHDENT.IR

Table 42-2 Implant Quality Scale

GROUP	MANAGEMENT	CLINICAL CONDITIONS
I. Success (optimum health)	Normal maintenance	No pain or tenderness upon function 0 mobility <2 mm radiographic bone loss from initial surgery Probing depth <5 mm No exudate history
II. Survival (satisfactory health)	Reduction of stresses Shorter intervals between hygiene appointments Gingivoplasty Yearly radiographs	No pain 0 mobility 2-4 mm radiographic bone loss Probing depth 5 to 7 mm No exudate history
III. Survival (compromised health)	Reduction of stresses Drug therapy (antibiotics, chlorhexidine) Surgical reentry and revision Change in prosthesis or implants	No pain upon function 0 mobility Radiographic bone loss > 4 mm Probing depth > 7 mm May have exudate history
IV. Failure (clinical or absolute failure)	Removal of implant	Any of the following: Pain upon function Mobility Radiographic bone loss >1/2 length of implant Uncontrolled exudate No longer in mouth

From International Congress of Oral Implantologists, Consensus Conference, Pisa, Italy, 2007.

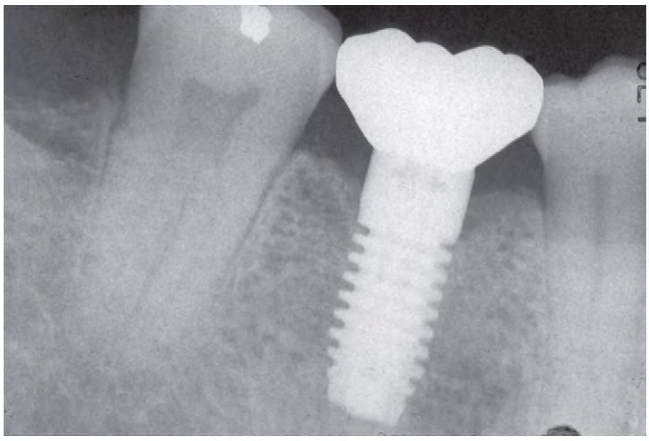


Figure 42-1 Group I represents optimum health conditions around an implant. Less than 1.5 mm of crestal bone loss occurs during the first year of occlusal loading from the time of prosthesis delivery. A periapical radiograph can be obtained to assess mesiodistal bone levels.

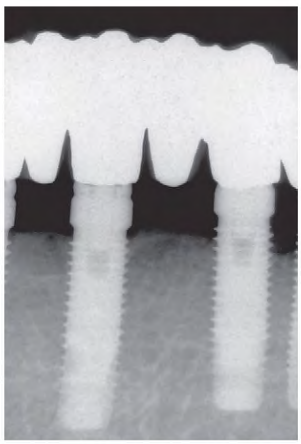


Figure 42-2 Group II represents satisfactory health around an implant. Radiographic bone loss between 1.5 and 3 mm from stage II uncovery is visible on the left implant. The right implant has lost an additional 1 mm of bone since the prosthesis delivery. The probing depths are stable. The tissue was reduced, and the threads above the bone on the right implant were reduced in depth.

Group II implants exhibit satisfactory health and are stable but show a history of or potential for clinical problems. No pain or tenderness is observed on palpation, percussion, or function. No observable mobility exists in the horizontal or vertical direction with loads less than 500 g. Crestal radiographic bone loss is between 2 and 4 mm from implant placement (Figure 42-2). The probing depths may be greater than 5 to 7 mm because of the original tissue thickness and marginal bone loss but are stable. The treatment indicated for group II implants consists of a stress reduction protocol for the implant system, shorter intervals between hygiene appointments, reinforcement of oral hygiene instructions, annual radiographs until the crestal bone has stabilized, and gingivoplasty or sulcus reduction procedures where indicated. The prognosis is good to very good depending on the depth of the implant sulcus.

WWW.HIGHDENT.IR

For pockets 3 to 4 mm in depth, the following can be concluded[63]:

1. Mechanical therapy alone or combined with chlorhexidine results in the clinical resolution of peri-implant mucositis lesions.
2. Histologically, both treatments result in minimal inflammation compatible with health.
3. The mechanical effect alone is sufficient to attain clinical and histologic resolution of mucositis lesions.[63]

Group III implants are also classified as *survival*, but exhibit a slight to moderate peri-implantitis and compromised health status. Peri-implantitis is defined as an inflammatory process affecting the tissue around an implant that results in loss of supporting bone.[75] Group III implants are characterized by radiographically evident vertical bone loss, peri-implant pocket, bleeding on probing (plus suppuration), mucosal swelling and redness, but no pain upon function (Figure 42-3). These implants warrant more aggressive clinical therapy. No pain is apparent in function, but tenderness may be slight on percussion or function. No vertical or initial horizontal mobility (IM-0) is evident. Greater than 4 mm of crestal bone loss has occurred since implant insertion. Greater than 7 mm and increasing probing depths are also present, usually accompanied by bleeding when probing. Bone loss is less than 50% around the implant. Exudate episodes may have lasted more than 1 to 2 weeks and may be accompanied by a slight radiolucency evident around a crestal region of the implant.

Group III implants warrant aggressive surgical and prosthetic intervention. Stress factors are addressed first. The prosthesis may be removed in nonesthetic regions or the bar may be removed under overdentures, especially when IM-2 exists. Modification of the occlusal scheme and methods to decrease the forces in the afflicted regions after hard and soft tissue surgical treatment include decreasing cantilever length, occlusal adjustment, and occlusal splint therapy. In cases of rapid bone changes, the prosthesis design may be modified completely from a fixed to a removable restoration to stress relief and soft tissue support. Additional implants to support the restoration may be indicated, especially if the patient is unwilling to wear a removable prosthesis. When additional implants and a new prosthesis are considered, implants with any detectable movement should be removed.

Systemic and topical antibiotics and local chemical agents such as chlorhexidine are indicated in the presence of exudate. However, this method is usually of short-term benefit if the causative agents of implant failure are not eliminated. Culture and sensitivity tests may be indicated, especially if existing signs and symptoms do not subside within a few weeks. Surgical management most often consists of soft tissue removal or exposure of a portion of the implant (Figure 42-4). Bone grafts may be used along with these approaches around the implant. A three-step approach is implemented for this category in the following order: (1) antimicrobial therapy (local or systemic); (2) stress reduction; and (3) surgical intervention. The prognosis is good to guarded, depending on the ability to reduce and control stress once the surgical corrections have improved the soft and hard tissue health.

Group IV of implant health is clinical or absolute failure (Figure 42-5). The implant should be removed under any of these conditions: (1) pain on palpation, percussion, or function; (2) greater than 0.5 mm of horizontal mobility (IM-3); (3) any vertical mobility (IM-3); (4) uncontrolled progressive bone loss; (5) uncontrolled exudate; (6) more than 50% bone loss around the implant; (7) generalized radiolucency; or (8) implants surgically placed but unable to be restored

Figure 42-3 Group III implants have a comprised health status and warrant a surgical procedure to decrease the risk of further deterioration. Probing on the facial of this implant indicates a 6-mm pocket, and exudate is present.

Figure 42-4 This implant required surgical reentry to decontaminate the surface of the implant and remove the noxious elements. A reduction of thread, depth, and a bone graft or apical-positioned flap was indicated.

WWW.HIGHDENT.IR

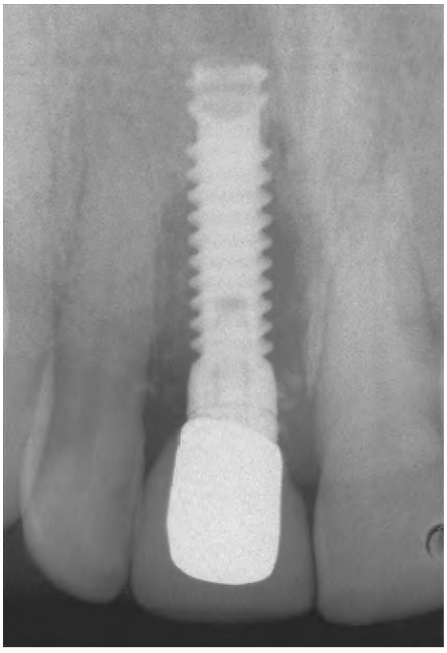


Figure 42-5 The mesial or distal bone height around an implant can be assessed on a periapical radiograph. This radiograph demonstrates bone loss to the apex of the implant. Although the implant exhibits no mobility or discomfort, it should be removed because more than half the implant has crestal bone loss.

(sleepers). Implants that are surgically removed or exfoliated are also in the category of failure. Occasionally, the patient will not permit removal of the implant. Regardless of whether the patient returns for implant removal, the implant is recorded as a failure in all statistical data. The patient should be warned against the irreversible damage to the surrounding bone with implants retained in this condition. Consideration should be given to their removal because future treatment may be compromised.

REPAIR OF THE AILING, FAILING DENTAL IMPLANT

I. If an active infection (purulence, bleeding, swelling) is present with radiographically visible bone loss and the disease process is continuing, the following steps should be implemented:

   A. Reflect the tissue and degranulate the defect (metallic curettes are acceptable).
   B. If the implant is hydroxyapatite (HA) coated and the HA is undergoing resorption and has changed color and texture, remove all the HA until the metallic surface is visible. Use of ultrasonics such as Cavitron (Dentsply) is best; use of hand curettes is too slow, and use of air abrasives is dangerous because of danger of air emboli in marrow spaces.
   C. Detoxify the dental implant with citric acid applied with cotton pledget or camel's hair brush. Thirty seconds per surface is sufficient. The super-saturated citric acid solution (40%, pH 1, crystals mixed with sterile water) will last in the refrigerator for about 1 year.
   D. Graft with freeze-dried bone or alloplast if completely detoxified. Graft with an alloplast such as HA or bioglass if not completely detoxified.
   E. Protect the graft with a membrane for guided bone regeneration if needed. Resorbable membranes (such as Alloderm [Lifecell, Branchburg, N.J.; distributed by BioHorizons, Birmingham, Ala.] and Biomend [Zimmer Dental, Carlsbad, Calif.]) are acceptable. If the defect cannot be closed via primary intention healing with soft tissue, use the dense 3-polytetrafluoroethylene membrane (e.g., TEF-Gen; Lifecore Biomedical, Chaska, Minn.).
   F. Leave the repaired implant out of function and "covered" for 10 to 12 weeks.
      NOTE: If the surface of the implant is metallic (titanium, Ti-6Al-4V, titanium plasma spray), go from Step A to Step C.

II. If no active infection is present and if an HA-coated implant is in place and the HA looks intact without ongoing resorption (bone loss from traumatic occlusion, overloading, off-axis loading, and so on), the following steps should be implemented:

   A. Reflect the tissue and degranulate the defect with metallic curettes.
   B. Detoxify the HA surface with citric acid (40%, pH 1) for 30 seconds per surface. Flush and irrigate with sterile water or sterile saline to stop demineralization process of the citric acid. Thirty seconds of citric acid application will detoxify and "freshen" the surface.
   C. Continue with grafting, guided bone regeneration (GBR) materials, and procedures as noted previously for treatment of the "infected" implant.
      NOTE: The only difference is that removal of the HA is not necessary because the coating is relatively noncontaminated and still capable of biological healing.
      IMPORTANT: Do not use tetracycline on intact HA because it changes the calcium/phosphate ratio of HA. Do not leave citric acid on HA surface for more than 1 minute; it continues to "remove".

Group V, the last category for implant quality, is absolute failure. It consists of implants surgically removed or exfoliated and no longer in the mouth (Figure 42-6). The remaining edentulous area often is treated with autogenous or synthetic bone graft procedures, which are performed to replace the missing bone. Once the favorable bony conditions are restored, implants may be inserted again with good prognosis.

WWW.HIGHDENT.IR

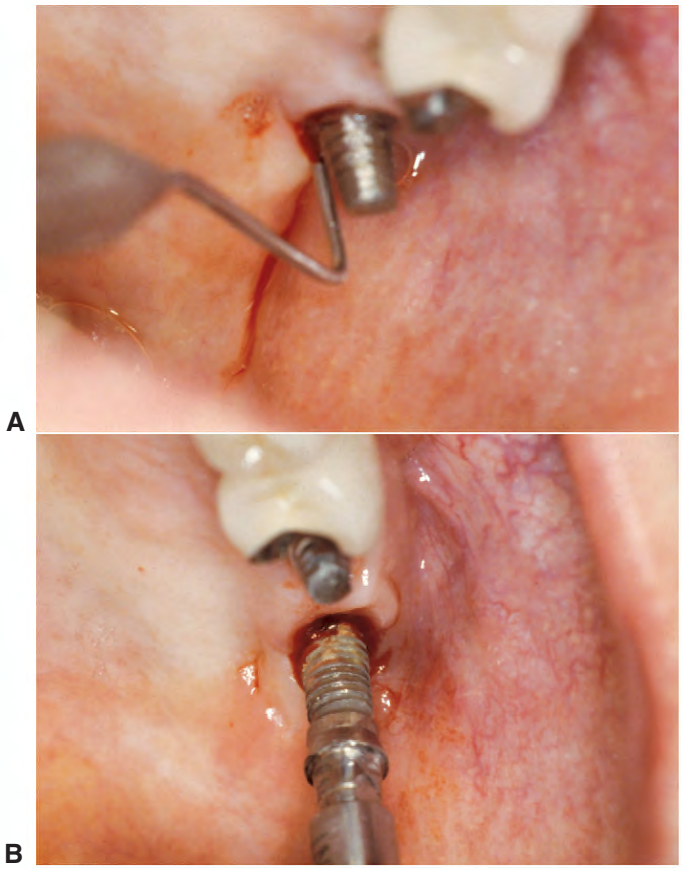


Figure 42-6 **A,** Implants in group V represent absolute failure and implants no longer in the mouth. The implant in this picture is an implant with more than 50% loss: it is in category IV. **B,** The implant should be removed when in group IV; therefore the implant is unthreaded from the site. The implant now is converted into group V.

IMPLANT FAILURE

The terminology for implant failure often is confusing, with different terms describing similar situations and, worse, vice versa. As such, Jividen and Misch[76] have suggested a terminology for implant failure using the time period of failure as a primary criterion. Many implant failures are not described ideally by the time of the complication and are not addressed in this nomenclature.

IMPLANT CROWN ESTHETIC INDEX

An implant crown esthetic index was developed by Henny et al. as an objective tool in rating esthetics of implant-supported single crowns and adjacent soft tissues.[77] The important item of esthetics is rarely included in evaluation studies. Esthetics can be rated in both a subjective and an objective manner. A subjective method is the use of questionnaires, which must be completed by the patient.

An objective method with a rating score, which has to be carried out by a professional observer, has never been described in the field of dental implants. Jemt introduced an index to assess the height of interproximal mucosa adjacent to single-implant restorations, but did not account for entire peri-implant contour and surface structure. An objective rating score, with a division in different items, provides insight into the esthetic result of a specific treatment and also facilitates analysis to improve surgical or prosthetic treatment. It is also possible to compare the esthetic result as a function of time to analyze the stability of a treatment procedure.[78]

1. Implant-supported single-tooth restoration in position 21. Labial convexity of the crown is slightly overcontoured, slight mismatch in color of the crown, slight deviation in position of the labial mucosal margin, and the labial surface of the mucosa is slightly undercontoured. The total score is 4 points on the index, which means moderate esthetics.
2 Implant-supported single-tooth restoration in position 11. The mesiodistal dimension of the crown is slightly overcontoured. The total score is 1 point on the index, which means satisfactory esthetics.
3. Implant-supported single-tooth restoration in position 21. The mesiodistal dimension of the crown is slightly overcontoured, a slight mismatch in color of the crown, slight deviation in position of the labial mucosal margin, and a gross mismatch in color and surface of the mucosa. The total score is 8 points on the index, which means poor esthetics.

The nine selected items were as follows:

Mesiodistal Dimension of the Crown. The mesiodistal dimension must be in harmony with the adjacent and contralateral tooth; a judgment can be given on a 5-point rating scale (grossly undercontoured, slightly undercontoured, no deviation, slightly overcontoured, grossly overcontoured).

Position of the Incisal Edge of the Crown. The position must be in harmony with the adjacent and contralateral tooth; a judgment can be given on a 5-point rating scale (grossly undercontoured, slightly undercontoured, no deviation, slightly overcontoured, grossly overcontoured).

Labial Convexity of the Crown. Convexity of the labial surface of the crown must be in harmony with the adjacent and contralateral tooth; a judgment can be given on a 5-point rating scale (grossly undercontoured, slightly undercontoured, no deviation, slightly overcontoured, grossly overcontoured).

Color and Translucency of the Crown. Color and translucency of the crown must be in harmony with the adjacent and contralateral tooth; a judgment can be given on a 3-point rating scale (gross mismatch, slight mismatch, no mismatch).

Surface of the Crown. Labial surface characteristics of the crown, such as roughness and ridges, must be in harmony with the adjacent and contralateral tooth; a

WWW.HIGHDENT.IR

judgment can be given on a 3-point rating scale (gross mismatch, slight mismatch, no mismatch).

Position of the Labial Margin of the Peri-Implant Mucosa. The labial margin of the peri-implant mucosa must be at the same level as the contralateral tooth and in harmony with the adjacent teeth; a judgment can be given on a 3-point rating scale (deviation of 1.5 mm or more, deviation less than 1.5 mm, no deviation).[78]

Position of Mucosa in the Approximal Embrasures. The interdental papillae must be in their natural position; a judgment can be given on a 3-point rating scale (deviation of 1.5 mm or more, deviation less than 1.5 mm, no deviation).

Contour of the Labial Surface of the Mucosa. The contour of the mucosa at the alveolar bone must be in harmony with the adjacent and contralateral tooth; a judgment can be given on a 5-point rating scale (grossly undercontoured, slightly undercontoured, no deviation, slightly overcontoured, grossly overcontoured).

Color and Surface of the Labial Mucosa Color (redness) and surface characteristics (presence of attached mucosa) must be in harmony with the adjacent and contralateral tooth and must have a natural appearance; a judgment can be given on a 3-point rating scale (gross mismatch, slight mismatch, no mismatch).

It has been decided to use the adjacent and contralateral tooth as a reference, rather than the generally accepted rules for shape and position of teeth.[78] These rules are derived from young female patients and cannot be applied to all patients, as the proportions between the general shape of the face, size, sex, and other teeth have to be maintained. Penalty points were given to each of these items if not matching to the desired situation: 1 penalty point for minor (slight) deviations and 5 penalty points for major (gross) deviations. The total score leads to a judgment about esthetics (Box 42-5).[78]

It should be noted that one major deviation automatically leads to a poor esthetic result and can never be accepted as moderate or satisfactory. The making of the implant crown is the last part of the total implant treatment. Prosthodontists are daily involved with prosthetic restorations and evaluation of the total treatment. This experience could be a reason why prosthodontists are more consistent in their scores than oral-maxillofacial surgeons.

Box 42-5 Esthetic Scale

0 points = Excellent esthetics
1 or 2 points = Satisfactory esthetics
3 or 4 points = Moderate esthetics
5 or more points = Poor esthetics

SUMMARY

Implant success is as difficult to describe as the success criteria required for a tooth. A range from health to disease exists in both conditions. The primary criteria for assessing implant quality are pain and mobility. The presence of either greatly compromises the implant; removal usually is indicated. Probing depths may be related to the presence of local disease or preexisting tissue thickness before the implant was inserted. An increasing probing depth is more diagnostic and signifies bone loss, gingival hyperplasia, or hypertrophy. Bone loss usually is evaluated best with probing rather than with radiographs. The most common cause of bone loss during the first few years of function is related to factors of stress. The bleeding index is observed easily and indicates inflammation of the gingiva. However, implant health status is not as related to sulcular inflammation as would be the case with a natural tooth.

Implant failure is easier to describe and may consist of a variety of factors. Any pain, vertical mobility, uncontrolled progressive bone loss, and generalized peri-implant radiolucency warrant implant removal. Implant quality factors were established by James and modified by Misch into an implant quality scale that not only assesses the implant health-disease continuum but also relates treatment and prognosis to the existing conditions.

References

1. Jalbout Z, Tabourian G: *Glossary of implant dentistry,* Upper Montclair, NJ, 2004, ICOI.
2. LeBeau J: Maintaining the long-term health of the dental implant and the implant-borne restoration, *Compend Cont Ed Oral Hygiene* 3:3-9.
3. Strong S, Strong S: The dental implant maintenance visit, *J Pract Hygiene* 4:29-32, 1995.
4. Koutsonikos A, Fedcrio J, Yunka R: Implant maintenance, *J Pract Hygiene* 5:11-15, 1996.
5. Terracciano-Mortilla L: Hygiene and soft tissue management. In Babbush C, editor: *Dental implants: principles and practice,* Philadelphia, 2001, Saunders.
6. Meffert RM: Maintenance of dental implants. In Misch CE, editor: *Dental implant prosthetics,* St Louis, 2005, Mosby.
7. Matarasso S, Quaremba G, Coraggio F et al: Maintenance of implants: an in vitro study of titanium implant surface modifications subsequent to the application of different prophylaxis procedures, *Clin Oral Implants Res* 7:64-72, 1996.
8. Berglundh T, Lindhe J, Ericsson I et al: The soft tissue barrier at implants and teeth, *Clin Oral Implants Res* 2:81-90, 1991.
9. Bollen CM, Papaioanno W, Van Eldere J et al: The influence of abutment surface roughness on plaque accumulation and peri-implant mucositis, *Clin Oral Implants Res* 7:201-111, 1996.
10. Quirynen M, deSote M, Steeberghe D. Infectious risks for oral implants: a review of the literature, *Clin Oral Implants Res* 13:1-19, 2002.

WWW.HIGHDENT.IR

11. Lindquist LW, Rockler B, Carlsson GE: Bone resorption around fixtures in edentulous patients treated with mandibular fixed tissue-integrated prostheses, *J Prosthet Dent* 59:59-63, 1988.
12. Lee KH, Maiden MF, Tanner AC et al: Microbiota of successful osseointegrated dental implants, *J Periodontol* 70:131-138, 1999.
13. Mombelli A, Mericske-Stern R: Microbiological features of stabile osseointegrated implants used as abutments for overdentures, *Clin Oral Implants Res* 1:1-7, 1990.
14. Mombelli A, Van Oosten MAC, Schurch E et al: The microbiota associated with successful or failing osseointegrated titanium implants, *Oral Microbiol Immunol* 2:145-151, 1987.
15. Becker W, Becker B, Newman MG et al: Clinical and microbiologic findings that may contribute to dental implant failure, *Int J Oral Maxillofac Implants* 5:31-38, 1990.
16. Haanaes HR: Implants and infection with special reference to oral bacteria, *J Clin Periodontol* 17:516-524, 1990.
17. Mombelli A, Buser D, Lang NP: Colonization of osseointegrated titanium implants in edentulous patients: early results, *Oral Microbiol Immunol* 3:113-120, 1988.
18. Mombelli A: Microbiology of the dental implant, *Adv Dent Res* 7:202-206, 1993.
19. Rams TE, Feik D, Slots J: Staphylococci in human periodontal diseases, *Oral Microbiol Immunol* 5:29-32, 1990.
20. Mombelli A, Marxer M, Gaberthuel T et al: The microbiota of osseointegrated implants in patients with a history of periodontal disease, *J Clin Periodontol* 22:124-130, 1995.
21. Leonhardt A, Adolfsson B, Lekholm U et al: A longitudinal microbiological study on osseointegrated titanium implants in partially edentulous patients, *Clin Oral Implants Res* 4:113-120, 1993.
22. Apse P, Ellen RP, Overall CM et al: Microbiota and crevicular fluid collagenase activity in the osseointegrated dental implant sulcus: a comparison of sites in edentulous and partially edentulous patients, *J Periodontal Res* 24:96-105, 1989.
23. Quirynen M, Listgarten MA: The distribution of bacterial morphotypes around natural teeth and titanium implants ad modum Brånemark, *Clin Oral Implants Res* 1:8-12, 1990.
24. George K, Zafiropoulos GG, Murat Y et al: Clinical and microbiological status of osseointegrated implants, *J Periodontol* 65:766-770, 1994.
25. Lindhe, J, Karring, T, Lang, N: *Clinical periodontology and implant dentistry,* Copenhagen, 2000, Munksgaard.
26. Silverstein L, Kurtzman D, Garnick J et al: The microbiota of the peri-implant region in health and disease, *Implant Dent* 3:170-174, 1994.
27. Haffajee AD, Smith C, Torresyap G et al: Efficacy of manual and powered toothbrushes (II). Effect on microbiological parameters, *J Periodontol* 28:947-954, 2001.
28. Chung DM, Oh TJ, Shotwell JL et al: Significance of keratinized mucosa in maintenance of dental implants with different surfaces, *J Periodontol* 77:1410-1420, 2006.
29. Wilken E: *Clinical practice of the dental hygienist,* ed 7, Philadelphia, 1994, Williams & Wilkins.
30. Humphrey S: Implant maintenance, *Dent Clin North Am* 50:463-478, 2006.
31. Terracciano-Mortilla L: Hygiene and soft tissue management. In Babbush C, editor: *Dental implants: principles and practice,* Philadelphia, 2001, Saunders.
32. Kracher CM, Smith WS: Oral health maintenance of dental implants: a literature review, *Dental Assist* 67:2-15, 1998.
33. Garber DA: Implants—the name of the game is still maintenance, *Compend Contin Educ Dent* 12:876, 878, 880, 1991.
34. Papaioannou W, Quirynen M, Van Steenberghe D: The influence of periodontitis on the subgingival flora around implants in partially edentulous patients, *Clin Oral Implants Res* 7:405-409, 1996.
35. English C: Hygiene, maintenance, and prosthodontic concerns for the infirm patient: clinical report and discussion, *Implant Dent* 4:166-172, 1995.
36. Briner WW, Grossman E, Buckner RY et al: Effect of chlorhexidine gluconate mouthrinse on plaque bacteria, *J Periodont Res* 16:44-52, 1986.
37. Brough Muzzin K, Johnson R, Carr P et al: The dental hygienist's role in the maintenance of osseointegrated dental implants, *J Dent Hyg* 62:448-453, 1988.
38. American Academy of Periodontology: *Maintenance and treatment of dental implants* [position paper], April 1995.
39. Hallmon W, Waldrop T, Meffert R et al: A comparative study of the effects of metallic, nonmetallic, and sonic instrumentation on titanium abutment surfaces, *Int J Oral Maxillofac Implants* 11:96-100, 1996.
40. Baily G, Gardner J, Day M et al: Implant surface alterations from a nonmetallic ultrasonic tip, *Periodontal Abstracts* 46:3:69-73, 1998.
41. Sato S, Kishida M, Ito K: The comparative effect of ultrasonic scalers on titanium surfaces: an in vitro study, *J Periodontol* 75:1269-1273, 2004.
42. Brookshire FV, Nagy WW, Dhuru VB et al: The qualitative effects of various types of hygiene instrumentation on commercially pure titanium and titanium alloy implant abutments: an in vitro and scanning electron microscope study, *J Prosthet Dent* 78:286-294, 1997.
43. Rapley JW, Swan RH, Hallmon WW et al: The surface characteristics produced by various oral hygiene instruments and materials on titanium implant abutments, *Int J Oral Maxillofac Implants* 5:47-52, 1990.
44. Meschenmoser A, d'Hoedt B, Meyle J et al: Effects of various hygiene procedures on the surface characteristics of titanium abutments, *J Periodontol* 67:229-235, 1996.
45. Mengel R, Buns CE, Mengel R et al: An in vitro study of the treatment of implant surfaces with different instruments, *Int J Oral Maxillofac Implants* 13:91-96, 1998.
46. Augthun M, Tinschert J, Huber A: In vitro studies on the effect of cleaning methods on different implant surfaces, *J Periodontol* 69:857-864, 1998.
47. Matarasso S, Quaremba G, Coraggio F et al: Maintenance of implants: an in vitro study of titanium implant surface modifications subsequent to the application of different prophylaxis procedures, *Clin Oral Implants Res* 7:64-72, 1996.
48. Reference deleted in page proofs.
49. Technique for implant polishing, *J Pract Hyg* March/April 35, 1997.
50. Ramaglia L, di Lauro AE, Morgese F et al: Profilometric and standard error of the mean analysis of rough implant surfaces treated with different instrumentations. *Implant Dent* 15:77-82, 2006.
51. Steele D, Orton G: Dental implants: clinical procedures and homecare considerations, *J Pract Hyg* June/July: 9-12, 1992.

WWW.HIGHDENT.IR

52. Yukna R: Optimizing clinical success with implants: maintenance and care, *Compend Contin Educ Dent* 15:554-561, 1993.
53. English C: Hygiene, maintenance, and prosthodontic concerns for the infirm patient: clinical report and discussion, *Implant Dent* 4:166-172, 1995.
54. Reference deleted in page proofs.
55. Siegrist AE, Gusberti F, Brecx M et al: Efficacy of rinsing with chlorhexidine digluconate in comparison of phenolic and plant alkaloid compounds, *J Periodont Res* 21:60-74, 1986.
56. Arweiler N, Boehnke N, Sculean A et al: Differences in efficacy of two commercial 0.2% chlorhexidine mouthrinse solutions: a 4-day plaque regrowth study, *J Clin Periodontol* 33:334-339, 2006.
57. Minichetti J, Colplanis N: Considerations in the maintenance of the dental implant patient, *J Pract Hygiene* 2:5;15-19, 1993.
58. Felo A, Shibly O, Ciancio S et al: Effects of chlorhexidine irrigation on per-implant maintenance, *Am J Dent* 10:107-110, 1997.
59. Probster L, Lin W: Effects of fluoride prophylactic agents on titanium surfaces, *Int J Oral Maxillofac Implants* 2:390-394, 1992.
60. Toumelin-Chemla F, Rouelle F: Corrosive properties of fluoride-containing odontologic gels against titanium, *J Dent* 24:109-115, 1996.
61. Renvert S, Lesse J, Dahlen G et al: Topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement of incipient peri-implant infections: a randomized clinical trial, *J Clin Periodontol* 33:362-369, 2006.
62. Salvi GE, Persson GR, Heitz-Mayfield LJA et al: Adjunctive local antibiotic therapy in the treatment of peri-implantitis II: clinical and radiographic outcomes, *Clin Oral Implants Res* 18:281-285, 2007.
63. Trejo PM, Bonaventura G, Weng D et al: Effect of mechanical and antiseptic therapy on peri-implant mucositis: an experimental study in monkeys, *Clin Oral Implants Res* 17:294-304, 2006.
64. Mombelli A, Lang NP: Antimicrobial treatment of peri-implant infections, *Clin Oral Implants Res* 3:162-168, 1992.
65. Schnitman PA, Shulman LB: Recommendations of the consensus development conference on dental implants, *J Am Dent Assoc* 98:373-377, 1979.
66. Cranin AN, Silverbrand H, Sher J et al: The requirements and clinical performance of dental implants. In Smith DC, Williams DF, editors: *Biocompatibility of dental materials*, vol 4, Boca Raton, Fla, 1982, CRC Press.
67. McKinney RV, Koth DC, Steflik DE: Clinical standards for dental implants. In Clark JW, editor: *Clinical dentistry*, Harperstown, Pa, 1984, Harper & Row.
68. Albrektsson T, Zarb GA, Worthington P et al: The long-term efficacy of currently used dental implants: a review and proposed criteria of success, *Int J Oral Maxillofac Implants* 1:1-25, 1986.
69. Smith DC, Zarb GA: Criteria for success of osseointegrated endosseous implants, *J Prosthet Dent* 62:567-572, 1989.
70. Misch CE: Implant success or failure: clinical assessment in implant dentistry. In Misch CE, editor: *Contemporary implant dentistry*, St Louis, 1993, Mosby.
71. Misch CE: The Implant Quality Scale: a clinical assessment of the health disease continuum, *Oral Health* 15:15-25, 1998.

71a. Misch CE, Wang HL, Palti A et al: The International Congress of Oral Implantologists Consensus Congress on Implant Success, Padua, Italy, 2007.

72. Rateitschak KH: Periodontology. In Rateitschak KH, Rateitschak EM, Wolf HF et al, editors: *Color atlas of dental medicine*, ed 2, New York, 1989, Thieme.
73. Council on Dental Care Programs: Reporting periodontal treatment under dental benefit plans, *J Am Dent Assoc* 17:371-373, 1988.
74. James RA: Peri-implant considerations, *Dent Clin North Am* 24:415-420, 1980.
75. Mombelli A, Lang NP: The diagnosis and treatment of peri-implantitis, *Periodontol 2000* 17:63-76, 1998.
76. Jividen G Jr, Misch CE: Reverse torque testing and early loading failures help or hindrance? *J Oral Implantol* 26:82-90, 2000.
77. Henny JA, Meijer K, Stellingsma K et al: A new index for rating aesthetics of implant-supported single crowns and adjacent soft tissues—the Implant Crown Aesthetic Index: a pilot study on validation of a new index, *Clin Oral Implants Res* 16:645–649, 2005.
78. Jemt T: Regeneration of gingival papillae after single implant treatment, *Int J Periodontics Restorative Dent* 17: 327-333, 1997.

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Appendix A

Extraoral and Intraoral Evaluation

IMPLANT EVALUATION & TREATMENT RECORD

Copyright © 2007 MIII

Printed in the United States of America and published by Nierman Practice Management. All rights reserved. No part of this book may be reproduced in any manner whatsoever without written permission of MIII.

For Information & Ordering:

Nierman Practice Management
221 Old Dixie Highway
Suite 5
Tequesta, Florida 33469

1-800-879-6468
www.dentalwriter.com
Fax: 561-744-5606

Dental Evaluation-Completely Edentulous Arches

Extraoral Evaluation

PHYSICAL CHARACTERISTICS

Cosmetic Index	Poor ❑	Average ❑	Good ❑
Countenance	Tense ❑	Average ❑	Pleasing ❑
Color and Skin Texture	❑ Normal	❑ Other	
Complexion	❑ Fair	❑ Average	❑ Dark
Patient Attitude	❑ Receptive ❑ Exacting ❑ Indifferent		
Neuromuscular Coordination	Poor ❑	Average ❑	Good ❑

SEX ❑ Male ❑ Female
AGE ❑ Young ❑ Middle ❑ Mature
ESTHETIC/AGE POTENTIAL Low ❑ Average ❑ High ❑

PERSONALITY Delicate ❑ Average ❑ Vigorous

LIPS Short ❑ Long ❑

EYES ❑ Brown ❑ Hazel ❑ Blue ❑ Green ❑ Gray

HAIR ❑ Brunette ❑ Brown ❑ Blonde ❑ Red ❑ Gray

FACE ❑ Square ❑ Tapering ❑ Ovoid

PROFILE ❑ Normal ❑ Retrognathic ❑ Straight ❑ Prognathic

PRESENT DENTURE EVALUATION

Type of Denture ❑ Maxillary ❑ Mandibular

Denture History Previous Dentures	Type	Quantity

Denture Experience ❑ Favorable ❑ Unfavorable

❑ Masticatory efficiency	❑ Poor	❑ Average	❑ Good
❑ Stability and retention	❑ Poor	❑ Average	❑ Good
❑ Esthetics	❑ Poor	❑ Average	❑ Good
❑ Phonetics	❑ Poor	❑ Average	❑ Good
❑ Comfort	❑ Poor	❑ Average	❑ Good
❑ Occlusal vertical dim.	❑ Poor	❑ Average	❑ Good

Intraoral Evaluation Completely Edentulous

SIZE OF JAWS	Small	Normal	Large
Maxilla	❑	❑	❑
Mandible	❑	❑	❑

ARCH FORM
Maxilla ❑ Square ❑ Ovoid ❑ Tapered
Mandible ❑ Square ❑ Ovoid ❑ Tapered

RESIDUAL RIDGE
Maxilla ❑ Square ❑ Ovoid ❑ Tapering ❑ Flat
Mandible ❑ Inverted "U" Shape ❑ Parallel Wall ❑ Flat ❑ Inverted "U" Shape ❑ "U" Shape ❑ Thin ❑ Parallel Walled Undercut

RIDGE PARALLELISM
Both ridges parallel ❑
Maxilla divergent anteriorly ❑
Mandible divergent anteriorly ❑
Both divergent anteriorly ❑

RIDGE RELATION ❑ Normal ❑ Prognathic ❑ Retrognathic

INTERARCH DISTANCE Insufficient ❑ Sufficient ❑

SOFT PALATE ❑ Class I ❑ Class II ❑ Class III ❑ Class IV ❑ Sufficient ❑ Excessive

SOFT TISSUES ❑ Thin ❑ Thick ❑ Healthy ❑ Irritated ❑ Pathologic

MUSCLE ATTACHMENTS	Low	Average	High
	❑	❑	❑

PALATAL THROAT FORM	Flat	Normal	Sharp
	❑	❑	❑

TONGUE POSITION	Subnormal	Normal
	❑	❑

LIP CONTOUR	Needs Support	Normal
Maxilla	❑	❑
Mandible	❑	❑

MAND/MOVEMENTS	Limited	Unlimited
Protrusive	❑	❑
Right Lateral	❑	❑
Left Lateral	❑	❑

Opening_____mm

MUSCLE TONE	Poor	Average	Good
	❑	❑	❑

SALIVA
Amount ❑ Insufficient ❑ Average ❑ Excessive
Character ❑ Normal ❑ Thin ❑ Thick ❑ Ropy

SAMPLE

Appendix Figure A-1 The data obtained during the extraoral and intraoral examination of partially edentulous and completely edentulous patients is recorded in the patient evaluation forms.

WWW.HIGHDENT.IR
مرجع دندانسازان و دندانپزشکان

Dental Evaluation

Initial Soft Tissue Exam

LIPS	FLOOR OF MOUTH	PALATE	TONGUE	NECK & NODES
❑ WNL ❑ +				

Available Bone Classification

COMPLETELY EDENTULOUS ARCHES	TYPE I	TYPE II	TYPE III	
PARTIALLY EDENTULOUS ARCHES	CLASS I	CLASS II	CLASS III	CLASS IV
BONE DIVISION	DIVISION A	DIVISION B	DIVISION C	DIVISION D
BONE CHARACTER	DENSITY 1	DENSITY 2	DENSITY 3	DENSITY 4

Partially Endentulous

Implant Site

BONE CHARACTER	❑ Favorable Normal Trabeculae	❑ Marginal Recent Extraction	❑ Unfavorable Pathology
BONE TRAJECTORY	❑ Favorable Vertical	❑ Adequate Slight Flare	❑ Unfavorable Gross Flare
ATTACHED GINGIVA	❑ Favorable 3mm	❑ Adequate 1-2mm	❑ Unfavorable None
MUSCLE ATTACHMENT	❑ None	❑ Moderate	❑ Severe
INTERARCH SPACE	❑ Favorable	❑ Adequate	❑ Unfavorable
PERMUCOSAL POSITION	❑ Favorable	❑ Adequate	❑ Unfavorable
TISSUE SUPPORT AREA	❑ Favorable	❑ Adequate	❑ Unfavorable

Initial Perio Exam

GINGIVAL INFLAMMATION	❑ Slight	❑ Moderate	❑ Severe
SOFT PLAQUE BUILD-UP	❑ Slight	❑ Moderate	❑ Severe
HARD CALCULUS BUILD-UP	❑ Slight	❑ Moderate	❑ Severe
STAINS	❑ Light	❑ Moderate	❑ Severe
PERIODONTAL CONDITION	❑ Good	❑ Fair	❑ Poor
PERIODONTAL DIAGNOSIS	❑ Normal	❑ Gingivitis	❑ Periodontitis
PERIODONTITIS	❑ Early	❑ Moderate	❑ Advanced

General Dental Considerations

GENERAL HOME CARE	❑ Favorable	❑ Adequate	❑ Unfavorable
DENTAL I.Q.	❑ High	❑ Normal	❑ Unfavorable
DENTAL MUSCLE DYNAMICS	❑ Weak	❑ Normal	❑ Strong
GENERAL TMJ STATUS	❑ Symptom Free	❑ Incipient	❑ Dysfunction
OCCLUSAL PLANE	❑ Favorable	❑ Adequate	❑ Unfavorable
ARCH RELATIONSHIP	❑ Favorable	❑ Adequate	❑ Unfavorable
OPPOSING DENTITION	❑ Removeable	❑ Combination	❑ Fixed
EXISTING OCCLUSION	❑ Favorable	❑ Adequate	❑ Unfavorable
EXISTING PROSTHESIS	❑ Favorable	❑ Adequate	❑ Unfavorable
PONTICS	❑ 1	❑ 2	❑ 3 or more
TISSUE SUPPORT AREAS	❑ Normal	❑ Average	❑ Excessive
RESTING LIP LINE	❑ Low	❑ Normal	❑ High
HIGH LIP LINE (SMILE)	❑ Low	❑ Normal	❑ High
LOW LIP LINE (SPEECH)	❑ High	❑ Normal	❑ Low
ARCH POSITION	❑ Class I	❑ Class II	❑ Class III
ARCH FORM (Ant.-Post)	❑ Tapering	❑ Ovoid	❑ Square

Stresses

PARAFUNCTION			
BRUXISM	❑ None	❑ Occasional	❑ Frequent
CLENCHING	❑ None	❑ Occasional	❑ Frequent
TONGUE THRUST	❑ None	❑ Occasional	❑ Frequent
ORAL HABITS	❑ None	❑ Occasional	❑ Frequent
MASTICATORY DYNAMICS	❑ Light	❑ Normal	❑ Heavy
CROWN HEIGHT SPACE	❑ <1	❑ 1/1 TO 2/1	❑ >2/1
DENSITY OF BONE	❑ D1,2	❑ D3	❑ D4
DIRECTION OF FORCE	❑ Vertical	❑ <15 Degrees	❑ >15 Degrees
TONGUE SIZE	❑ Small	❑ Normal	❑ Large
MISSING TEETH _____			

SAMPLE

WWW.HIGHDENT.IR

Dental Evaluation

Subjective Considerations

ORAL COMFORT POTENTIAL	❑ Excellent	❑ Normal	❑ Guarded
ORAL FUNCTION POTENTIAL	❑ Excellent	❑ Normal	❑ Guarded
ORAL ESTHETIC POTENTIAL	❑ Excellent	❑ Normal	❑ Guarded
PSYCHOLOGICAL STATUS	❑ Positive	❑ Normal	❑ Negative
PATIENT EXPECTATIONS	❑ High	❑ Normal	❑ Low
PATIENT ECONOMIC PRIORITIES	❑ High	❑ Normal	❑ Low

Natural Abutment (Optional: Repeat For Each Quadrant)

MOBILITY	❑ Minimal	❑ Moderate	❑ Severe
ENDODONTIC STATUS	❑ Favorable	❑ Potential	❑ Pathology
ATTACHED GINGIVA	❑ 3mm	❑ 1-2mm	❑ None
MUSCLE ATTACHMENT	❑ None	❑ Moderate	❑ Severe
ROOT CONFIGURATION	❑ Divergent	❑ Normal	❑ Tapered
TOOTH POSITION	❑ Favorable	❑ Exfoliation	❑ Angulation
PARALLELISM	❑ Favorable	❑ Guarded	❑ Unfavorable
CARIES	❑ None	❑ Moderate	❑ Severe

Medical Evaluation

RESULTS	❑ POSITIVE	❑ NORMAL	❑ NEGATIVE
VITAL SIGNS	mm Hg	BPM	
URINALYSIS			
CBC			
SMA 12			
PT PTT INR			
OTHER			

Patient Habits

TOBACCO HABITS	❑ SMOKING	❑ ORAL	❑ None	❑ Moderate	❑ Heavy
ALCOHOL CONSUMPTION			❑ None	❑ Moderate	❑ Heavy

Photographs

FULL FACE	❑ Before	❑ After	INTRAORAL	❑ Before	❑ After
PROSTHETIC	❑ Before	❑ After	SURGERY	❑ Before	❑ After

Diagnostic Records

- ❑ PANORAMIC X-RAY
- ❑ PERIAPICALS
- ❑ FULL MOUTH SERIES
- ❑ LATERAL SKULL, CEPHALOMETRIC VIEW
- ❑ CT SCAN
- ❑ TRANSCRANIAL
- ❑ MRI
- ❑ STUDY MODELS
- ❑ OTHER

SAMPLE

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Index

A
Abfraction, 108f, 113-114f
Abscess, acute/chronic
  of brain, 508
  circumscribed mandibular, 506
  definition of, 503
  implant success and, 1068-1069
Abutments, implant. *See also* Key implant positions; Number, implant.
  angled, 746f
  attaching, 28-29
  categories of, 31-33, 31-35f
  connection area of, 30
  fracture of, 71-72, 71f
  imaging to verify, 59
  loosening of, 70-71, 70f
  natural teeth vs.
    evaluating, 255b, 256-267
    for implant prosthesis, 20, 255-256
    for partial dentures, 6, 12, 16-17
  permucosal, selection of, 731-733
  purpose of, 1055
Acetaminophen, 477-478, 478t, 480t
Acetylsalicylic acid (ASA). *See* Aspirin.
Actinobacillus actinomycetemcomitans, 1074-1075
Adapted zone, 808
Adhesive, denture, 17, 21
Adjuvant therapy. *See* Analgesics.
Adrenal gland disorders, 445-446, 446t
Adrenal suppression, 475
Age
  bite force by, 544t
  limitations for implants, 346-347
Aging population
  dental implants and, 3-5, 423
  edentulism and, 5-9
  increase of, 4f
  periosteal apposition in, 574f
  physiologic changes in, 421-423, 500-501
Akinosi technique, 685-686, 685f
Alabama, University of, 319-320, 320f
Alcohol use, 459-460
Alkaline phosphatase, serum, 433
Allergic sinusitis, 921
Allografts, 859t, 888, 975-976
Alloplasts, 861t, 878
Aluminum oxide in biomaterials, 518-519
Alveolar bone
  dental implants and, 17, 18f
  edentulous effects on, 9-11, 10f
  facial changes and loss of, 13
  imaging of changes in, 60, 61f
  resorption of, 839
Alveolar nerves, 492f, 493, 497
American Academy of Implant Dentistry, 28
American Academy of Oral and Maxillofacial Radiology, 39
American Academy of Periodontology, 1078
American Dental Association
  on antibiotic regimens, 453t
  on implant success criteria, 1055
American Heart Association (AHA), 438t, 441b
Amoxicillin, 470
  dosage information for, 473t
  for prophylaxis, 472
  protocol for prescribing, 485t
Amoxicillin/clavulanic acid, 470
  dosage information for, 473t
  mechanism of action of, 471f
  for prophylaxis, 472
  protocol for prescribing, 485t
  for sinus infection, 965
Analgesia, preemptive, 476
Analgesic therapy, combination, 480-482, 480t, 481b
Analgesics
  adjuvant, 480t, 481b
  dental classifications of, 477b
  nonopioid, 477-479, 477f, 478t
  opioid, 478t, 479-480
  protocol ladders for, 481b, 481t
  for sinus graft surgery, 929
  for specific procedures, 485t
Analog, implant, 31f, 33, 34f, 35f
Anastamosis, 911b, 911f
Anchorage control, 588-595
Anemia, 447
Anesthesia block
  Akinosi technique for, 685-686b, 685f
  V2, for sinus graft, 940f
Anesthetics, local, 481-483. *See also* Sedative agents.
  dosage information for, 481-482t
  for surgical harvest, 985-986
  toxicity of, 483b
Angina pectoris, 437-438, 438t
Angiogenic stage, initial, 875
Angulation
  bone, 183-184, 184f
  implant, 745-750, 745f, 747f
Anisotropy, 204-205, 629
Ankylosis, implant, 27
Anodontia, 351-354
Anteroposterior distance (A-P spread)
  cantilevers and, 149f, 553
  for fixed prostheses implants, 318f
  for immediate loading, 818
  implant position/number and, 154-155, 244
Antibiotics, 467-474
  for bone graft infection, 840
  colitis induced by, 470b
Antibiotics—cont'd
  collagen membrane with, 946
  for postoperative infection, 472-474
  prophylactic
    for cardiovascular conditions, 438t
    for endocarditis, 441b, 442t
    for oral implantology, 467-470, 472
    for prosthetic joints, 453t
    for sinus graft surgery, 926-928, 927b
  for sinus infection, 965
  for specific procedures, 485t
  used in dentistry, 470-472
Anticoagulant medications, 457-458
Antiestrogen therapy, 581
Antiinflammatories, 474-476, 475t, 485t
Antimicrobials, oral, 1074-1075. *See also* Antibiotics.
Antirotational movement, 70
Antisepsis, 929
Antral fistula, oral, 967, 968f
Antral septa, 959-960, 960-961f
Antroliths, 924, 925f
Anxiolytics, sedative, 486
Apex of implant body, 29f, 30, 220-221
Apicoectomy, 251
Applegate rules, 406
Arc of closure, 369f
Arches, dental. *See also* Mandible; Maxilla.
  classification of edentulous
    Kennedy, 406-413
    Misch-Judy, 413-417
  distance between, 245
  force distribution of, 131f
  form in, evaluating, 253-254, 254f
  free-end edentulism of, 8
  implants to maintain, 18
  molars to maintain, 5
  position of, and opposing, 125-126, 126f, 813
  relationship between, 243-244
  segments of, 155f, 373f, 414f
  treatment plans for
    completely edentulous, 379-387, 380f, 413-417, 414-417f
    partially edentulous, 406, 407-408f, 409-413
Area, functional cross-sectional, 544f, 546-547
Arterial arcade, double, 910-911, 911b
Arterial supply of face, 494f
Arthritis, rheumatoid, 454
Articaine, 481-482t, 482
Asepsis, 840, 926
Aspirin, 458, 479
Augmentation, bone. *See* Available bone; Bone grafts/grafting.
Augmentin. *See* Amoxicillin/clavulanic acid.
Autogenous graft. *See* Bone grafts/grafting.
Autoimmune diseases, systemic, 454-455

Page numbers followed by *b* indicate boxes; *f*, figures; *t*, tables.

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Autoimmune response, 74f
Autoradiography, 568
Available bone
  bone dimensions and, 180f, 182-184f, 184b
  crown height space and, 120f, 184
  divisions of, 180f, 980f
    abundant (A), 184-186, 185-186f, 186b
    barely sufficient (B), 186-190, 187-188b, 187-189f
    compromised (C), 190-194, 190-191b, 190-193f
    deficient (D), 194-197, 194-196f, 195b
  for fixed prostheses appearance, 95, 96f
  immediate loading and, 813-814
  implant dimensions and, 180-181
  preimplant evaluation of, 256-257, 258f
  for single tooth replacement, 349, 740-741
  in stress theorem, 87
  for tissue support, 733
  treatment planning and, 178, 197-198
Axial loads, 630
Axonotmesis, 712b
Azithromycin, 473t, 965

B

Backscatter imaging, 567-568, 567f
Bacterial flora
  around implants/teeth, 733, 1068b, 1068t
  in maxillary sinus, 913
Bacterial plaque complications
  crestal bone loss and, 722
  early loading failure vs., 70
  implant design and, 200
  implant maintenance and, 1074-1075, 1074b
  implant success and, 1068
  marginal bone loss and, 74
Bacteroides forsythus, 1074
Barbiturates, 484t
"Barrier by bulk" technique, 845-846, 845f
Barrier membrane (BM)
  biodegradation times of, 888f
  guided bone regeneration and, 890-894, 891-893f
  in socket grafting, 878, 882-888
Basal bone
  edentulous effects on, 9, 11
  loss of, 10f, 12-13f, 15f
Benzodiazepines, 483, 484t, 486
Big-nose variant, 919
Biingner, band of, 501
Bilirubin level, 433
Biochemical profiles, 431-434
Biodegradation of implant material, 513-515, 885, 888f
Biofilm, plaque, 1074-1075, 1074b
Bioglass ($CaPO_4$), 520t, 522-524
BioHorizons Implant Systems, 332
Bioinert ceramics, 861t, 863
Biological complications, 68, 69b
Biological response to loads
  gene expression in, 627-629
  indicators of, 624-627
  mechanotransduction in, 621-624
Biomaterials. *See also* Coatings, biomaterial; Design, implant/prosthesis; Materials; *specific material*.
Biomaterials—cont'd
  biocompatibility of
    bulk properties/force in, 512-513
    vs. force load capability, 222
    future of, 525-526
    history/research of, 511-512
    surface properties for, 526, 533-535
  comparison of, 100t, 383f
  corrosion/biodegradation of, 513-515
  for FP-1 prosthesis, 95
  for FP-2 prosthesis, 96-97
  for FP-3 prosthesis, 98-99, 99f, 100f
  fracture and, 221-223
  mechanical properties of, 546-547t
  for single-tooth replacement, 335
  standardization of, 513, 515
  toxicity of, 515
Biomechanics
  of bone response
    factors influencing, 632-634
    to loading, 623f, 629-632
    mechanical loading and, 575t, 582, 584
  complications due to
    force delivery and, 549-555
    treatment theorem based on, 68, 69b, 87-88
  of extraction forceps, 871-874
  fundamentals of, 543-549
Bisphosphonates, 454-455b, 455-457
Bite force, normal. *See also* Occlusal force.
  arch position and, 125f
  changes in, 124
  for different teeth, 544f
  increasing range of, 107
  vs. masticatory forces, 105-106, 105b
Bitewing radiographs, 61
"Black particles," 516
"Black spaces," 95
Bleeding
  elderly patient and, 422
  index of, 1067-1068
  life-threatening, 713-715
  probing to measure, 1063f
  sinus graft surgery and, 960-962
  supplements associated with, 458b
  tests for, 429-431
Blood clot formation
  platelet-rich plasma and, 851f
  prothrombin time and, 430-431, 430t
Blood glucose levels, 433t
Blood pressure evaluation
  classification of, 435t
  implant therapy and, 437t
  in medical evaluation, 421, 426
Blood supply/circulation. *See* Vascular supply/circulation.
Blood tests/plasma levels. *See also* Disease, systemic; Laboratory evaluation.
  dental implants and, 20
  dentures and, 16
  indicating disease, 432t
  in patient evaluation, 428-429
Body, implant. *See also* Design, implant/prosthesis; Root form implant.
  attachments to, 31-33
  categories of, 28-29, 28f
  cleanliness of, 533-535
  geometry vs. occlusal load of, 207-208
Body, implant—cont'd
  regions of, 29-30
  for single-tooth replacement. *See* Single-tooth implant.
  two-stage, 30-31
Bone. *See also* Skeletal system; *specific bone*.
  anatomy of
    evaluating adjacent, 256-257
    surgical, 490-491, 490f
  availability of. *See* Available bone.
  defects of, 724-728, 853-854, 854f
  mechanics/physiology of. *See also* Bone modeling/remodeling.
    adaptability of, 131
    immediate loading and, 813-814
    marginal bone loss and, 77-78, 78f, 82-83, 82f
    metabolism in, 576-577, 577f
    strength under compression in, 161f
  morphology (osteology) of, 557f, 558-562
  resorption of. *See* Bone loss/resorption.
  response of, to loads
    assessment methods of, 562-568
    biological, 621-629, 626f
    biomechanical, 575t, 582, 584, 629-634
  tissue
    classification of, 568-570, 570f
    structural integrity of, 579f
Bone angulation, 183-184, 184f
Bone augmentation. *See* Available bone; Bone grafts/grafting.
Bone cell function, 562-568
Bone density. *See also* Cortical bone; Elasticity (E), modulus of; Lamellar bone; Trabecular bone.
  bone strength and, 138f, 139f
  classifications of
    literature review of, 645-646
    surgery/healing and, 646-647
    treatment planning and, 134-135, 134-136f
  considerations for maxillary, 390, 390f
  etiology of variable, 131-134
  imaging to evaluate, 48b, 60, 61f, 137-138
  implant failure and, 70
  implant size and, 166-168, 167f, 169f
  implant success and, 18, 130-131, 131f
  location and, 136-137
  marginal bone loss and, 77-78, 78f
  response to stress and, 633-634, 634f
  stimulation to maintain, 9
  stress vs. stimulation of, 82-83, 82f, 629-633
  tactile sense and, 138
  tooth loss and, 10
  treatment planning and, 85, 138-144
Bone diseases, 449-454, 582
Bone Gla protein, 625, 628
Bone grafts/grafting. *See also* Available bone; Elevation; Iliac crest block bone graft; Mandibular ramus; Mandibular symphysis; Sinus graft surgery; Socket grafting; Tibia bone graft.
  autogenous/autologous
    bone for, 857-858, 857f, 858b, 863
    de-/mineralized bone for, 858-860, 859t
    from extraoral origin, 1013-1015
    as gold standard, 976

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Bone grafts/grafting—cont'd
autogenous/autologous—cont'd
imaging for, 57
for single-tooth replacement, 744f
comparing donor sites for, 977, 1005t
complications of
from extraoral origin, 1034-1035, 1046-1047
from mandibular origin, 1005-1007, 1006-1007f
evaluation for
of extraoral origin, 1013f, 1014-1015
of mandibular origin, 977, 978-979f, 980, 980f
graft fixation after, 846, 998-1005, 1003-1004f
heterologous (xenograft), 861-862
immediate loading and, 813-814, 814f
instructions after harvesting, 1005
materials for, 855-863, 855b
maxillary implants after, 19f
preparation of host site for, 981-982, 982-984f, 985
rationale for types of, 975f
resorption of materials for, 840f
surgical keys to, 839-855
Bone healing
and bone density, 655, 658, 662, 665
and immediate loading, 800-801, 801f
Bone loss/resorption. *See also* Marginal bone loss.
in bruxism, 113
classification of, 10f, 178-180
compromising premaxilla, 368-369
dental implants and, 18, 20, 1069f
design and, 201t
edentulism and, 9-11, 10-11f, 11b
facial changes due to, 13-15, 14-15f, 15b
implant design and, 83-87, 83f
over time, 190f
peri-implant, 1075
in posterior maxilla, 390f
removable prostheses and, 12f, 13f, 17, 20
after single-tooth implant, 350f, 742f
at stage II recovery
causes for, 725b
range of, 725f
resolution of, 726t
thread design and, 220
treatment planning and, 84-87
Bone modeling/remodeling
control factors for, 575b
cutting/filling cones in, 571f, 573, 575, 588f
dynamics of, 558f
etiology of, 131-134
implant design and, 208-209, 215-217
mechanotransduction in, 622
occlusal stress and, 76-77
physiology of, 570-571, 571-574f
after surgery, 801-806, 805f
Bone morphogenetic proteins (BMPs), 852, 876
Bone regeneration, stages of, 875, 876f
Bone remodeling rate (BRR), 208-209, 215-216
Bone spreading
of Division A premaxilla, 779-787, 783-787f
Bone spreading—cont'd
of Division B premaxilla, 787-789, 788-790f
Bone turnover. *See* Bone modeling/remodeling.
Bone volume atrophy, 178-180. *See also* Bone loss/resorption.
Bone-implant contact (BIC). *See also* Surface-tissue interface.
body design and, 203-204f
bone density and, 140-141f
phases of, 599-601, 600t
posterior maxilla and, 393f
strength of, after surgery, 805f
thread geometry for, 209-215
in uncovery procedure, 724
Bosker endosteal staple, 518
Brain abscess, 508
Brånemark, Per-Ingvar
in implant history, 27
implants vs. ITI implants, 202f
mandibular approach by, 318-319
two-stage approach by, 69, 686
Brevital, 484t
Brushing. *See* Hygiene, oral.
Bruxism. *See also* Clenching.
diagnosis of, 107-109, 108f
force of
fatigue fractures due to, 110-111, 111f
occlusal guards for, 110-111
treatment planning and, 85
removable prostheses and, 102
torsion due to, 316-317
treatment planning for, 109-112
Buccal nerve, 492f, 498
Buccal space, 504
Buccinator muscle
anatomy of, 491-492, 492f, 497
denture effects on, 18-19
edentulous effects on, 11f, 15
Bundle bone, 570
Bupivacaine, 481t, 482, 482t

C

Calcitonin, 577
Calcium
calcium conservation and, 579-581
conservation of, 577, 579-581
diet and, 580t
homeostasis of, 576-577, 578f
metabolism of, 577f
Calcium, serum, 432-433
Calcium carbonate, 861t
Calcium phosphate ceramics (CPCs)
advantages of, 519-520, 519b
as implant coating, 615, 812
names/formulas/atomic ratios for, 521t
physical/mechanical properties of, 520-524
uses for, 519
in xenografting, 862
Calcium phosphate minerals, 860-863, 861t
Calcium sulfate polymer, 861t
Calculus removal, 1076
Camper's plane, 239f
Cancellous compaction, 570
Canine fossa, 504
Canine teeth
bite force of, 544t
Canine teeth—cont'd
implant positions for, 151-153
missing anterior maxillary, 370-372
Caninus muscle, 492
Cantilevers
crown height space and, 118-119, 119f
fixed partial denture as, 343
force delivery of, 551, 553-554, 553f
hidden, in overdenture, 309
immediate loading and, 813
key implant positions and, 85, 147-154, 148-149f
loosening screws and, 70
occlusal guard for, 111
preimplant evaluation for, 256-257
removable prostheses and, 103f
Carbon/carbon silicon compounds, 524
force loads and, 222
physical/mechanical properties of, 520t, 524
surface-tissue interface of, 529-530
Cardiovascular disease
antibiotic regimens for, 438t
dental disease and, 16
implant therapy and, 435-442
Cardiovascular medications, 422
Caries
and abutment prognosis, 255-256, 266
and tooth prognosis, 250-251
Carotid artery, 494f
Carotid sheath, 508
Casts, diagnostic, 276-278
Cavernous sinus thrombosis, 508
Cefadroxil, 471, 473t
Cefazolin, 841f
Cefpodoxime proxetil, 965
Cefuroxime axetil, 965
Cellular biomechanics, 76-77
Cellulitis, acute/chronic, 503
Cement, 72
Cement-enamel junction (CEJ)
esthetics and, 740
implant size and, 170-171, 360f, 362f
for soft tissue health, 751f, 765
Cement-osseous dysplasia, 452
Centric occlusion, 244
Centric relation (CR), 244-245
Centripetal circulation, 499
Cephalexin, 471, 472, 473t
Cephalometric radiography
alternate technique for, 44f
image from, 43f
for presurgical imaging, 43-44
summary of, 44b
Cephalosporin, 470b
for bone graft infection, 840
for sinus infection, 965
Ceramic materials
force loads and, 222-223
physical/mechanical properties of, 520t
based on calcium phosphates, 519-524, 519b
from metal oxides, 518-519, 518t
surface-tissue interface of, 529-530
Cervical abfraction, 113
Cervical collar, 30
Cervical erosion, 113
Cervical lymph nodes, 509

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Chemotherapy, 460
Chewing. *See* Mastication process.
Children, guidelines for implants in, 346-347, 346f
Chin, 14f, 15
Chinese, 26
Chlorhexidine gluconate
  in implant maintenance, 1076-1077
  implant therapy and, 473-474
  protocol for prescribing, 485t
Christensen's phenomenon, 238
Chromium in biomaterials, 517-518
Chronic illness. *See* Disease, systemic.
Chronic obstructive pulmonary disease (COPD), 448-449, 448t
Cingulum of crown, 745f, 746-747, 747f
Cirrhosis, 449
Clarithromycin, 473t, 965
Clean, defined
  for bone grafting, 840
  for implant body surface, 533-535
  for implant insertion, 686-687
  for sinus graft surgery, 926
Cleaning. *See* Hygiene, oral.
Clenching
  diagnosis of, 112-113, 115f
  fatigue fractures due to, 113-115, 113f
  prosthetic considerations for, 115-116
  torsion due to, 316-317
  treatment planning with force of, 85
Clindamycin
  for bone graft infection, 840
  dosage information for, 473t
  in implant dentistry, 471
  for prophylaxis, 472
  pseudomembranous colitis and, 470b
  for sinus infection, 965
Coatings, biomaterial. *See also* Surface, implant.
  for biocompatibility, 530-533
  for bone-implant contact
    plasma-sprayed, 605-606
    pros/cons of, 612-615
  and force loads, 201f, 203-206, 205f, 220, 222
  immediate loading and, 812
Cobalt-based alloys, 517, 529
Coccoids, 1068t, 1075
Codeine
  as adjuvant, 480t
  for pain management, 478t, 479
  for sinus graft surgery, 929
Cold dressings, 476
Colitis, pseudomembranous, 470b
Collagen, 855-857, 946
Columella-philtrum angle, 15f
Compliance, patient, 341-342, 423, 1073
Complications, dental, 502. *See also* Failure/complications, implant; Infection.
Composite beam analysis, 77
Composite bone, 570
Computed tomography (CT)
  for bone density evaluation, 137b
  and CAD for surgical guides, 284f, 285-288
  to evaluate donor site, 980f
  to identify vital structures, 57f, 58f
  of maxillary sinus, 917, 918f
  postprosthetic, 62, 63f
Computed tomography (CT)—cont'd
  preprosthetic, 62-63
  presurgical, 47-51
    bone/tissue quality in, 48b
    images from, 49f, 52f
    interactive, 52-54, 53-55f
    magnetic resonance, 54-56, 56b
    overview of, 50b
    scanner types for, 49-51, 51f, 51t
Computer-assisted design/manufacturing (CAD/CAM), 284f, 285-288, 287f
Concha bullosa, 918-919
Condylar disc assembly, 109
Cone, cutting/filling, 571f, 573, 575, 588f. *See also* Bone modeling/remodeling.
Cone beam volumetric tomography (CBVT), 50-51, 51-52f
Congenitally missing teeth, 351-354
Congestive heart failure (CHF), 439-440
Connectors, nonrigid, 262
Contact angles, 534
Contaminates, 533-534
Contamination
  of bone graft, 840
  risk of implant, 686-687
  of sinus graft, 913, 929
Contours of restoration, 95f, 103f
Copings, transfer. *See* Transfer copings.
Corrosion of implant material, 513-515, 514f
Cortical bone
  dense (D1), 647-655
  dense-to-thick porous (D2), 655-658
  force applied to, 545t
  grafts from, 976-977, 1015-1019
  growth/maturation of, 571-576, 571-574f
  load angle on, 162t
  modeling of, 573f
  morphology (osteology) of, 557f
  remodeling of, 558f
  structural fraction of, 575-576, 576f
  thin porous (D3), 658-662
Cortical plates, 633, 863
Cortisone, 475t
Costs. *See also* Economic considerations.
  lifetime, of crown, 5
  of precious metal in restorations, 99
  of single-tooth replacement, 342
Coumadin. *See* Warfarin sodium.
COX-2 inhibitors, 479
Creatinine level, 433
Creep
  clenching and, 113-114
  curve of, for bone, 114f, 631f
  tooth extraction and, 874
Creeping substitution, 860, 862
Crest module of implant body, 29f, 30
  design considerations for, 217-220
  for single tooth implant, 360-362
Crestal bone loss. *See also* Marginal bone loss.
  implant design and, 361f
  implant success and, 1059-1060
  prosthetic options for, 95
  in stress theorem, 72-73, 74-75f, 83-84f
Crown height space (CHS)
  available bone and, 184
  excessive
    consequences of, 118-120, 119-120f, 245-246, 246f
Crown height space (CHS)—cont'd
  excessive—cont'd
    evaluation of, 246-248, 246-247f, 246b
    treatment planning and, 120-124, 120b, 120f, 122-124f
  measurements of, 118f, 245f
  occlusal vertical dimension and, 234
  principles related to, 118b
  prosthesis design and, 85, 98-101, 382-383
  reduced, 248-250, 248b, 249f
  single-tooth implants and, 348
Crown/root ratio, 265
Crowns
  implant
    contours of, in design, 95f
    emergence profile of, 171f
    esthetic index of, 1082-1083
    lengthening of, for bruxism, 109
  longevity/survival of, 5-6
    adjacent crowns and, 7f
    fixed partial denture and, 6f
Crown-to-crest interface, 553t
Cryotherapy, 476, 929
Cylinder root form implant, 29-30
Cysts, maxillary, 922-923, 967-968
Cytokines, 77

D

Damage, implant, 1073
Death. *See* Mortality.
Decadron. *See* Dexamethasone.
Decongestants, 928, 965-966
Deformation, 547-548, 548f
Demineralized freeze-dried bone (DFDB)
  bone morphogenetic proteins and, 852
  osteoinduction and, 858-860, 859t, 860f
Dental treatment classification, 435t
DentaScan (generic) imaging, 49
Dentistry, implant
  biological vs. mechanical in, 68-69
  goals of, 92, 93f, 102-104
  prosthetic options for, 94t
Denture, complete, removable soft tissue–borne
  bone/tissue changes and, 10-13
  vs. implant supported, 5-7
  need for periodic evaluation of, 10, 15
  performance changes in, 15-16, 16b
  retention of. *See* Retention.
Denture, partial
  fixed (FPD)
    consequences of, 5-6, 6b, 6f
    pros/cons for, 330-331, 341-343
    resin-bonded, 328-329, 328b
  removable (RPD)
    bone loss and, 10-12, 13f
    consequences of, 16-17
    over healing implant, 115f
    for partial edentulism, 8
    performance changes in, 16b
    pros/cons for, 328b, 343-344
    resin-bonded, 344-345, 345b
Design, implant/prosthesis. *See also* Biomaterials; Body, implant; Size, implant; Surface, implant; Surface area, implant.
  bioengineering of, 215-221
  bone-implant contact and, 599-601

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Design, implant/prosthesis—cont'd
formula for assembly of, 171, 172f
fracture and, 223-225
immediate loading and, 810-812
occlusal forces and, 202-208, 223
rationale for, 200-202, 208-209, 225-226
in stress theorem, 80-81, 83-87
thread geometry in, 205f, 209-215
treatment planning and, 85-87, 92, 93f, 102-104
Dexamethasone, 475t, 485t
Diabetes mellitus, 442-444, 442t
Diagnosis
casts for, 276-278
restorative vs. surgical, 279-280
Diazepam, 483, 484t
Diet
calcium conservation in, 579-581, 580t
immediate loaded prosthesis and, 818
Dietary supplements. *See* Herbal supplements.
Digital radiography
vs. film radiography, 42t, 59b
for intraoperative imaging, 59, 60f
for presurgical imaging, 41-42
system for, 42f
Disease, systemic
dentures and, 16
elderly patient and, 422
evaluation of risk in, 435t
implant therapy and, 434
physical status classification and, 434b
soft tissue changes in, 13
Distraction osteogenesis, 1045
Ditching, 72
Dolder bar, 298-299
Doxycycline, 965
Drugs. *See* Medications.
Dynamization, 624

E

Early loading failure. *See* Occlusal loading, early.
Eating. *See* Mastication process.
Economic considerations. *See also* Costs.
age and, 4
for fixed implant prosthesis, 12, 325
for implant overdenture, 296
pretreatment prosthesis for, 270
for prosthetic options, 92, 94
for selecting limited treatment, 293-294, 314
for tooth replacement options, 5, 7, 252
Ectodermal dysplasia (ED), 453-454
Edentulism
free-end, 8
partial. *See also* Mandible; Maxilla; Premaxilla.
classification of, 406-413
evaluating teeth adjacent to, 255, 819b
immediate loading for, 819-823, 820-824f
prevalence of, 8
prosthetic options for, 94b, 369-370
treatment planning for, 378-379
single tooth. *See also* Single-tooth replacement.
causes of maxillary, 340b
Edentulism—cont'd
single tooth—cont'd
comparing options for, 5-7
cosmetic replacement of, 343
maintaining vs. replacing, 329-331, 329b
posterior, 327, 668
prevalence of, 5, 5f
total/complete. *See also* Arches, dental.
bone loss in, 9-12, 10-11f, 11b, 178
classification of, 413-417
comparing options for, 15-21
evaluating arch form in, 253-254, 254f
facial esthetics and, 13-15
immediate loading for, 800-801
prevalence rates of, 8-9, 9f
prosthetic options for, 92-94, 92b, 293-294, 314-315, 369-370
psychological effects of, 17b
soft tissues changes in, 12-15, 13f
treatment planning for, 379-387
Education
on bone loss, 293-294
and edentulism rate, 8
on implant care, 1073, 1075
Effort arm, 148
Egyptians, 26
Elasticity (E), modulus of
and bone density, 138-140, 140f, 813f
and bone response to stress, 632-633, 632f, 634f
immediate loading and, 813-814
in implant design, 222
and stress/force, 77, 78f, 549
Elderly. *See* Aging population.
Electrochemical couples, 515
Electronic surgery (ES), 52-53, 53-55f
Elevation
posterior maxilla, 396-399f
sinus membrane, 944-946
subnasal, 789-795, 792-794f
Elevator, dental, 871-872
Embolism, air, 502
Embrasure, 98f
Emergence profile, 171f, 362f, 751f
Enamel stress lines, 113
Enameloplasty, 115, 240, 339
Endocarditis, bacterial, 440-442
Endocrine disorders, 442-446
Endocrinology
bone adaptation and, 625
calcium conservation and, 581
Endodontic treatment and prognosis, 252f, 267
Endosseous implant. *See* Endosteal implant.
Endosteal implant. *See also* Root form implant.
bone health and, 18
definition/types of, 26
generic terminology needed for, 27-28
Endosteal plexus, 498
Endurance limit, 110f
Engineering principles, 77
Engram bruxing pattern, 108f, 109
Epinephrine, 483
Epistaxis. *See* Bleeding.
Epithelium, junctional, 74-75f, 1064-1065t
Erythrocytic disorders, 446-447
Erythromycin, 470b, 473t, 965
Essix appliance, 334, 344f, 351
Esthetic zone
and bruxism, 109
for FP-1 prosthesis, 94-95, 95-96ff
for FP-2 prosthesis, 96-97f
for FP-3 prosthesis, 98-99f
Esthetics
bone grafting and, 975f
bone loss and, 13-15, 15b
dental implants and, 21
implant dimensions and, 174-175, 175b
index of, for crown, 1082-1083
maxillary arch replacement and, 367
pretreatment prosthesis for, 270
single-tooth implant and, 347-348, 740-744
tooth position and, 14f
Estrogen replacement therapy, 581, 582
Ethnic background, 582
Evaluation
postimplant
criteria for, 1056b, 1077b
imaging for, 59-62
literature review of, 1055
of natural teeth, 1078b
preimplant, 233-240, 270-271, 271b
Exposure, implant
in mandibular surgery, 715
procedure for, 723-731
spontaneous early, 721-723, 721f
Extraction, tooth. *See also* Socket grafting.
atraumatic, 870-874, 871-872f, 874f
bone loss after, 10
edentulism due to, 8
healing of sockets after, 874-875, 876f
immediate implant after, 755-758, 897, 899
risk for, 5
for site development, 354b
treatment rationale and, 6-7, 250-253, 252b
Exudate, 74f, 1069f

F

Fabrication
computer-assisted, 285-288
drill/3D printing for, 284f, 287f
prosthesis/laboratory, 33
radiographic templates for, 62-63, 64f
of surgical guides, 280-283
Facebow transfer, 277-278, 278-279f
Facial artery hemorrhage, 714
Facial changes
bone/tooth loss and, 13-15, 15f
dental implants and, 19, 20f
teeth position and, 14f
Facial deformities, 1039-1043
Facial sutures, 585-588
Faciolingual axis, 550, 551-552f
Faciopalatal width, 350, 362f, 742
Failure/complications, implant. *See also* Fatigue fracture/failure.
assessment scale for, 1079t
biomaterial cracking and, 512-515
criteria for, 1077
design and, 200-202, 201f, 225-226
force delivery and, 549-555
immediate loading and, 812f

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Failure/complications, implant—cont'd
of mandibular surgery, 696t, 709-716
nomenclature for, 720-721, 720t
parafunctional forces and, 106-107
of premaxilla surgery, 793, 795
repair algorithm for, 1081-1082, 1082f
risk factors for, 84-85, 105
single-tooth, 342, 765-766
size and, 160-166, 167f, 169f
in stress theorem, 80-81, 80f, 83-87
treatment of, vs. natural tooth, 1068-1069
types of, 68-72, 68f, 69b
Fat, body, 20
Fatigue fracture/failure
biomaterials and, 512-515, 554f
due to bruxism, 110
due to clenching, 113-115
fundamentals of, 554-555, 554f
implant design and, 223-225f
in stress theorem, 71-72, 71f
Fatigue strength, 631-632
Fear, patient, 341-342
Fentanyl, 484t, 486
Fibroblast growth factors (FGFs), 846, 850
Fibrous dysplasia, 451
Financial considerations. *See* Economic considerations.
Fingerprints on implant surface, 533
Finite element (FE), 629
Fistula
chronic skin, 503
oroantral, 967, 968f
Fixation, rigid
definition of, 27
determination of, 728
parafunctional forces and, 106
percussion and, 1059
surgical methods to achieve, 28
Fixed partial denture (FPD). *See* Denture, partial.
Fixed prosthesis (FP)
advantages of, 94b, 315b
comparing contours of, 103f
force factors on, 315-316
immediate loading for, 814-818, 815-817f
indications/materials for, 94-101
comparison of, 100t
FP-1, 94-95, 95-96f
FP-2, 95-97, 96f, 98f
FP-3, 97-101, 99-100f
for mandibular replacement, 314-315, 318-325
for maxillary replacement, 379-383
vs. removable, 315f
Fixture, 212
Flipper
definition of, 343, 351f
for transitional restorations, 334, 344
Flossing. *See* Hygiene, oral.
Flumazenil, 486
Fluorescent labels, 562-563, 563f, 576f
Fluoroquinolones, 472, 965
Follow-up/recall. *See* Evaluation.
Food entrapment, 94, 315
Force. *See also* Bite force, normal; Cantilevers; Masticatory forces; Occlusal force; Stress; Torque force.
clenching as "sneaky," 112
Force—cont'd
delivery of, and failure, 549-555
biomaterials and, 512-515
implant design and, 203-204, 206-207
parafunctional, 85, 105
direction of, 161-162, 204-205. *See also* Load angle.
fundamentals of, 543-546, 544f, 545-546f, 545t
immediate loading and, 812-813, 818
to implant-fixed prosthesis, 315-316
magnification of, 162-163
cantilevers and, 85
treatment planning and, 118-120
magnitude/duration of, 161, 222-223
maxillary/mandibular, 369f
normal, on teeth, 105b
in stress theorem, 69-72, 80-84, 85
treatment planning and, 84-87, 126-127
Forceps, extraction, 871-873
Foreign body in maxillary sinus, 924, 925f
Fractures, implant. *See* Fatigue fracture/failure.
Framework fracture, metal, 71f
Freeze-dried bone (FDB), 859t, 878
Fretting corrosion (FC), 513
Frost's chondral growth force, 623f
Fungal sinusitis, 921-922
Furcation treatment, 252
Fusobacterium nucleatum, 1075

G

Gag reflex, 21
Galvanic corrosion (GC), 513, 514f
Gastrointestinal complications
antibiotics and, 470b
NSAIDs and, 478t
Gene expression, osteoblast, 627-629
Genioglossus muscle, 496
Gingiva
bleeding of, 1067-1068
ditching of, 113
edentulism and, 12-13
fiber groups of, 75
keratinized, 1062-1063
margin of, 74f, 75f
microflora of, 1068t
Gingivitis, 1068, 1078t
Glass ceramics, bioactive, 861t, 863
Glucocorticoids
for inflammation reduction, 474-476, 475t
protocol for prescribing, 485t
for sinus graft surgery, 928b
Glucose, serum, 432
Gluteus medius muscle, 1039-1043
Glycosylated hemoglobin, 433-434, 434t
Gnathodynamometer, 106
Gold, 518
Grafting. *See* Available bone; Bone grafts/grafting; Sinus graft surgery; Socket grafting.
Grinding. *See* Bruxism.
Growth factors
in bone graft, 846, 849-850
in bone healing, 849b
in platelet rich plasma, 850b
in socket graft, 888-890
Guards, occlusal. *See* Occlusal guards.
Guided bone regeneration (GBR)
for barrier membrane bone graft, 890-894, 891-893f
immediate implant after, 897-901, 900f
for one bony wall defect, 882-888

H

Hader clip/bar, 102, 299f
Haemophilus influenzae
after graft surgery, 927, 965
in maxillary sinus, 913, 920
Hafnium, 518
Halo formation, 399
Hamular notch, 490, 492
Head/neck infections. *See* Infection.
Healing abutment, 31
Healing period
after bone grafting, 852-855, 853f
and bone-implant contact, 599
after guided bone regeneration, 899-900
of sinus graft, 396t, 939t, 951-952
Health-disease continuum, 1077-1081, 1079-1080f, 1079t
Heart disease. *See* Cardiovascular disease.
Heat stress
factors for minimal, 691b
implant failure and, 69f
marginal bone loss and, 73
osteotomy and, 648-649, 648b, 659t
surgery and, 803-804
Hematologic disorders, 446-448
Hemidesmosomal seal, 75
Hemoglobin evaluation, 429
Hemorrhage, life-threatening, 694t, 713-715
Herbal supplements
bleeding and, 458b
implant therapy and, 458
swelling/blood pressure and, 436b
"Herodontics" and tooth prognosis, 251
Hert's curve, 623f
High water restorations, 247
Histochemistry of bone response, 562
Home care, 1073
Hooke's law, 549
Human immunodeficiency virus (HIV), 454-455
Hydrocodone, 478t, 479, 480-481t
Hydrocortisone, 475t
Hydroxyapatite (HA)
bulk properties of, 520-523, 520-521t, 522f
as implant coating
for bone-implant contact, 612-615
force loads and, 201f, 203-205, 205f, 220f
immediate loading and, 812
surface-tissue interface of, 530, 532-533
socket grafting and, 878
xenografting and, 861-862
Hygiene, oral
marginal bone loss and, 74
options for maintenance, 1073, 1075-1076
plaque biofilm and, 1074
removable prostheses and, 102
splinted implants and, 157-158
Hygienist, dental, 1074b
Hyperalgesia, 476

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Hyperbaric oxygen therapy, 461
Hypertension, 435-437, 437t
Hypnotic sedative, 484t
Hypodontia, 351
Hypoplasia of maxillary sinus, 919

I

Ibuprofen
  as adjuvant, 480t, 481t
  for pain management, 478-479, 478t
  protocol for prescribing, 485t
Ice bags, 476
iDent Imaging, 286
IGI (Image Guided Implantology), 289
Iliac artery, 1022
Iliac crest block bone graft
  complications after, 1034-1035
  considerations for, 1014-1015
  fixation of, 846f
  harvest surgery of, 1019-1021, 1019-1020f
  history of/rationale for, 976, 1014f, 1017t
  implant survival and, 1015-1019, 1016-1018t, 1016f
  neurovascular at risk in, 1022-1023f
  recipient site surgery for
    Division C–h, 1023-1026, 1028-1031f
    Division C–w, 1023, 1024-1027f
    Division D, 1026-1033, 1031-1038f
  reconstruction after, 1021-1022
  vascularized, 1039-1043
Iliohypogastric nerve, 1022
Imaging, diagnostic
  of bone response, 564-569
  intraoperative, 38, 54, 58-59, 288-290
  overview/summary of, 38-39, 39b, 64
  postprosthetic, 38, 59-62
  preprosthetic, 40b, 62-63, 285-288
  presurgical
    to identify vital structures, 42f, 56-58
    objectives for, 38, 39-40
    radiography options for, 40-46
    summary of, 58
    tomography options for, 46-56
    for treatment planning, 58b
Immediate loading. *See* Occlusal loading.
Immunosuppressive medications, 457
Impact load, 549
Implant positions. *See* Key implant positions.
Implant systems (manufactured), 27-29, 29f, 332
Implantation, dental. *See* Osteotomy, implant; Surgery, implant.
Implants, dental. *See also* Endosteal implant.
  advantages of, 17-21
  criteria for evaluating, 1055, 1056b, 1077-1078b
  mini-type of, 595
  number of, in U.S., 3
  rationale for, 3, 9, 21
Implant-tissue interface. *See* Bone-implant contact (BIC); Surface-tissue interface.
Impression transfers, 33, 34-35f, 59
Incas, 26
Incisal guidance, angle of, 109, 110f
Incision, surgery
  in root form surgery, 687, 715
  in sinus graft surgery, 941, 962-963
  for surgical harvest, 986-987f
Incision, surgery—cont'd
  suture design principles for, 844-845f
  for tissue drape, 750-755, 752f
Incisive foramen
  implant in region of, 795, 796-797f
  implant position and, 744-745, 745f
  number of implants and, 318
Incisivus fossae, 496
Incisivus labii superioris muscle, 491
Incisors
  mandibular, 238
  maxillary
    in bruxism, 109
    extraction of, 758
    implant positions for, 749f
    missing anterior, 370-372
    single-tooth implants, 740-743f
Income level and edentulism rate, 9
Indexes, physiologic, 562-568
Inertia, moment of, 555
Infection
  in bone graft, 840
  classification of wound, 468b
  complications/spread of, 508-509, 966-967
  dental implants and, 509-510
  of head/neck, 503-505f, 507
  mandibular, 505-508
  maxillary, 503-508, 913, 915t
  microorganisms in, 469b
  probability of wound, 469t
  repair algorithm for, 1081
  risk factors for, 468b
  sinus graft surgery and
    evaluation/treatment of, 926b, 963-967
    of sinus mucosa, 963-964
  stages in/routes for, 502-503
  terms used for, 503
Inflammation
  of maxillary sinus, 919-922
  peri-implant, 1074-1075
  postoperative, 474-476
Infraorbital artery, 494f
Infraorbital nerve, 492f, 493, 963
Infratemporal space, 503-505
Instruments
  maintenance, 1076
  surgical, 716f
Insulin-like growth factors (IGFs), 846, 850
Integration. *See* Osteointegration.
Interactive computed tomography (ICT), 52-54, 53-55f
Interarch distance, 245
Interfacial zones. *See* Bone-implant contact (BIC); Implant-tissue interface; Surface-tissue interface.
Iron-based alloys, 517-518
Irradiation, 459-461, 460b
Irrigation, oral, 1076
Isotropic, transversely, 629f

J

Jaw
  bone loss and atrophy of, 14-15, 14f
  opening of, in clenching, 113
  osteonecrosis of, 456
  reconstruction of, 1039-1043
Joints, prosthetic, 453
Jowls, 14f, 15

K

Kennedy classification
  Class I, 407b, 409-410
  Class II, 408b, 410-412
  Class III, 408b, 412
  Class IV, 408b, 412-413
  history of, 406
Key implant positions
  abutment options for, 147-154, 147b, 148f, 152b
  early exposure of, 730-731
  for edentulous premaxilla, 769-770
  maxillary reconstruction and, 370-374
  stress/force factors of, 85-86
Kiesselbach's plexus, 791-792
Kois Dento-Facial Analyzer System, 277, 279f

L

Laboratory evaluation. *See also* Blood tests/plasma levels.
  in medical evaluation, 427-434
  normal values in, 428f
Laboratory fees for prosthesis, 94
Laboratory technician, dental, 348
Lactic dehydrogenase (LDH) level, 433
Lactobacillus acidophilus, 840
Lamellar bone, 569-570, 805-806
Landmark ID. *See* Reflection.
Laser ablation, 615
Late failure. *See* Occlusal loading.
Leukocytic disorders, 447-448
Levator anguli oris muscle, 492
Levator labii superioris muscle, 492
Levofloxacin, 472, 473t, 965
Lidocaine, 481-482t
Life expectancy
  average, 3f
  dental implants and, 4-5
  dentures and, 16
  estimated, 5f
  increase of, 4f
Lifestyle-related issues and implant therapy, 458-460
Lincosamides, 965
Lingual artery hemorrhage, 713-714
Lingual nerve, 492f, 497-498, 713
Lip infection, 504
Lip line/position
  bone loss and changes to, 13-15, 14-15f
  evaluation of, 241-243, 242-243f
  fixed prosthesis design and, 96-97f, 96f, 98-99f
  maxillary reconstruction and, 370
  support of maxillary, 775, 775-783f
Liver disease, 449
Load. *See* Force.
Load angle
  of applied force, 162f
  body design and, 204-205
  cortical bone strength and, 162t
  force magnification and, 119-120
  force of, 85
  fundamentals of, 161-162, 162f
Load transfer. *See* Stress.
Load-bearing zone, ideal, 808
Loading. *See* Occlusal loading.
Longevity, 1056
Lorazepam, 484t

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Ludwig's angina, 507
Lung function, 421. *See also* Chronic obstructive pulmonary disease (COPD).
Lupus erythematous, systemic, 454
Lymphadenitis, 502-503
Lymphatic drainage, 495, 508-509

M

"Mach band effect," 60
Macrolides, 471, 965
Maestro Dental Implant System, 332
Magnetic resonance imaging (MRI), 54-56, 56b, 917
Maintenance of implants
  issues in, 1073-1074
  procedures for, 1076
  quality scale for, 1078-1081, 1079t
  repair algorithm for, 1081
Malocclusion
  age-related tooth loss and, 13
  evaluation/management of, 583f, 589, 595
  temporomandibular joint and, 560f, 561, 583b, 583f
Mandible. *See also* Arches, dental; Mandibular ramus; Mandibular symphysis.
  anatomical features of, 300f, 490f, 495
  bone loss in, vs. maxilla, 83
  dynamics of, 316-317, 316f
  edentulous
    effects of, 9-12
    fixed restorations for, 314-315, 318f, 325
    implants vs. dentures for, 18-19, 18f
    overdentures for, 293-294, 310
  infections of, 505-508
  innervation of, 492f, 497-498
  morphology (osteology) of, 558-559
  muscles attached to, 491-492f, 495-497
  preimplant evaluation of
    incisal edge position, 238
    lip line, 243
    occlusal planes of, 238-240, 239-240f
  stress patterns in, 560f
  surgery complications of, 696t, 709-716
  surgical approach to
    anterior, 684-698
    posterior, 698-709
  vascular supply of, 494f, 498-501
Mandibular canal, 56-57, 702-703
Mandibular lingual concavities, 57
Mandibular ramus
  block bone graft site of, 976f, 1005t
  complications after harvesting, 1006-1007
  evaluating graft site of, 991-992, 996f, 999f
  imaging to locate/evaluate, 57f, 1003f
  surgical harvest of, 993-998, 997-998f, 1000-1002f
Mandibular symphysis
  block bone graft site of, 976f, 982, 985, 1005t
  complications after harvesting, 1005-1006
  guidelines for harvest of, 988f
  imaging to locate/evaluate, 57f, 993f
  surgical harvest of, 985-991, 988-992f, 994-996f
Marginal bone loss
  animal/clinical studies on, 78-80, 79f
  bone physiology and, 82-83
Marginal bone loss—cont'd
  description of, 72-73, 72f
  hypotheses for, 73-76
  implant design and
    coatings for, 205f
    hypotheses of, 80-81, 80f, 83-87
    occlusal load in, 207-209
    rates of, 201-202, 201t, 202f
  implant success and, 1059-1060
  occlusal force overload and, 76, 81-82
  other disciplines on, 76-78
Markers (bone labels), time, 562-568
Mass, 543, 544f
Masseter muscle, 316-317, 497
Masseteric space, 497
Mastication process
  dental implants and, 20
  dentures and, 16
  edentulism and, 13
Masticatory forces. *See also* Bite force, normal; Occlusal force.
  arch position and, 124-125
  in bruxism, 109
  edentulism and, 11-12
  evaluation of, in treatment planning, 85
  increasing range of, 107
  natural vs. implant, 105-106, 105b, 106t
Masticatory space, 506
Materials
  for bone grafting, 855-863, 855b
  for implant maintenance, 1076
  for restorations. *See* Biomaterials.
  for sinus graft surgery, 946-947
Maxilla. *See also* Arches, dental; Maxillary sinus.
  anatomical features of, 490-491, 490f
  anterior. *See* Premaxilla.
  bone loss in, vs. mandible, 83
  edentulous
    effects of, 9-12
    treatment guidelines for, 370-374
    treatment plans for, 379-387, 380b
  implant advantages for, 18
  implant design and, 392-394
  infections of, 502-505
  innervation of, 492-494, 492f
  morphology (osteology) of, 558-559
  muscles attached to, 491-492f
  posterior
    anatomical considerations for, 389-391
    graft option for, 395-396
    implant contraindications for, 905-907
    treatment classifications for, 396-402, 929-930
    treatment history of, 394-395
    treatment plans for, 391-394
  preimplant evaluation of
    incisal edge position, 233-234, 234f
    lip line, 241-243
    occlusal planes, 238-240, 239-240f
  prosthetic options for, 293, 314
  single-tooth replacement in, 346b
  teeth dimensions of, 335t, 348t, 381t, 743t
  tuberosity region of, as donor site, 879, 882f
  vascular supply of, 494-495, 494f, 498-499
Maxillary nerve (V2), 492-493, 940f
Maxillary sinus. *See also* Sinus graft surgery.
  anatomy of
    microscopic mucosa, 912, 912f
    surgical, 491f
    variants in, 917-919, 919f
    walls in, 908-910, 910f
  bacterial flora (normal) in, 913
  clinical assessment of, 913-917
  congestion of, 929, 965-966
  development of, 391-392f, 907-908
  expansion of, after tooth loss, 390-391, 393f
  graft options for, 396-401
  imaging to locate, 58
  implant penetration into, 925f, 967
  infection of
    buccal space, 504
    signs of, 915t
    treatment of, 963-967
    types of, 920-922, 926
  mucus clearance in, 912-913, 912f
  overfilling of, 967
  pathology of, 911f, 919-924
  perforation of membrane of, 956-959, 958-959f
  surgical cysts of, 967-968
  vascular supply/innervation of, 910-911, 911b
Maximal intercuspation (MI), 244f
Mechanical advantage, 148
Mechanical loading, 582, 584
Mechanostat, 584f
Mechanotransduction, 621-624, 622f
Mediators, intracellular, 624
Medications. *See also specific medication or class of.*
  elderly patient and, 421-423
  for implant therapy, 467, 486
  medical evaluation of, 455-458
  for specific procedures, 485t
Medullary plexus, 498
Menopause, 582, 584f
Mental foramen, 56-57f, 687-689f, 701-702, 701f
Mental protuberance, 496
Mentalis muscle, 15, 492f, 496-497
Meperidine, 478t, 479-480
Mepivacaine, 481-482t, 482
Mesiodistal axis, 550, 551f
Mesiodistal space, 348-349, 348f, 669-672, 741-744
Messengers, second, 624
Metals/alloys
  flexure of, 150f
  physical/mechanical properties of, 515-518
  surface-to-tissue interface of, 526-529
  toxic elements from, 515
Metronidazole, 470b, 472, 473t, 965
Microbiota, peri-implant. *See* Bacterial flora; Bacterial plaque complications.
Microcomputed tomography, 567-568, 567-568f
Microgap, 76, 81
Microindentation, 567-568, 567f
Micromovement, 69f
Microradiography, 563-567, 563f, 565-566f
Microstrain zones, 208, 584, 807-808
Midazolam, 483, 484t

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Mini-implants, 595
Misch, Carl E.
available bone classification by, 180f
"barrier by bulk" concept by, 845-846, 845f
bone density classification by, 134-135, 135f, 646
classification of dental treatment by, 435t
edentulous arch classification by, 413-417
on implants in younger patient, 347
maxillary posterior classification by, 395f, 396-401, 399b, 915f, 929-930, 949b
occlusal analyzer by, 240f
phase IV of bone grafting by, 863
Stress Treatment Theorem by, 17
Molars
bite force of, 544t
diameters of, 173f
implant positions for, 151-153
implants for replacing, 6-7, 669-672
prevalence of missing, 5f, 327, 393f
prostheses for replacing, 5-7, 8
replacing mandibular, 329-330f, 329b, 335-340, 340b
replacing maxillary, 370-372
Molybdenum in biomaterials, 517
Moment loads, 550-551, 550-553f
Moment of force, 873
Moment of inertia, 555
Moraxella catarrhalis, 920, 927, 965
Mortality
in dental practice, 502
and dentures, 16
Motile rods, 1068t, 1075
Movement/mobility. *See also* Micromovement.
bone grafting and, 846
classification of prosthesis, 298-299
force loads and, 119f, 553t
implant failure and, 69f
implant success and, 1058-1059, 1058b, 1059f
of mandible, 316
after mandibular surgery, 715-716
of natural teeth, 1057
adjacent to edentulous region, 342-343
in bruxism, 113
when evaluating abutments, 259-261
Moxifloxacin, 473t, 965
Mucocele, primary/secondary, 923
Mucoepithelial attachment, 1073
Mucoperiosteum, 499, 912
Mucositis, 920, 1075
Mucous retention cyst, 922
Multiple myeloma, 452
Muscles. *See also specific muscle.*
attached to mandible, 491-492f, 495-497
attached to maxilla, 491-494, 491-492f
in bruxism/clenching, 107, 112-113
extraoral, for grafting, 1039-1043
Mylohyoid motor branch, 492f, 498
Mylohyoid muscle
anatomy of, surgical, 492f, 495
denture effects on, 18-19
edentulous effects on, 11f
Myocardial infarction (MI), 438-439, 439t

N

Naber's probe, 690f
Naloxone, 486
Nanoindentation, 630
Naproxen, 478t
Narcotics, 484t
Nasal septum deviation, 919f
Nasal spine
anatomical considerations for, 790-791, 790f
edentulous effects on, 11, 12f, 15f
Nasopalatine nerve, 494
National Institute of Dental Research, 9
National Institutes of Health, 27
Navigation, CT-based surgical, 54, 288-290
Neck infections. *See* Infection.
Neoplasms of maxillary sinus, 923, 924f
Nerves. *See also specific nerve.*
and iliac bone harvesting, 1022
of mandible
anatomy of, 492f, 497-498
damage to, 709-713
of maxilla, 492-494, 492f
of maxillary sinus
anatomy of, 910-911
damage to, 963
Neurapraxia, 711b
Neurosensory exam, 710f, 710t
Neuroticism and tooth loss, 17
Neurotmesis, 712b
New York Heart Association (NYHA), 440b
Nickel in biomaterials, 517-518
Night guard
for bruxism, 110, 112f, 114-115
for clenching, 115f
Nitroimidazoles, 965
Nocturnal bruxism. *See* Bruxism.
Noma, 503
Nonfunctional immediate teeth (N-FIT)
advantages of, 825b
indications for, 819b
protocol for, 819-823, 825b, 826-828f
Nonfunctional immediate/early restoration
definition of, 799
for single tooth replacement, 823-832, 826-828f, 830-832f
Nonopioid analgesics. *See* Analgesics.
Nonsteroidal antiinflammatory drugs (NSAIDs)
gastrointestinal complications and, 478t
for inflammation reduction, 474f
for pain management, 478
protocol for prescribing, 485t
supplements interacting with, 458b
Nuclear volume morphometry, 568
Number, implant
for fixed prostheses, 318f, 320-321f, 323f
for immediate loading, 808, 809f
in stress theorem, 85-86, 86f
treatment plans for, 154-157, 155-156t

O

Obesity and dentures, 16
Occlusal awareness, 19-20, 126
Occlusal disharmony, 110-111
Occlusal force. *See also* Bite force, normal.
abutment options and, 255-256
arches and, 125-126, 126f
denture vs. implant, 20
implant design and, 202-207
in marginal bone loss theory, 76, 81
Occlusal force—cont'd
measuring, 106
normal vs. edentulous, 15-16
posterior vs. anterior, 391-392
in stress theorem, 68-69
in surgical failure, 69
Occlusal guards
for bruxism, 110-111, 112f
for clenching, 114-115, 115f
Occlusal height, 551, 552f
Occlusal load
bone density and, 85
direction of, 628-630
duration of, 631-632
immediate loading and, 813
implant design and, 207-208
in implant failure, 69-70
implant size and, 86
location of, 632-633
in marginal bone loss, 72f
animal/clinical studies on, 78-80, 79f
bone physiology and, 82
discussion on, 81
rate of, 630-631
types of, 543
Occlusal loading
bone strength and, 82-83, 83f, 85
early
bone-implant contact and, 599, 600t
definition of, 799
failure during, 68f, 69b, 70, 81, 201-202, 729
immediate
benefits/risks of, 833-834
definition/example of, 799, 800f
diet/guidelines for, 818
of fixed prosthesis, 814-818, 815-817f
histologic response of bone to, 806-807, 806f
protocols for, 800-801, 801-804f
surgical approach to, 29f, 31
trauma as rationale for, 801-806
marginal bone loss after, 72, 76-80, 78f
mature/delayed/staged
bone-implant contact and, 599, 600t, 601
definition of, 799
failure during, 729
plasma spray coating for, 203
treatment planning and, 84
Occlusal planes, evaluation of, 109, 238-240, 239-240f
Occlusal radiography, 42-43, 42b, 43f
Occlusal stress/overload. *See also* Strain; Stress.
bone resorption and, 77f
bone type and, 82
bone vs. titanium and, 808f
decreasing risk of, 802-803, 807-814
immediate loading and, 801
implant design to manage, 202-208
implant failure and, 69-72, 69f, 70f
marginal bone loss and
animal/clinical studies on, 78-80, 79f
bone physiology and, 82-83
discussion on, 81-82
implant design and, 80-81, 83-87
theories of, 76-77
parafunctional forces and, 106-107

WWW.HIGHDENT.IR

Occlusal vertical dimension (OVD)
bruxism and, 109
preimplant evaluation of, 234-238, 235f, 237f
pretreatment prosthesis and, 268f, 269-270
Occlusal width, 554
Occlusion, dental. *See also* Malocclusion.
esthetics of, 13, 14f
evaluation of existing, 244-245, 244f, 277-278
Occlusoapical axis, 550
Offset loads, 119
Opioid analgesics. *See* Analgesics.
Orbicularis oris muscle, 491, 492f
O-rings, 34f, 298-299
Orthodontics, implant-anchored
mechanics of, 588-595
site development for, 353-354
Orthotropic, 629
Osseointegration
biomechanical environment of, 621
early loading failure after, 70
history of term of, 27
probing to measure, 1064-1065t
surface energy and, 534
titanium and, 516
Osteitis deformans, 452
Osteocalcin, 625, 628
Osteoconduction, 860-863
Osteogenesis
after bone grafting, 857-858
by distraction, 1045
Osteogenesis imperfecta, 452
Osteoinduction, 858-860, 859t
Osteointegration. *See* Osseointegration.
Osteology, 557f, 558-562
Osteolysis, 81f
Osteomalacia, 451
Osteomyelitis, 452, 503, 503f
Osteonecrosis of jaws, 456
Osteopontin, 624-625, 628
Osteoporosis, 449-451, 582
Osteoradionecrosis, 461
Osteotomes, 664f, 784f
Osteotomy, implant
and bone density
D1, 648-655, 648b
D2, 656-657
D3, 661-662
D4, 663
preparation for, 659t
bone tap in, 653f, 662f
bone trauma from, 801-806, 805f
calibrated ("graduated wedges"), 778
and marginal bone loss, 73
for single-tooth replacement, 679-682
stage two of, 673-674. *See also* Uncovery procedure.
complications during, 721-723, 731-736
successful implants at, 721b
transitional prosthesis for, 736-737
Overdenture, implant-supported
mandibular
vs. fixed prosthesis, 296b, 314-315
from, to fixed prosthesis, 293-296
immediate loading for, 818-819, 820-824f
implant site selection for, 299-300
Overdenture, implant-supported—cont'd
mandibular—cont'd
movement of, 298-299
pros/cons for, 295-298, 295b, 297f, 298b
selection criteria for, 301b, 303b, 305b, 308b
treatment options for, 300-310, 301t, 302-307f, 309f
maxillary, 369, 383-387, 774
Overjet, vertical, 109, 113
Overload. *See* Occlusal stress/overload.
Over-the-counter supplements. *See* Herbal supplements.
Oxycodone, 478t, 479, 480t
Oxygen therapy, hyperbaric, 461

P

Paget's disease, 452
Pain
absence of, as criteria, 1056-1057
classification of, 480
edentulism and, 11, 17
elderly patient and, 423
Pain management. *See also* Analgesics; Anesthetics, local.
postoperative, 477-481, 478t
preemptive, 476
protocols for acute, 480-481, 481b, 481t
Palatine nerve, 493-494
Palladium, 518
Panoramic radiography
with ball bearings, 46f
beam angles in, 45f
to evaluate donor site, 996f, 1003f
of maxillary sinus, 915-917, 916f
positioning problems in, 46t
for postprosthetic evaluation, 62
for presurgical imaging, 44-46
pros/cons for, 45b
Papilla. *See also* Tissue, soft; Uncovery procedure.
position/contouring of, 752-754f
presence/absence of, 359f, 741f
in socket grafting, 891f
solution for inadequate, 766f
Papilla deficiency, interdental, 765, 766f
Parafunctional forces, 85, 106-107, 584-585
Parallelism, 266
Parapharyngeal space, 507
Parathyroid hormone (PTH), 625
Parathyroidism, hyper-, 451
Paresthesia, 11, 12f
Parkinson's disease, 423
Parotid space, 507
Patient, implant
fear/compliance by, 341-342
guidelines for younger, 347
laboratory evaluation of, 427-434
medical evaluation of, 421
medical history of, 423-427, 424-425f
physical status classification of, 434-435, 434b
"Pelican" for extraction, 871-872
Pellicle, 1074
Penicillin V, 470, 840, 965
Peptide (P-15), synthetic residue, 863
Peptide hormones, 581
Percussion of implant, 1059
Periapical (PA) radiography
to evaluate implant, 1061f
film positioning in, 41f
of maxillary sinus, 916
for postprosthetic evaluation, 60-61, 61f
for presurgical imaging, 40-41
summary of, 40b
Peri-implantitis, 1068
Periodontal disease/status
classification of, 1078t
implant prognosis and, 267
implant success and, 1068
tooth prognosis and, 251
Periodontal indices, 1055
Perioral forces, 105-106, 105b
Periosteal plexus, 499
Periosteal reflection hypothesis, 73
Periotest, 1059f
Periotomes, 872-873, 872f
Perivertebral space, 507-508
Perivisceral space, 507-508
Permucosal extension (PME)
definition of, 31, 32f
in single-tooth replacement, 754-755, 764
Pharmacology. *See* Medications.
Phlegmon, 503
Phonetics, 21
Phosphorus levels, 433
Physical status classification, 434b
Physics Forceps, 873-874, 874f
Physiologic zone, 808
Pier abutment, 262-263
Piezoelectric effect, 10
Pincers (forceps), 871-872
Plaque complications. *See* Bacterial plaque complications.
Platelet count, 431
Platelet-derived growth factors (PDGFs)
graft stabilization and, 846, 849-850
platelets/mL and, 851f
in socket grafting, 876-877
Platelet-poor plasma (PPP), 851
Platelet-rich plasma (PRP)
blood clot formation and, 851f
in bone graft, 844f, 850f, 852f
growth factors in, 850b
preparation of, 851-852
Platinum, 518
Poisson's ratio, 548
Polarized light, birefringence of, 562, 563f
Polycythemia, 446
Polymers/composites, 524-525, 525t
Population. *See* Aging population.
Porcelain. *See* Materials.
Porphyromonas gingivalis, 1074-1075
Position. *See* Arches, dental; Key implant positions; Single-tooth implant.
Prednisone/prednisolone, 475t
Pregnancy, 446
Premaxilla. *See also* Bone spreading.
arch form of, 374-377
edentulous
compromised conditions of, 368b
effects of, 367-369
prosthetic options for, 382-387
treatment guidelines for, 370-377
treatment options for, 369-370
treatment plans for, 374t, 378-382

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Premaxilla—cont'd
single-tooth replacement for, 340
surgical considerations for, 769, 795-796
bone spreading, 779-787
lip support, 775-778
subnasal elevation, 789-795, 792-794f
treatment planning, 769-774
Premaxillary wing, 795
Pressure, 544f
Pretreatment prosthesis, 268-271, 269b
Prevotella intermedia, 1074-1075
Probing depths, 74f, 1063-1067, 1063f, 1066f
Prognosis factors, 250-251, 252f, 255-256, 266-267
Prophylaxis. *See* Antibiotics.
Propofol, 484t, 486
Propoxyphene, 478t
Prosthesis, implant-supported. *See also* Design, implant/prosthesis; Key implant positions; Number, implant; Transitional restorations/prostheses.
advantages of, 6-7, 17-21, 18b, 21f
classifications of, 32-33, 94t
evaluation criteria for, 1055, 1056b, 1077b, 1078b
existing, evaluation of, 253
fixed. *See* Fixed prosthesis (FP).
removable. *See* Removable prosthesis (RP).
shift to, in dentistry, 4
teeth adjacent to, 255
Prosthesis, soft tissue–supported. *See* Denture, complete, removable soft tissue–borne.
Prosthetic joints, 453
Prosthodontic classification, 94t
Prosthodontics, preimplant, 233, 270-271, 271b
Prothrombin time, 430-431, 430t
Pseudocysts, 922
Pseudoperiodontium, 570
Psychological health
dental implants and, 20, 21, 93
elderly patient and, 423
implant therapy and, 459
pretreatment prosthesis for, 270
tooth loss and, 17b
tooth replacement options for, 7
Pterygoid muscles, 250, 496
Pterygopalatine fossa, 492-493
Pulmonary disease, 448-449, 448t
Pulse, evaluation of, 426-427
Pulsus alternans, 427

Q

Quality of health scale, implant, 1077-1081, 1079t
Quinolones, 470b

R

Radiation exposure, 39, 50
Radiation therapy, 461t
Radio-frequency glow discharge technique (RFGDT), 534-535
Radiography
of bone density, 137-138
of bone response, 563-568f, 564-569, 568f
to evaluate implant, 1060-1061, 1061f
to identify vital structures, 42f, 56-57, 56f
intraoperative, 58-59
Radiography—cont'd
of maxillary sinus, 915-917
postprosthetic, 59-62, 61f
postsurgical, 59, 60f, 61f
preprosthetic, 62-63
presurgical, 40-46
sequence for, 59b
Radiotherapy, 459-461
Recombinant human platelet-derived growth factor (rhPDGF), 890
Reflection (landmark ID)
in mandibular surgery, 687-691, 688-689f, 699-701
in single-tooth replacement, 679
in sinus graft surgery, 941
Regional acceleratory phenomenon (RAP), 575
in bone grafting, 847-848, 847f
in maxillary surgery, 791
in sinus graft surgery, 946
in socket grafting, 876
Reimbursement, 4-5
Removable partial denture (RPD). *See* Denture, partial.
Removable prosthesis (RP). *See also* Overdenture, implant-supported.
advantages of, 92b
comparing support of, 103f
indications/materials for
RP-4, 101-102, 101f
RP-5, 102
Repair bone. *See* Woven bone.
Resin veneer, fracture of, 71
Resin-bonded restoration
fixed (FPD), 328-329, 328b
removable (RPD), 344-345, 345b
Resistance arm, 148
Resorbable blast media (RBM) surface, 611f
Resorbable graft material (RGM), 876
Respiration, evaluation of, 427
Retention
abutments for, 31
dental implants and, 20, 21
dentures and, 17
Retention cysts, 922
Retromolar triangle, 496
Reversal agents, 486
Reverse torque testing (RTT), 728-730
Rheumatoid arthritis, 454
Rhinosinusitis, acute/chronic, 920-921, 926, 963-967
Rinse, oral
for sinus graft surgery, 928
for sinus infection, 966
Root configuration/surface area, 266
Root form implant. *See also* Body, implant.
abutments to
attaching, 28-29
categories of, 31-33, 32-33f
defining term of, 26, 28
fabrication in, 33, 34f, 35f
generic components of, 28-34, 31f, 34-35f
history of, 26-27, 26-27f
imaging to locate components of, 59, 60f, 61f
mandibular
anterior, 684-698
complications of, 696t, 709-716
Root form implant—cont'd
mandibular—cont'd
posterior, 698-709
manufacturers' systems of, 27-28
Root resorption, 354-356, 355f

S

Safety zone
imaging to locate, 56-58
in mandibular surgery, 700-708, 704f, 704t
Saucerization, 72
Scalers, ultrasonic/sonic, 1076
Scapula for jaw graft, 1043
Schneiderian membrane, 493
Schwann cell column, 501
Scialom implants, 168, 169f
Scleroderma, 454
Screw root form implant, 29-30
Screws, implant
fractures of, 71-72, 71f
loosening of, 70-71, 70f
types of, terminology for, 31f, 32
Seal phenomenon, biological, 75
Sedative agents, 483, 484t, 486
Sensation, altered, 710-711, 711b
Sensory innervation
damage to, 710-711
of mandible, 492f, 497-498
of maxilla, 492-494, 492f
Sex hormones, 581
Sharpey's fibers, 570
Side constraint, 633
Single-tooth implant. *See also* Single-tooth replacement.
anterior
adjacent dentition to, 356-357
complications of, 360-362, 765-766
contraindications for, 350b
design for, 360-362
dimensions for esthetic, 740-744, 748f
factors influencing, 345-351, 346b
indications for, 351-360
period of placement of, 751f
positions/angulations for, 744-750, 744-745f, 747f
site development of, 353-354
posterior
advantages of, 333b
body design for, 335, 336f
contraindications/limitations of, 333-335
dimensions for esthetic, 174-175, 340t
indications for, 331-333
Single-tooth replacement. *See also* Edentulism; Single-tooth implant.
anterior
alternate options for, 739-740
esthetics and, 174
after extraction, 755-758
flow pattern for, 762b
multiple, 174, 769, 773-774, 774-775f
options for, 341-345, 341b
soft tissue surgery for, 750-755, 758-765
transitional prosthesis for, 755
immediate restoration for, 823-832, 826-828f, 830-832f
implants vs. non-implants for, 5-7, 6-7b, 6-7f

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Single-tooth replacement—cont'd
overview/summary of, 327, 362, 739, 766
posterior
comparing approaches to, 673t
options for, 327-333, 328b, 334t, 668-669
pre-/first-molar implants for, 335-340, 340b, 669-672
steps of surgery for, 678-682
surgery approaches to, 672-677
transitional restorations for, 334-335
progressively-loaded, vs. non-, 82-83
Sinus and Allergy Health Partnership, 965
Sinus Graft Consensus Conference, 968
Sinus graft surgery
complications of
intraoperative, 956-962, 957b
postoperative, 504, 962-968
considerations for, 905, 924-926, 926b
history of, 929
implant insertion after, 949b, 955-956
instructions after, 952-955, 956b
long term results of, 968-969
for posterior maxilla, 395-401, 396t, 401-402f
preparation for, 926-929
techniques for
conventional SA-1, 930-934, 931-934f
SA-2 sinus lift, 934-939, 934-938f
SA-3 sinus graft, 939-950, 939f, 942-945f
SA-4 sinus graft/delay implant, 950-951, 951-955f
vascular healing of, 951-952
Sinuses. *See* Maxillary sinus.
Sinusitis
allergic, 921
fungal, 921-922
odontogenic, 920-922
treatment for, 965
Size, implant
based on natural teeth, 173-174, 173f
bone availability and, 181-183
diameter in, 168-173, 168f, 169-172b, 172-173f
esthetics and, 174-175, 174b
fracture and, 223-225
immediate loading and, 808-810
longer vs. short, 166-168, 166t, 167-169f, 168b
in stress theorem, 86-87
stress/force and, 160-162, 175
Sjögren's syndrome, 454
Skeletal system
adaptation of, 570-571
Angle's classification of, 238, 243, 250, 253
compromise of, 581-582
Skull, stress analysis of, 559f, 560f. *See also* Cephalometric radiography.
Smile
fixed prosthesis design and, 96f, 97f, 98f, 99f
mini-implants and, 596f
preimplant evaluation for, 241-243, 242-243f
after single tooth implant, 358-359f
Smoking. *See* Tobacco use.
Socket grafting, 875-888, 885-887f
bony wall classification for, 876, 877f
Socket grafting—cont'd
for four bony wall defect, 877-882, 879-884f
immediate implant after, 897-901, 900f
layered approach to
positions of layers in, 894-896, 895-899f
procedure, 890-894, 891-893f
for one bony wall defect, 882-888, 888f
soft tissue healing after, 888-890
for thick five bony wall defect, 876-877, 877-878f
for two bony wall defect, 882
Socket seal surgery, 879-882, 883-884f
Space maintenance, graft, 845-846
Spee, curve of, 239f
Speech
dental implants and, 20, 21
fixed prosthesis design and, 96, 97f, 98
preimplant planning and, 240
tooth loss and, 17
Sphenopalatine nerve, 494
Spirochetes, 1068t, 1075
Splinted implants
advantages of, 157-158
to natural teeth, 258-262
pros/cons for, 305-306b
Split finger technique, 761
Stability, 21
Stainless steel, surgical, 517-518, 1076
Staphylococci, 913, 920, 927, 965
Stereolithography, 53-54, 285f
Sterilization techniques, 534-535
Stomatognathic system
evaluation of, in treatment planning, 233
osseous morphology and, 558
parafunctional forces and, 106
tooth loss and, 3, 16
Strain
bone density and, 142-144, 142b, 629-634, 630f
definition of, 76
fundamentals of, 160-161, 547-549
microstrain zones of, 208, 807-808
osseointegration and, 621-624
skeletal health and, 584
Streptococci
in bacterial endocarditis, 440
after graft surgery, 927, 965
in maxillary sinus, 913, 920
Stress. *See also* Occlusal stress/overload; Strain.
vs. biological complications, 68
biomaterial cracking and, 513-515
bone density and, 141-144, 142b
bone loss and, 80-81, 81f
bone physiology and, 82-83
bone response to, 621-624
distribution of, 630
force as risk factors and, 85-86, 105
fundamentals of, 160-161, 546-549
implant design and, 69, 81, 84, 143f, 202, 204, 214f
implant failure and, 69-72, 69b
interim prosthesis for, 270
overload zone of, 132-133, 133f
patterns of, 559f, 560f
treatment planning and, 84-87
Stress corrosion cracking (SCC), 513-515
Stress Treatment Theorem
prosthetic options and, 92
rationale behind, 69, 84, 87-88
Sublingual artery hemorrhage, 713
Sublingual space, 506, 507f
Submandibular lymph nodes, 508-509
Submandibular space, 506
Submental lymph nodes, 508
Submental space, 506-507
Subtraction radiography, 61-62
Success rates
criteria for evaluating, 1056b, 1077b, 1078b
implant design and, 201t
for implants vs. prosthesis, 156t
of nonvital tooth, 251
vs. survival reports, 1055-1056
Sulcus, 74f, 75f
Sulfonamides, 965
Sunlight, 581
Supplements. *See* Herbal supplements.
Surface, implant. *See also* Coatings, biomaterial.
acid-etched, 606-610, 607t
anodized, 610-612, 612t
bone-implant contact and, 599-601, 600t
comparative studies on, 604t, 606t, 610t, 611t, 615-616
immediate loading and, 812
roughened, 220
sandblasted, 603-604, 608-610
turned, 602
Surface area, implant
functional vs. theoretical, 206-207
immediate loading and, 808-812, 818
of implant designs, 214t
length of implant vs., 163-166, 163-165f
of roots, 266, 810f
thread depth of, 214-215
thread pitch of, 210-213
thread shape of, 212-214
Surface-tissue interface. *See also* Bone-implant contact (BIC).
biomechanical environment of, 621
ceramic surfaces in, 529-530
coatings and, 530-533
metal/alloy surfaces in, 526-527, 528-529
surface cleanliness and, 533-535
surface modifications and, 533
tissue interactions in, 527-528
Surgery, implant. *See also* Osteotomy, implant.
bone density and, 646-647
bone-implant contact and, 599
as focus of implant design, 200
instruments for, 716f
nonsubmerged first-stage, 799
for two-piece implant system, 28-31, 29f
types of, terminology for, 799
Surgical ciliated cyst, 923
Surgical guidance
CAD/CAM guides for, 284f, 285-288
computer imaging for, 52-54, 288-290
preimplant guides for, 284-285, 286f
templates for, 280-283, 283f
SurgiGuide, 53-54, 287-288
Survival reports
of iliac crest grafts, 1018t
and implant design, 201-202f

WWW.HIGHDENT.IR
همیار دندانسازان و دندانپزشکان

Survival reports—cont'd
of implants, 294, 333, 343
of removal partial dentures, 16-17
vs. success rates, 1055-1056, 1077
of tooth replacement options, 5, 7
Sutural expansion, facial, 585-588
Swallowing, 16, 105

T

Tactile sense, 138
Tamoxifen, 581
Taste, 21
Teeth, natural
biological width of, 74f
evaluation of, 250-253, 252b, 255
ideal conditions of, 1078b
vs. implant, 1055, 1069, 1073-1074
joining/splinting implants to, 261-262
position of, 14f, 233-234, 265-266
size of, 173
Temperature, body, 427
Templates
diagnostic radiographic, 62-63, 64f
for surgical guidance, 54, 280-283, 283f
Temporalis muscle, 496
Temporomandibular joint (TMJ)
articulation of, 559-562, 561f
case study on, 583b
evaluation of, 250
muscles attached to, 496
Tensile load/force
bone response to, 621-624
fundamentals of, 548f
in marginal bone loss
animal studies on, 78-79
bone physiology and, 82
implant design and, 83
Terminology, generic, 26-27, 34
Tetracycline
for bone graft infection, 840
dosage information for, 473t
for prophylaxis, 472
pseudomembranous colitis and, 470b
for sinus infection, 965
Thermal injury. *See* Heat stress.
Thread geometry, 205f, 209-215
Threaded implant body, 29-30, 810-811
Thrombophlebitis, 502
Thyroid disorders, 444-445, 444t
Tibia bone graft, 1036-1039, 1040-1045f
Timing of therapies, 342, 475
Tissue, fibrous
formation of, after surgery, 805-806
implant design and, 160
proliferation of, after grafting, 862
Tissue, keratinized
concerns regarding, 1061-1063, 1062f
uncovery procedure and, 733-736
Tissue, soft. *See also* Reflection (landmark ID).
contouring, 756b, 758-765
coverage in bone graft, 841-845, 842-843f
edentulous effects on, 12-15, 13f
evaluating support of, 267-268
imaging to evaluate, 48b
maintenance of, 1073-1074, 1076
in mandibular surgery, 698
necrosis of, 503
after socket grafting, 888-890
Tissue, soft—cont'd
support by, 733
thickness of, 731-733
Tissue drape
after implant, 357-360, 358f, 765f
incision/closure for, 750-755, 752f
after tooth loss, 350-351, 351f
Tissue junction
marginal bone loss and, 74f
probing to measure, 75f, 1064-1065t, 1064f
Titanium and alloys
bone-implant contact and, 601-602
ceramics from oxides of, 518-519
coatings/plasma spray made of
bone-implant contact and, 605-606
immediate loading and, 812
for initial loading, 203
surface-tissue interface of, 531-532
instruments made of, 1076
mechanical properties of
and force loads, 222-223f
for single-tooth implant, 335
for surgical implants, 515-517, 516t
modulus of elasticity of
and bone density, 82, 139-140f
and implant strain, 77, 78f
surface-tissue interface of, 528-529
Tatum implant made with, 930f
Tobacco use, 458-459, 924-926
Tomography. *See also* Computed tomography (CT).
to identify vital structures, 56-58, 57-58f
intraoperative, 54
microcomputed, of bone, 567-568, 567-568f
postprosthetic, 62
preprosthetic, 62-63, 64f
presurgical, 46-56
computed, 47-51
cone beam volumetric, 50-51
conventional, 46-47, 47b, 48f
interactive computed, 52-54
magnetic resonance, 54-56
Tongue
edentulism and, 13
force of
clenching and, 114f
increased size and, 117f
measuring natural, 105
infection of, 507
scalloped border of, 113, 114f
soft tissue–borne prostheses and, 21
Tongue thrust habit, 85, 116-117, 116f
Tooth extraction. *See* Extraction, tooth.
Tooth loss. *See* Edentulism.
"Toothbrush abrasion," 113
Torque force
fundamentals of, 550
implant failure and, 69
implant length and, 86
reverse, 214t
testing for reverse, 728-730
when evaluating abutments, 257
Torsion of mandible, 316-317
Trabeculae
dental implants and, 17-18
tooth loss and, 10
Trabecular bone
calcium conservation and, 582
density of, 138f, 141f
coarse (D2), 655-658
fine (D3), 658-662
fine (D4), 662-665
grafts from, 976-977, 1015-1019
marginal bone loss and, 82f, 83f
in opposing arches, 131f
response to stress and, 632-634, 633f
stress vs. stimulation of, 82f
Tramadol, 478t, 479
Transfer copings, 31f, 33, 34f, 60
Transforming growth factor beta (TGF-β)
in bone grafting, 850
platelets/mL and, 851f
in socket graft, 888-890
in socket grafting, 877
Transitional restorations/prostheses
for bone graft healing, 854-855, 855-856f
for healing period, 736-737
for immediate restoration, 824-829
for pretreatment, 233, 269b, 270-271
for single tooth replacement, 334-335, 344-345, 351
in socket grafting, 899
Treatment planning
corollary factors in (listed), 87
goals of, 92, 102-104
limited treatment as, 293-294
long-term, 324-325
preimplant
advanced guidance in, 284-285
diagnostic casts for, 276-278
overall evaluation in, 233b
prostheses considerations for, 233, 268-270
sequence for, 271b
specific criteria in, 233b, 241-268
surgical guides for, 280-283
rationale for, 3, 6-7
sequence for, 84b, 92-94, 93f, 200
stress/force factors in, 69, 84-87, 87b
Trephine bur bone harvest, 882, 885-886f
Triazolam, 484t, 486
Tricalcium phosphate material, 520t, 521-522, 615
Trimethoprim/sulfamethoxazole, 473t, 965
Tubercles, genial, 11, 492f
Tuberosity donor site, 879, 882f, 1005t
Tungsten, 518

U

Ultrasound evaluation, 917
Ultraviolet light, 534-535
Uncinate process, deflected, 919
Uncovery procedure, 723-731, 758-765, 763f
Urinalysis, 428

V

V2 nerve. *See* Maxillary nerve (V2).
Valvular heart disease, 440-442
Vancomycin, 470b
Vascular bone graft, 1039-1043
Vascular endothelial growth factors (VEGFs), 851f, 889-890
Vascular supply/circulation
age-related changes in, 500-501

WWW.HIGHDENT.IR

همیار دندانسازان و دندانپزشکان

Vascular supply/circulation—cont'd
to bone graft, 848-849
edentulous effects on, 13
iliac bone harvesting and, 1022
to mandible, 494f, 498-500
marginal bone loss and, 73
to maxilla, 494-495, 494f, 498-499
to maxillary sinus, 910-911, 911b
probing to measure, 1064-1065t
to sinus graft, for healing, 951-952
Vector resolution, 545
Venous drainage, 494-495
Virtual Scope, 289
Viscoelastic material, 630
Vital signs, patient, 423, 426-427
Vitamin D absorption/disorders, 451, 581
Volkmann's canals, 498

W
Wallerian degeneration, 501
Warfarin sodium, 457-458
Waters' projection radiographs, 915
Wear patterns, tooth, 107-108, 108f
Weight, body, 421
Weight (gravitational force), 543-544
Width (BW), biological
marginal bone loss and, 74-76, 218-219
of natural tooth, 1064f
probing to measure, 1064-1065t
"Wigs," dentures as oral, 19
Wilson, curve of, 239f
Witch's chin, 14f, 15, 496
Wolff's law, 9
World Health Organization, 480-481, 481b
Wound. *See* Healing period; Infection.
Woven bone, 568-569, 805, 808

X
Xenograft, 862-863

Z
Zinc phosphate cement, 72
Zirconia, 615
Zirconium oxide, 518-519
Zonography, 46